Bailey & Love's Short Practice of Surgery

A. J. Harding Rains
and H. David Ritchie

LOW-PRICED EDITION

Eighteenth Edition

BAILEY & LOVE'S SHORT PRACTICE OF SURGERY

BAILEY & LOVE'S SHORT PRACTICE OF SURGERY

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BAILEY & LOVE'S SHORT PRACTICE OF SURGERY

EIGHTEENTH EDITION

Revised by

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PREFACE TO EIGHTEENTH EDITION

The pace of advance in our knowledge of surgical pathology and therapy shows no sign of slowing down. If anything during the four years since the last (17th) edition it has increased. It has always been the aim of this book to try to offer a clear, concise and authoritative account of the state of the play at any particular time. Hence we offer this edition of Short Practice of Surgery, the 18th to become available during the last 50 years.

New investigative and surgical techniques which have proven their worth are described and several distinguished contributors have joined us in our efforts to maintain the high standards achieved in the past. These are duly recognised by name on pages vi–vii.

Surgical practice is, it would seem to us, becoming slowly but surely more uniform throughout the world. Not all countries, however, are equal in the facilities they can provide for their surgeons. Our aim has been to try to ensure at least that they may all be equally well informed. If this goal is to be reached student and surgeon alike must have available an accurate account of contemporary surgical practice.

A. J. HARDING RAINS
DAVID RITCHIE

March 1981

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SAYINGS OF THE GREAT

Both Hamilton Bailey and McNeill Love, when medical students, served as clerks to Sir Robert Hutchison, 1871–1960, who was Consulting Physician to the London Hospital and President of the Royal College of Physicians. They never tired of quoting his " Medical Litany " which is appropriate for all clinicians, and, perhaps especially, to those who are surgically minded.

" From inability to leave well alone ;
From too much zeal for what is new and
contempt for what is old ;
From putting knowledge before wisdom,
science before art, cleverness before
common sense ;
From treating patients as cases ; and
From making the cure of a disease more
grievous than its endurance,
Good Lord, deliver us."

to which may be added:

" The patient is the centre of the medical universe around which all our works revolve and towards which all our efforts trend." J. B. Murphy, 1857–1916, Professor of Surgery, Northwestern University, Chicago, U.S.A.

" To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all." Sir William Osler, 1849–1919, Professor of Medicine, Oxford, England.

A SHORT PRACTICE OF SURGERY

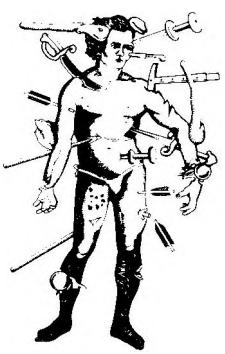
CHAPTER I

WOUND HEALING AND MANAGEMENT

' Skin is the best dressing '.—Lister

' A wise physician skilled our wounds to heal is more than armies for the common weal[1] '.—Homer

FROM the earliest times the healing of wounds has been the central problem in surgical practice (fig. 1). This applies equally to the wounds of warfare and human assault, to the wounds of accidents, and to the wounds which the surgeon makes deliberately in the course of surgical operations. Once made,



From a handbook for army surgeons, in the middle ages.
FIG. 1

FIG. 2.—Today! ' *Plus ça change, plus c'est la même chose* ' (' The more things change, the more they are the same ').—*Karr.*

the breach in the surface of the body, the skin, exposes the deeper tissues to the danger of bacterial infection (sepsis), and this danger persists until the healing process has restored an intact surface.

WOUND HEALING

Healing by First Intention.—Rapid healing of the wound in all its layers, particularly rapid skin cover, is the ideal objective—the first intention

[1] Weal = the general good, the welfare of a country.

Joseph Lister (Lord Lister), 1827–1912. Professor of Surgery, Glasgow, Scotland (1860–1869), Edinburgh (1869–1877), and King's College Hospital, London (1877–1892). He reported on the use of a carbolic dressing to prevent infection in a paper in the Lancet in 1867 (p. 22).

Homer, date of birth uncertain between 1050 and 850 B.C., somewhere in Greece. Still regarded as the great epic poet and author of the ' Iliad ' and the ' Odyssey '.

Jean Baptiste Alphonse Karr, 1808–1890. Journalist, Paris. Sometime editor of Figaro. Also a great gardener.

—of the surgeon. Accurate apposition using sutures[1], clips, staples or adhesive tape or film is essential (p. 22).

Alternative methods of wound closure become mandatory in the presence of tissue tension or infection. *Delayed primary suture*, *secondary suture*, and *skin grafting* can be used to provide skin cover.

Healing by Second Intention occurs: 1. When the wound edges are not brought together. 2. When there is skin loss which cannot be repaired. 3. When the wound becomes infected and breaks open, or has to be laid open.

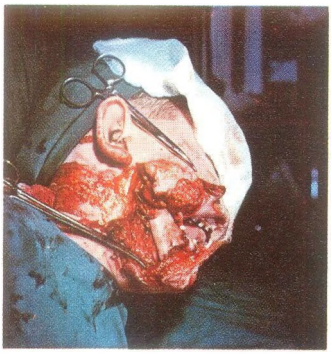


FIG. 3.—Severe facio-maxillary injury. (*Rainsford Mowlem F.R.C.S.*)

FIG. 4.—Accurate suture for healing by first intention (fig. 5). (*Rainsford Mowlem, F.R.C.S.*)

FIG. 5.—See figs. 3 and 4. Healing by first intention.

FIG. 6.—Ulcers of the leg healing by second intention.

Healing by second intention is the normal way of healing of an ulcer (fig. 6) or of an abscess after it has been drained.

Healing by second intention is much slower than healing by first intention and invariably involves the laying down of more scar tissue (p. 24). The longer the wound remains raw, the greater will be the amount of scar tissue laid down and the greater will be the contraction and deformity.

[1]Suture (Latin) = Sewing together (a stitch). A seam. Ligature (Latin) = Anything that binds together.

Factors Influencing Wound Healing

General State of the Patient.—A debilitated patient, whether due to malnutrition or carcinomatosis, is depleted of resources, and wound healing will often be considerably delayed. Other factors affect the fibroblastic activity of repair, notably uræmia, the use of cytotoxic drugs, the administration of steroids, and Vitamin C deficiency.

Inhibition of wound healing is a feature of the jaundiced patient, the cause unknown. Generalised infection, or a collection of pus somewhere, may delay healing of a wound in another part. It is important under these conditions that any deep sutures in muscle planes should either be of non-absorbable material, or deep tension sutures should be employed, and the skin stitches should be left in longer than usual. When much tissue healing is required, e.g. after an extensive burn, the patient needs a high protein, high calorie diet.

Local Factors.—*A good blood supply* is essential for wound healing. Wounds of the face and hands may bleed profusely but they heal well because of their excellent blood supply. Wounds below the knees, over the shin and calf and below heal badly owing to a relatively poor blood supply.

The presence of tension in the tissues is a major factor in loss of local blood supply and subsequent wound failure. Tension will be increased by dependency of a limb due to venous stasis. A localised hæmatoma is also a common cause of local tissue tension and wound breakdown.

Local infection delays wound healing in several ways. Local tissue tension is increased by the inflammatory response, with an increase in capillary permeability and the collection of pus. The metabolic demands of infection may increase the local demand for oxygen beyond the capability of the blood supply. Tissue necrosis will ensue with subsequent wound breakdown.

Relative arterial insufficiency is often seen in the atherosclerosis patient and is common in the diabetic, the presence of infection often tipping the balance between tissue life and death.

The technique of wound closure is an important iatrogenic cause of wound breakdown. The commonest error is suturing in the presence of tissue tension.

FACTORS AFFECTING WOUND HEALING

General Factors	*Local Factors*
Age	A poor blood supply due to vascular disease or trauma is unfavourable to wound healing.
Debilitation (Vitamin C deficiency)	Tissue tension kills.
Malignant disease	Local infection delays healing.
Uræmia	Hæmatoma formation is unfavourable.
Jaundice	The technique of wound closure influences repair.
Cytotoxic drugs and steroids	Local x-irradiation delays healing.
Diabetes	
Generalised infection	

Completion of Healing.—Complete healing of a wound takes a long time. The collagen fibres of the uniting scar tissue undergo repeated dis-

ruption and reformation before final maturation. The tensile strength of the wound only approaches that of normal tissue after 6 months, but 100% completion may take as long as 2 years.

Types of Wound and their Closure

It is useful for the correct management of wounds to know the mechanism of injury. In this way it is possible to plan wound exploration and closure in a logical sequence, varying the technique to the type of wound.

Incised Wounds — Primary Suture.—The majority of incised wounds are caused by a sharp knife or glass and are relatively clean. Structures will be damaged only along the track of penetration with minimal tissue injury to structures on either side. After suitable exploration these wounds may be closed by *primary suture*. Damaged tendons, nerves and major blood vessels should be repaired at the time of primary surgery.

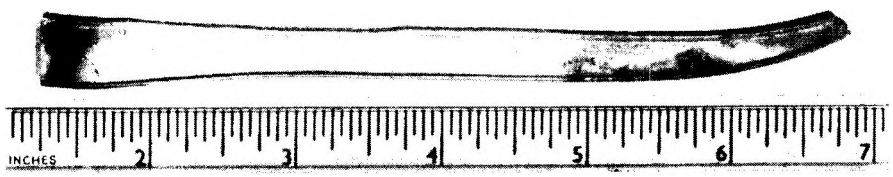


FIG. 7.—Following an air raid a man, aged fifty, walked into an adjacent hospital because of minor facial wounds. He collapsed and died a few minutes later, quite unaware that this slender piece of glass, which was just protruding through the skin, had penetrated three layers of clothing and his abdominal wall. It had severed the middle colic artery.

(*Mildmay Mission Hospital, London.*)

Penetrating wounds are dangerous (fig. 7). Beware of the innocent-looking stab wound of the abdomen! Not infrequently the long stiletto knife blade has traversed several intraperitoneal viscera. All such wounds must undergo exploration with a view to laparotomy, even in the initial absence of peritonism. Wounds in the thigh are common, especially in the meat trade (butcher's thigh[1]). Always look for damage to the neuro-vascular bundle and be prepared to secure proximal and distal control of the major vessel before exploration of the damaged arterial segment.

The razor blade drawn across the front of the wrist several times, as part of a suicidal gesture, usually results in only superficial damage. A more determined effort, with often only one slash, usually severs nerves and tendons on the anterior aspect of the wrist. A lengthy surgical exploration is required.

All these wounds should be closed by primary suture, if possible within six hours of injury. In all cases examine the arterial pulse and nerve supply distal to the injury.

Lacerated Wounds — Wound Excision and Primary Suture of Skin.—These wounds with jagged edges are commonly seen following road traffic accidents. They are frequently dirty and contaminated with organic

[1] Butcher's thigh. Accidental division of the femoral artery caused by the slipping of the boning knife during the preparation of joints of meat (see pp. 69 and 147).

matter. In the presence of devitalised tissue, especially muscle, the risk of infection is increased. The majority of these wounds can be treated by wound exploration and excision with primary closure. The key to success lies in the meticulous removal of all dead tissue, the removal of organic matter such as clothing and irrigation of the raw area prior to a layered closure of the wound. The object is to convert the lacerated wound into one that approximates to an incised wound. Complicated repair of tendons and nerves is not recommended at the time of initial surgery because of the risk of infection Their repair can wait for 4–6 weeks when good superficial cover can be assured. These wounds should be treated within six hours of injury.

Crushed and Devitalised Wounds = Wound Excision and Delayed Primary Suture.—Industrial and severe road traffic accidents account for the majority of these wounds. War injuries also fall into this group, but as the mechanism of wounding and subsequent management is slightly different they will be considered as a separate entity (Chap. 2).

All major crushing injury is accompanied by a degree of tissue necrosis. The area and depth of devitalised tissue will depend not only upon the area and weight of the crushing force but also on the duration and velocity of impact. Severe crushing injury may have a profound general as well as local effect especially if pressure is unrelieved. Three difficulties exist in this type of wound:

1. It may be impossible with certainty to distinguish between viable and non-viable tissue.

2. Because of the extensive tissue damage great swelling of the tissue may occur. If primary suture is performed the inflammatory œdema and therefore the tissue tension may rise to dangerously high levels resulting in local ischæmia[1] and local death in tissues which were previously viable. Dead muscle is the ideal environment for the development of infection (gas-gangrene).

3. The wound may be heavily contaminated by bacteria.

The key to the management of these wounds is the excision of all dead tissue and the prevention of tissue tension. All necrotic muscle must be removed along with indriven organic matter. Tension beneath the deep fascia must be relieved by a long fasciotomy incision. After irrigation the wound should be left open and dressed. The wound is re-examined 4–6 days later. If it is found that all remaining tissue is viable and the œdema has subsided sufficiently to allow the wound to be sutured without tension, delayed primary suture can be performed. This two-stage procedure is successful for all heavily contaminated wounds, especially in the presence of tissue tension. It is of paramount importance in missile injuries.

So important is it to avoid tension that it may be safer to close the wound by skin grafting even when no skin loss has taken place. When the wound has healed and the tissue tensions have returned to normal the graft can be excised and the skin sutured in the usual way.

If there is any doubt about the advisability of suture—DON'T.

[1] Ischæmia (Greek) = A lack of blood flow.

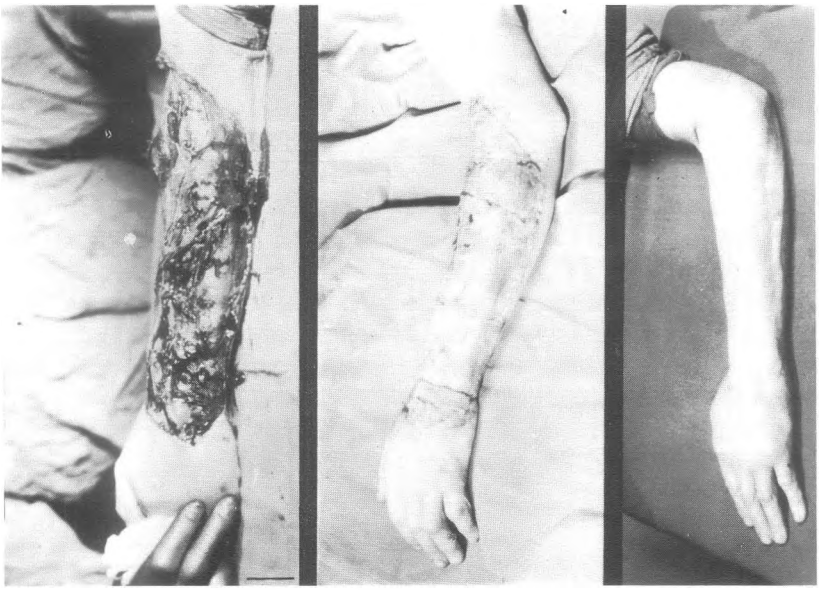


FIG. 8. FIG. 9. FIG. 10.

FIGS. 8, 9 and 10.—Wound excision and primary skin grafting.

Wounds with Skin Loss (figs. 8, 9 and 10).—The patient is in danger from superadded infections until the wound has completely healed by re-epithelialisation of the skin surface. Healing of the skin layer is of importance not only in its own right but because the healing of deeper structures, such as the tendons and bones, can only take place satisfactorily in the presence of an intact skin cover. Furthermore, the longer any exposed surface remains raw the greater will be the deformity and disability. In all wounds with skin loss therefore, the skin loss should be repaired as soon as is practicable and as follows:—

1. Clean incised wounds with skin loss—Primary grafting.
2. Lacerated wounds with skin loss—Wound excision and primary grafting.
3. Crushed and devitalised wounds with skin loss—Wound excision and delayed primary, or occasionally secondary grafting. For types of skin-grafting for burns wounds, see p. 131.

Secondary Suture, by freeing skin at the edge of a granulating wound is rarely used. It is easier and safer to perform secondary grafting and to carry out any reconstructive surgery later.

PRACTICAL MANAGEMENT OF THE SEVERELY INJURED

It is probably true to say that simple life-saving first-aid principles account for the saving of more lives than heroic surgery.[1] To save life it is frequently necessary to commence treatment before a definitive diagnosis is made.

[1] The essentials of *First Aid*—attention to the three B's—' Breathing ' (airway), ' Bleeding ' (Chap. 7) and ' Breaks ' (fractures). Beware of and prevent *the second accident*, namely loss of life due to lack of sustained first aid during transport and until handing over to a proper resuscitation team.

1. *Secure the casualty from further injury in the danger zone.* This is particularly applicable on motorways.

2. *Airway.* Ensure an adequate airway, removing blood and debris from the victim's mouth and throat. The use of a good light and mechanical suction are important aids. Maintain and protect the airway by placing the victim in the lateral position, head down, using an oropharyngeal airway if available. In the hands of skilled personnel endotracheal intubation has several advantages in major trauma victims, the airway is secured and protected in addition to providing the facility for artificial ventilation and tracheobronchial suction. The deeply unconscious patient and the severe chest injury associated with a large flail segment benefit greatly by intubation and assisted ventilation. Tracheostomy and cricothyroidostomy are reserved for those cases that cannot be intubated.

3. *Stop hæmorrhage* (Chap. 7). Bleeding can usually be controlled by the application of local pressure using a padded dressing. A tourniquet is sometimes life-saving in traumatic amputation of a limb. Elevate the injured limb.

4. *Relief of tension.* Tension, whether it be intrapleural or intrapericardial must be relieved at once. Venting a tension pneumothorax using a large bore catheter inserted through the second intercostal space about three inches (75 mm) from the midline, and connected to an underwater drain, can be life-saving. Acute cardiac tamponade is difficult to diagnose but, if recognised early, aspiration through a wide-bore needle inserted to the left of the xiphoid process is essential. Relief of acute gastric tension via a nasogastric tube is sometimes necessary.

5. *Secure an intravenous line.* The body compensates so well for blood loss that its response can lull the resuscitation officer into a false sense of security. The estimation of loss of blood is sometimes difficult. Place a large bore cannula into a vein before it collapses due to shock. Perfusion of tissues by an adequate circulation volume is the aim of early resuscitation. Initially a litre of crystalloid (Hartmann's solution) followed by a litre of colloid solution (Dextran 70) can be infused while waiting for cross-matched blood. Only rarely, in the severely exsanguinated, is universal donor O-negative blood infused.

6. *Oxygen administration.* It has been shown that the inhalation of oxygen is of advantage to the major trauma victim.

7. *Splint fractures.* To prevent pain and further damage to soft tissue structures all fractures of the limbs should be splinted.

The Practical Technique of Wound Management

1. Examine the patient for other injuries. Is the patient shocked?

2. Examine the wound. Bleeding and pain often make inspection difficult. Much useful information can be obtained from a brief superficial look at the wound.

3. Have any deep structures been divided by the injury? More complications occur simply because the observer has failed to think about what structures might have been involved than because of any technical short-comings in the operation. Limbs—has a major blood vessel been injured? Examine the peripheral pulse. Has a tendon been involved? Test movement. Has a nerve been involved? Test

movement and sensation. Examine for fractures. The abdomen—has a viscus been involved? If so, which viscus? Examine the urine after catheterisation if necessary. The head—has the skull been damaged or the dura breached with injury to the brain, eye or the ear? (See Head Injuries, Chap. 26.)

Procedure in the Operating Theatre.—Dirty wounds are best initially cleaned in the anæsthetic room with a detergent antiseptic solution, i.e. Cetavlon or Povidone iodine. Once the temporary dressing is removed the wound often bleeds profusely, especially under general anæsthetic. Have an intravenous line in position. A brief inspection of the large wounds enables one to plan surgery before operation. Tattered skin should be trimmed but viable skin must not be excised. If necessary the wound must be extended, preferably in a longitudinal direction, to obtain a good view of underlying tissue. The deep fascia must be freely excised in the presence of tension or hæmatoma beneath it, in order to open up the muscle compartments. Damaged muscle must be excised until the muscle bleeds freely and contracts when it is cut. Bone is often viable and any fragment should be replaced in position. Foreign bodies should be located and removed. Any tendons, nerves or major blood vessels crossing the wound should be carefully examined to determine if they have been injured or not. In a contaminated wound tendons and nerves should not be sutured at initial operation. If the abdomen, chest or head have been involved by the wound, it must be suitably enlarged so that the deeper parts can be thoroughly examined and any necessary repair carried out. Guide-lines with regard to the management of contaminated wounds and of their skin closure are given in Chap. 2. Any necessary splintage should be applied both to immobilise the fracture and to rest the soft tissues, *even* in the absence of a broken bone.

Antibiotics in the Treatment of Wounds

The most important factor in the avoidance of infection in wounds is careful operative treatment as described above, because if only healthy, well vascularised tissues are left in the wound, the body's defences will be able to deal with all but the most virulent organisms. With extensive wounds with much tissue damage, or if there has been any delay in treatment, systemic antibiotic therapy should be administered. Systemic penicillin over a period of 5 days should be routine, further therapy being dependent upon clinical progress and on the result of bacteriological examination of wound swabs (p. 20). When the gastrointestinal tract is wounded a combination of metronidazole and a cephalosporin or gentamicin is required at the time of assessment and operation (Chap. 3).

Prophylaxis against tetanus is described on p. 31.

Foreign Bodies in the Tissues

In every case of suspected foreign body which is opaque to x-rays, a radiograph must be taken in at least two planes. When removal is attempted, a good light, ample time, proper anæsthesia and a bloodless field are all invaluable together with the use of x-rays in the localisation of the object by means of metal skin markers, needles and in particular an x-ray image intensifier with a T.V. display screen.

Hypodermic needles may break at the neck. The most difficult needle to recover is one situated in the internal pterygoid muscle, which has broken when a dental surgeon has attempted a mandibular block. The glistening tendinous intersections in the muscle continually raise the surgeon's hopes, but patience is usually rewarded.

Domestic needles commonly become impacted between the small bones of the hand or foot. The patient may be entirely unaware of their entry. Unless a small

fragment is lodged deeply, removal is advisable, as infection may otherwise develop, and startling cases are on record in which a needle has entered the venous circulation and become embedded in the heart muscle, or travelled to some distant part of the body.

Sewing-machine needles occasionally transfix the terminal phalanx and nail, and then break. The finger should be forcibly pressed on to a hard surface, so that the fragment retraces its path. The end then projects through the nail and is removed with forceps.

Fish hooks, and similar articles which possess barbs, are removed by pushing the hook in such a direction that it emerges through the skin at the nearest point. The barb is nipped off, and the hook withdrawn.

Gravel is not uncommonly driven into the subcutaneous tissues of the face, hands, or knees. Ugly scars are the penalty for incomplete removal—a particularly distressing sequel if occurring on the face—' tattoo marks '. In all but minor cases an anæsthetic and meticulous removal are indicated. Brisk rubbing with a nail-brush is sometimes helpful.

Glass splinters from windows usually contain sufficient lead to make them radio-opaque. In fact, contrary to popular belief, most glass is radio-opaque in soft tissues. Every lacerated or punctured wound caused by glass must be examined radiographically, as it is surprising how often fragments of glass are otherwise missed. In the majority of cases removal is indicated.

Surgical operations in which metallic sutures are used occasionally lead to trouble. Thus, **wire sutures** in the abdominal wall sometimes break within a few days of their insertion, and the fragments may then cause a pricking pain in the wound. It is remarkable that even stout wire undergoes ' metal fatigue ' in time and breaks into small pieces. The fragments in some situations cause serious trouble.

Pieces of clothing may be found in gunshot wounds (see above and Chap. 2).

Bites and Stings

Insect bites in the United Kingdom are usually inflicted by wasps or bees. Anaphylaxis can follow a bee or wasp sting in sensitised people (p. 32), especially if the venom is injected directly into a vein, e.g. in the dorsum of the hand. Wasps are fond of alcoholic drinks, and picnickers especially should keep an eye on their beer, lest, after drinking, they suddenly develop œdema of the glottis. Most bees, as distinct from wasps, suffer avulsion of their sting and poison gland, which is left protruding from the wound. Pressure on the gland squeezes poison into the tissues, so the gland and sting should be removed by scraping. *Bee venom is acid*, and should be neutralised by the application of ammonia, soda, or methylene-blue. On the contrary, the *venom of the wasp is alkaline*, and requires an acid, such as vinegar or lemon juice, for its neutralisation.[1] Antihistamine drugs are given orally, and local application is of value in allaying local irritation. If an anaphylactic reaction occurs, adrenaline (1:1000) 0·5 ml, is injected intramuscularly every 10 minutes until the patient recovers.

Bites of animals such as the horse, cat, and dog require the usual treatment of wounds. Where there is the slightest suspicion that the animal is suffering from rabies, the bite should be freely excised. If possible, the responsible animal should be kept under observation, or, if it has been killed, Negri bodies should be sought in the brain. Prophylactic treatment can be obtained only at a special institute.

Snake Bites.—In England the only poisonous reptile is the adder, or viper (*Vipera berus*), but a fatal result is unlikely. First-aid measures consist of the application of a tourniquet, and within the first few minutes sucking the wound may remove some of the venom. Antihistamine drugs are given. Drastic measures such as excision of the wound and the application of permanganate of potash cause unnecessary trauma or necrosis of tissue. Antivenom serum is not used in England because of the risk of serum anaphylaxis and serum sickness.

Scorpion stings, well known in the deserts, cause intense pain for many hours and constitutional upset. The patients respond well to emetine injected locally, 65 mg for adults, and proportionately less for children (Dodge). Hydrocortisone is perhaps a more readily available alternative.

Human bites are discussed on p. 208.

[1] Bicarbonate for Bee, Vinegar for Vasp!

Adelchi Negri, 1876–1912. Professor of Bacteriology, Pavia, Italy. Discovered deeply staining inclusion bodies in the cells of the central nervous system in cases of Rabies in 1903.
Jeffery Samuel Dodge, Contemporary. Director of Community Services, Otago Hospital Board, Dunedin, New Zealand.

CHAPTER 2

WAR WOUNDS

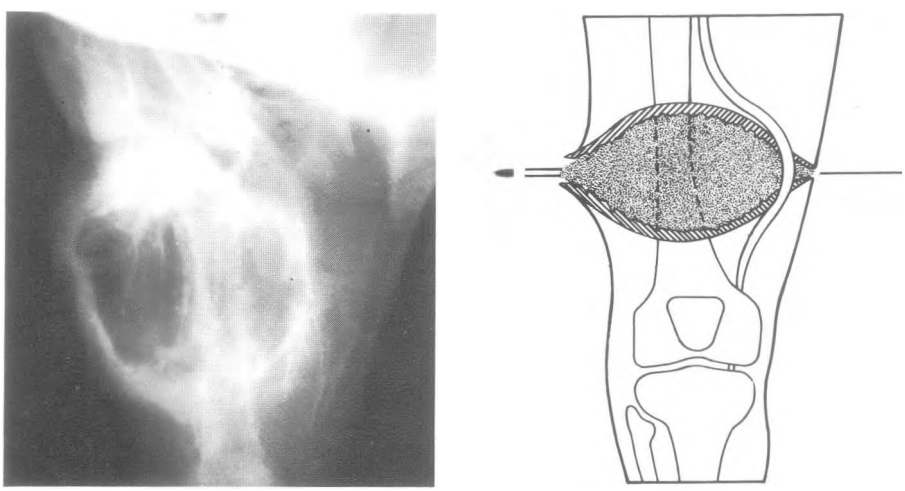
OUTBREAKS of violence in the world make it necessary to understand the mechanism and the management of missile (bullet) and bomb blast injuries.

MISSILE INJURIES

Mechanism of Missile Injuries.—The amount of damage to the tissues of the body is dependent upon the kinetic energy (KE) released by the missile as it traverses the tissues, calculated from the formula $KE = \frac{1}{2}MV^2$ (M = mass, V = velocity).

The low velocity missile, such as a bullet from a pistol, travelling below 1100 feet per second, injures by a direct effect on the tissue by laceration and crushing within the narrow confines of the missile track.

The high velocity missile, the rifle bullet has a greater lethal potential because tissue destruction will occur many centimetres on either side of the missile track due to a rapid high pressure shock wave and a phenomenon known as *temporary cavitation*



(*a*) (*b*)

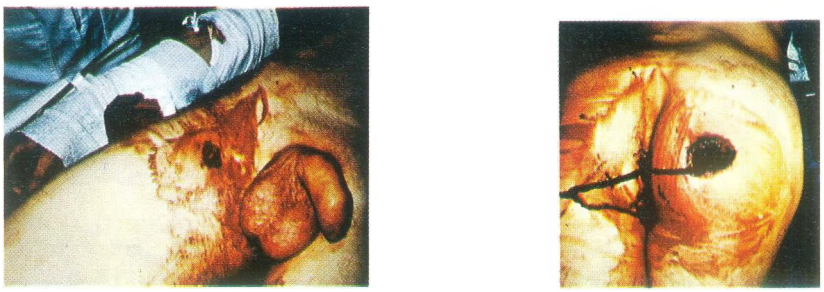
FIG. 11.—High velocity missile injury (experimental). (*a*) X-ray taken 30 msec after the bullet had traversed the limb. The cavity is collapsing. (*b*) Cavitational destructive effect due to the energy of the high velocity missile. The arteries and nerves are often temporarily displaced by such an insult.

(fig. 11). The amount of damage inflicted depends upon the density and elasticity of the target organ. Thus, comparatively little damage occurs in a 'through-and-through' wound of a lung, whereas the passage of a missile through or even close to the liver may completely shatter that organ. Blood vessels, nerves and bone may be damaged at a distance from the violent cavitation. Fragments of bone may act as secondary missiles. Particularly dangerous is the necrosis which occurs in the depths of muscle some distance from the missile track, a perfect culture medium for the

growth of pathogenic bacteria which are sucked in from the entrance and exit wounds due to the pulsatile nature of the temporary cavity.

Management of Missile Injuries.—The principles of management of soft tissue injury is routinely a two-stage procedure. The first step is a thorough wound excision, after which, with very few exceptions, the wound is left open. The second step is delayed primary closure, preferably within four to ten days after injury.

The entrance and exit wounds, frequently of only small diameter do not indicate the considerable damage that may have occurred to the deeper structures (fig. 12). It must be repeatedly emphasised that the higher the velocity of the missile, the greater the area of damage around the missile track, and the greater the accompanying tissue necrosis and œdema.



(*a*) (*b*)

FIG. 12.—High velocity missile wound. (*a*) Entrance wound. (*b*) Exit wound.

Therefore:—

1. *After cleaning with an antiseptic,* generous longitudinal incisions are made through the skin in order to examine thoroughly the deeper structures and to facilitate subsequent extension of the exposure, should this be required. A minimal amount of skin edge (i.e., only that which has been contaminated) is excised around the entrance and exit wounds, because skin is elastic and therefore relatively capable of withstanding the insult of being pierced by the rapid missile.

2. *The deep fascia is exposed,* and beneath it a tense bulging hæmatoma will be discovered. Therefore the deep fascia must be incised extensively in a longitudinal direction in order to decompress the underlying tissue and to allow adequate inspection of the necrotic area. *This step is of the utmost importance.*

3. *Identification of the neurovascular bundles* is now imperative (fig. 13), but it can be difficult in the damaged tissue. The search may be rewarded by finding nerves and blood vessels still intact in the midst of a large necrotic area.

4. *Dead muscle* that does not bleed, does not contract, and has an unhealthy colour must be excised. Sometimes it is difficult to distinguish bruised from dead muscle.

5. *Foreign matter* should be removed from the wound. Pieces of clothing are looked for especially, both in the missile track and in the tissue planes on either side. It is not necessary to remove every piece of metal seen on an x-ray. In mine injuries their particular retrieval will probably only cause further morbidity.

6. *Tendon repair* should not be performed at this particular operation. Tattered ends should be trimmed.

7. *Severed nerves* are tacked together without tension using two sutures. No formal repair is carried out. If the ends cannot be joined together they are tacked to

Note on 'Debridement'. (Débridement = unleashing).—This term was introduced by Dominique Jean Larrey, 1766–1842, the famous French surgeon in Napoleon's army. He used it to describe the removal of bullets, bits of cloth, loose bits of bone and soft tissue—a much less extensive procedure than the thorough wound excision which is now practised.

(*a*) (*b*)

FIG. 13.—High velocity missile wound. (*a*) Extensive exploration of the entrance wound in the groin was required in order to adequately complete wound debridement and gain proximal control of the femoral artery should this have been damaged. The extent of the skin incision is clearly demonstrated, which was partially restored at initial operation after exploration of the large vessels. (*b*) The exit wound in the buttock is shown here at the end of wound debridement prior to delayed primary closure.

adjacent tissue in order to prevent retraction. It is important to record in the operation notes the type of nerve injury (Chap. 28), for postoperatively a neurological deficit is manifest in spite of the appearance of an intact nerve at operation.

8. *Bone* is invariably shattered by a high velocity missile, yet at operation most of the fragments will be found still to have an attachment to periosteum or muscle. On no account should the fragments be discarded, because loss of bone results in loss of limb length or in non-union. Any contaminated bone is cleaned as well as possible by using that useful instrument of military surgery, the Volkmann's spoon or curette. Internal fixation is not employed at the time of initial surgery, except in some cases of multiple fractures in the hand or foot, or if it is necessary to protect an arterial anastomosis. External rigid fixation is permitted and is especially useful when there is a large area of skin loss associated with an underlying fracture. In the most cases the other important aid of military surgery, the padded plaster of Paris cast, will suffice. Alternatively the standard type of skeletal traction with e.g., a Steinmann's pin will provide adequate support for the bone.

9. *Major artery or vein damage* should be repaired if time permits. The ends of the vessel are trimmed half a centimetre beyond the macroscopic appearance of vessel wall injury. If any tension is likely a reversed vein graft may be inserted to bridge the arterial gap (Chap. 11). The repair is best covered by muscle and the rest of the wound left open for later closure.

10. *Joints* injured need thorough inspection, and cleansing with copious irrigation to remove organic matter. For ease of inspection a tourniquet may be needed (p. 74). Any exposed articular cartilage should be covered by at least one layer of tissue, preferably synovium, otherwise muscle or skin should be used.

11. *At the end of the operation* the wound is irrigated thoroughly to remove remaining debris. Hæmostasis is secured with the aid of hot packs and fine sutures. The wound is then left open without closure of either the fascial layer or the skin, even in the presence of exposed bone. A lightly fluffed gauze dressing is placed over the wound to allow free drainage.

Immobilisation in a well padded plaster of Paris splint aids rest to the soft tissues, whether or not there is an accompanying fracture.

Antibiotic cover with benzylpenicillin is advised for all wounds, and broad spectrum antibiotics are added in all cases of abdominal injuries and those that involve bones and joints.

Delayed Primary Closure.—All wounds treated by wound excision and left open are inspected under a general anæsthetic on about the 4th to 6th day after injury. Provided the wound looks healthy delayed primary suture is performed.

In certain wounds especially in the swollen limb, split skin grafting may be used to effect wound closure in order to avoid skin tension. If, at the

Richard von Volkmann, 1830–1889. Professor of Surgery, Halle, Germany.
Fritz Steinmann, 1872–1932. Surgeon, Berne, Switzerland. Described a pin for skeletal traction in 1907.

time of delayed primary closure dead muscle is found, perhaps having been retained due to doubt at the first operation, the muscle is excised and the wound left open for a further period before closure.

SUMMARY—DO'S AND DON'TS OF MISSILE INJURIES

DO	*DON'T*
INCISE THE SKIN GENEROUSLY	EXCISE TOO MUCH SKIN FROM WOUND MARGIN
INCISE THE DEEP FASCIA WIDELY	PRACTISE KEYHOLE SURGERY
IDENTIFY NEUROVASCULAR BUNDLES	STITCH TENDONS OR NERVES
EXCISE ALL DEAD MUSCLE	REMOVE BONE
REMOVE ALL INDRIVEN CLOTHING	CLOSE THE WOUND
LEAVE WOUND OPEN AT END OF OPERATION	PACK THE WOUND TIGHTLY BUT ALLOW FREE DRAINAGE: USING LIGHTLY FLUFFED GAUZE
RECORD THE EXTENT OF DAMAGE IN OPERATION NOTES	

Gunshot Wounds of the Abdomen.—Every gunshot wound of the abdomen should be explored. Adequate fluid replacement therapy must be available as severe blood loss may have occurred. Prior to surgery a catheter must be passed into the bladder. The rectum should be examined.

A longitudinal incision is used as a rule. It has the advantage of facilitating rapid access and extension when required. Usually much blood is encountered in the peritoneal cavity either from damage to the smaller mesenteric vessels or to a major vessel. The hæmorrhage must be controlled, and a careful examination is then made of all the abdominal contents.

In all wounds of the stomach the lesser sac must be opened in order to inspect the posterior gastric wall. It becomes essential to inspect the posterior wall of the duodenum by Kocher's method (Chap. 43), and also the retroperitoneal parts of the ascending and descending colon, (a) if there is any damage to these structures and (b) if any damage is suspected due to the shock wave and cavitational effect of a high velocity missile passing in proximity. The lethal potential of bruised bowel may well go unrecognised, and if it is returned to the abdominal cavity it is likely to become gangrenous and give way 24–48 hours after injury.

All the surfaces of the liver are inspected. Deep rents may be found in it following the passage of a high velocity missile close to it.

Small intestinal perforations are either excised and closed transversely, or the damaged section is resected if there are multiple holes in a short length. Mesenteric tears may necessitate bowel resection (Chap. 48).

Colonic and rectal wounds require a colostomy (Chap. 49). A proximal colostomy must be established in order to defunction the distal bowel whenever a damaged section of the left side of the colon is resected and an anastomosis performed. Alternatively the injured colon is resected and the proximal and distal ends are brought out on the surface of the abdominal wall, the proximal as a colostomy and the distal as a mucous fistula. Small wounds of the cæcum and ascending colon may be excised and repaired, using a cæcostomy to vent the bowel. Major wounds of the cæcum and ascending colon require resection of the damaged segment and a vented ileo-transverse anastomosis, or finishing with two ends on the surface (see above), but the proximal opening may have to be the ileum. In the very ill, simple exteriorisation of damaged colon (except the right side) is permissable. It must be emphasised that primary anastomosis without a drainage procedure courts disaster from leakage, sepsis, peritonitis and fistula formation.

Renal injury is treated by conservative surgery. Immediate nephrectomy is seldom indicated. The closure of a damaged calyx, with nephrostomy (Chap. 56), ring nephrostomy, along with the control of bleeding from the renal cortex by a free muscle graft will often suffice. It is unnecessary to try and repair the renal capsule.

Pelvic injuries are often fatal owing to the secondary hæmorrhage which follows sepsis. It is essential at the time of initial surgery to provide adequate drainage by excising the coccyx and placing drains in the presacral area.

Bladder and urethral injuries are treated by suprapubic cystotomy with placement of a retropubic drain after wound excision.

Liver injuries can be difficult to treat (Chap. 45). The principles involved in handling liver trauma include adequate drainage, suture for hæmostasis and resection of devitalised tissue. Suture of liver substance is primarily to secure hæmostasis and to stop biliary leakage. Suture should be placed deeply into the liver parenchyma to eliminate dead space. It is essential to provide adequate external drainage.

Damage to the spleen and tail of the pancreas is treated by resection. Missile injury of the head of the pancreas is seldom seen in the operating room because damage to it and the surrounding structures is usually fatal. Even in the unlikely event of a repair, it is necessary to fall back on creating a formal fistula by adequate external drainage, leaving resection or repair for a subsequent occasion.

Peritoneal toilet using a weak antiseptic is important to assist in the removal of spilled bowel contents and blood clot. Intraperitoneal drainage should be employed whenever bowel has been breached.

The entrance and exit wound should be debrided and the peritoneum closed. The rest of a missile wound of the abdominal wall can be left open for subsequent late closure.

Gunshot Wounds of the Chest (see also Chap. 40).—It is important to secure an air-tight seal of a sucking wound of the chest. Such a wound, if left open, will collapse the lung on the affected side with alteration of the ventilation/profusion ratio and in addition will decrease both the quality and the quantity of air entering the uninjured lung. As dyspnoea increases due to anoxia the mediastinum shifts on respiration and decreases venous return to the heart. This disturbance of cardiopulmonary function can be prevented by the simple first-aid measure of sealing the hole in the chest wall.

All penetrating wounds of the pleura and lung require adequate venting of the pleural cavity in order to prevent and deal with the embarrassment to respiration caused by the rapid accumulation of air under tension (tension pneumothorax) and blood (hæmothorax). It may be necessary to insert chest drains before a chest x-ray can be done.

Once pulmonary function is stabilised the operation of wound excision is carried out. During the operation of wound excision the pleural cavity is often entered and an opportunity should be taken to remove any retained foreign material, arrest hæmorrhage (usually from an intercostal or internal mammary vessel), and to oversew holes in the lung. Correct placement of the chest tubes is then confirmed. The pleural is closed and the wound left open for subsequent delayed primary closure.

A formal thoracotomy is indicated for (1) persistent air leak (2) continuing hæmorrhage from a venting tube (3) suspected mediastinal injury (4) thoraco-abdominal injury.

It is important to stress that the placement of a chest venting tube prior to positive pressure ventilation anæsthesia is essential in cases of lung injury because of the risk of causing a tension pneumothorax.

Gunshot Wounds of Head

The penetrating high velocity missile wound of the brain is lethal. The management of the low velocity penetrating missile and tangential wound depends initially on the maintenance of a satisfactory airway and restoration of blood volume in order to maintain adequate oxygenation of the brain. Good x-rays are mandatory to localise indriven bone fragments. Wound debridement is carried out using gentle irrigation and suction to remove necrotic brain and bony fragments. Every effort, including the use of a piece of temporalis fascia, is made to close the dura.

It has been shown that the application of intermittent positive pressure ventilation (PCO_2 4-4·7 kPa) assists in the reduction of intracranial pressure by reducing brain swelling. Intracranial transducers inserted through burr holes are now being employed to monitor intracranial pressure. In war wounds more maimed brains have been caused by the lack of oxygen than by the bullet.

Shotgun Injuries

Accidents from 12-bore shotguns are common and often lethal. It is never possible to retrieve all the shot and indeed to do so would result in too much damage to the

body. Wound excision should be carried out on the major wound, particularly looking for indriven clothing. Laparotomy is essential if it is thought that any of the shot has traversed an abdominal viscus.

The retention of lead shot in the body can result in a dangerously high lead concentration which should be monitored. After a time this concentration will fall due to encapsulation of the lead pellets.

BLAST INJURIES

Mechanism of Explosive Blast Injury.—The explosive pressure that accompanies the bursting of bombs or shells ruptures their casing and imparts a high velocity to the resulting fragments. These fragments cause even more devastating damage than bullets to the tissues as they are unstable in flight and tear through the tissues at great speed in a tumbling fashion. In addition, all explosions are accompanied by a *complex blast wave*. The two main components of this wave are a *blast pressure wave*, with a positive and negative phase, and the *mass movement of air*.

The positive pressure phase of the blast wave lasts only a few milliseconds, but close to an explosion it may rise to over 1 000 pounds per square inch (6894 kN/m^2). As the tympanic membrane ruptures at about 20 pounds per square inch (138 kN/m^2), it is evident that the effects on the human body of such an explosion can be devastating, especially in confined areas. Like sound waves, the blast pressure waves flow over and around an obstruction and affect anyone sheltering behind a wall. The pressure affecting such a person is known as the *incident pressure* (defined as the pressure level at 90° to direction of travel of the blast shock front). Also any person standing in front of a wall facing an explosion is subjected to the added effect of *reflected pressure*.

The negative phase of a pressure wave is of less amplitude but lasts longer than the positive phase.

A mass movement of air results from the rapidly expanding gases at the heart of an explosion which displace air at high velocity. This mass movement of air causes a *blast wind* which may result in total disintegration of the body and, at lesser levels, traumatic amputation. A person standing behind a wall will be protected from the blast wind unless, of course, the object or building collapses and causes a crushing injury.

Blast waves travel more rapidly and further in water being a more dense medium than air. Blast injuries are therefore more severe in water for a given distance than they are in air.

In the body, when the ' front ' of pressure travels from one medium to another of less density, fragmentation of tissue occurs. The pulmonary alveoli and the gas-filled intestinal viscera, both structures with fluid/air interfaces, are particularly susceptible to damage by blast waves.

Management of Blast Injuries.—The most vulnerable organs to the blast wave are the ear drums, lungs and gastrointestinal tract. Casualties may present in a dramatic form with traumatic amputation, the hæmorrhage from which can only be controlled by a tourniquet. The deafness of the victims of blast, due to rupture of the tympanic membrane, makes communication with them difficult. They must be carefully examined for many have multiple wounds. The management of these wounds is in no way different from that of missile injuries. Very often the soft tissues are heavily contaminated with organic debris due to the force of the explosion opening up tissue planes and driving in foreign bodies at some distance from the wound. Some cases are associated with multiple small wounds affecting a

limb. It is not always practical to explore fully every wound. The deeper wounds should have priority of management due to the more serious consequences of infection. In many blast injuries one cannot be sure of complete wound excision and therefore it becomes imperative that all blast wounds should be left open at the end of initial operation and delayed primary suture performed four to seven days later.

Blast Injury to the Auditory System.—Blast damages the hearing in three ways. There may be rupture of the tympanic membrane, dislocation of the ossicles or damage to the inner ear. The last is sometimes accompanied by permanent deafness.

Blast Injury to the Lung.—Occasionally post-traumatic pulmonary insufficiency develops with intra-alveolar œdema and hæmorrhage. Usually there is a delay of twelve to twenty-four hours before the patient develops a cough accompanied by mild hæmoptysis, dyspnoea or a feeling of general apprehension. Blood gas analysis will show arterial hypoxia and a raised pCO_2. A chest x-ray may show a bilateral opaque, fluffy appearance mostly around the hilum of each lung. *Management.* These cases will require the administration of oxygen and steroids. Blood gas analysis may indicate the need for assisted positive pressure ventilation with a positive expiratory end-pressure.

All victims of blast should therefore be carefully watched during the initial forty-eight hours for late onset pulmonary insufficiency.

Blast Injury of the Gastrointestinal System.—Injury to the gas-filled viscera is more common in underwater explosions than air blasts. Perforation of stomach or cæcum may occur. The clinical presentation is one of increasing abdominal pain accompanied by signs of peritonism and often gas under the diaphragm. Urgent laparotomy is indicated.

Blast Wounds to the Eye.—So often one fails to notice damage to an eye at the time of initial examination. It is important to appreciate that the conjunctival hæmorrhage, accompanying a blast injury, may herald a more serious underlying problem of penetration of the globe by foreign bodies. The pupil must be carefully examined and any distortion of the iris investigated.

Other factors which increase the morbidity and mortality following bomb-blast injuries are the associated chemical and thermal burns, and the inhalation of toxic gases and smoke.

CHAPTER 3

SEPSIS, ANTIMICROBIAL THERAPY, SCARS

SEPSIS

INFECTION is the invasion of the body by pathogenic micro-organisms. For this to happen, a sufficient number of pathogens must enter the tissues, overcome the patient's resistance, and multiply. *General factors* that lower resistance include uncontrolled diabetes, steroids and immunosuppressive therapy. *Local factors* include hæmatoma formation, crushing of tissues at injury or operation, fractures and foreign bodies.

The Principal Organisms of Sepsis.—See Table over page.

Other Organisms of Sepsis

Salmonella typhi and **Salmonella paratyphi** cause enteric fever; the organisms may persist in the biliary passages or kidney and the patient becomes a carrier. These and other salmonellæ sometimes cause septicæmia, osteitis and septic arthritis.

Clostridia are anaerobic sporing bacilli, inhabitants of intestine and found in soil. Some are non-pathogenic. *Cl. tetani* causes tetanus. *Cl. perfringens* (*Cl. welchii*) is the principal cause of gas-gangrene but is also commonly found in wounds without serious harm.

Cl. difficile according to recent work, appears to proliferate in intestine during antibiotic treatment, especially after surgery. This may cause post-operative diarrhœa and pseudomembraneous colitis.

Other important organisms include *Mycobacterium tuberculosis* (p. 37). *M. leprae* (p. 40), Gonococcus and *T. pallidum* (p. 44), Actinomyces (p. 36). Candida (p. 36).

ANTIMICROBIAL THERAPY

Antimicrobial drugs damage microbes without harming the tissues (though some may be toxic, especially if dosage is wrong). Some, the **antibiotics**, are produced by organisms. Others are synthetic (e.g. sulphonamides).

Some drugs are bactericidal (e.g. penicillins and aminoglycosides) and kill susceptible bacteria. Others (e.g. tetracycline, erythromycin) are bacteriostatic and mainly inhibit bacterial growth; however, they are also effective when leucocytes can enter the lesion and complete the destruction of the microbe.

Penicillin was the first therapeutically useful antibiotic and is still the most valuable. It was discovered by Fleming in 1928 but not used clinically until Florey and Chain produced a reliable preparation in 1941. It acts best on multiplying bacteria and hence may be antagonised by bacteriostatic drugs. Of the many modifications now available Benzylpenicillin (Penicillin G) is still the best drug against many Gram-positive pathogens e.g. *Str. pyogenes*, pneumococci, clostridia and non-penicillinase-producing staphylococci (nowadays a minority). It usually is the drug of choice for gonorrhœa (though some strains are now resistant), and syphilis. Procaine penicillin is a slow-release compound of benzylpenicillin. Penicillin sensitive bacteria rarely acquire resistance during treatment but may be replaced by resistant organisms. Other penicillins include *Penicillin V*, acid-stable and given orally for mild infections; *ampicillin* and *amoxycillin* (a better absorbed form) which act on some Gram-negative bacilli (e.g. most strains of *E. coli* and *H. influenzæ*) as well as on Gram-positive organisms. *Carbenicillin* in high dosage acts on some Proteus and

Howard Walter Florey (Lord Florey), 1898–1968. Professor of Pathology, Oxford, England (1935–1962).
Sir Alexander Fleming, 1881–1955. Professor of Bacteriology, St. Mary's Hospital, London (1928–1948).
Ernst Boris Chain, 1906– . Professor of Biochemistry, University of London (Imperial College), London.
Daniel Elmer Salmon, 1850–1914. American Pathologist who isolated the first member of the group of organisms, now bearing his name, in 1886.

PRINCIPAL ORGANISMS OF SEPSIS

GRAM-POSITIVE COCCI

Staphylococcus aureus	*Habitat and Spread*	*Principal Infections*	*Antibiotic Sensitivity*
Gram-positive cocci in clusters; forms coagulase (fig. 14).	Lives in nostrils of 30% of population and sometimes in skin. Present in infected discharges. **Spread** by contact, on hands etc. Airborne spread by skin squames and garment dust.	Boils, styes, septic hands, breast abscess, wound infection, osteomyelitis, septicæmia, post-operative pneumonia.	**Outside hospital** most strains now form penicillinase and are penicillin-resistant. **In hospital,** many are resistant also to several other drugs. Most are still sensitive to flucloxacillin and cloxacillin.
Staph. epidermidis (Staph. albus) and other coagulase-negative staphylococci, normally non-pathogenic, can infect damaged tissue.	Nasal and skin carriers are very common. **Spread** same as *Staph. aureus*, but more numerous in air of rooms.	Minor skin sepsis and stitch abscess; cause dangerous infection of prosthetic heart valves and hip replacements.	As *Staph. aureus*. Multi-resistant strains are common.
Staph. saprophyticus	Found on skin though not common.	Urinary tract infection in young women.	Usually sensitive to sulphonamide and ampicillin.
Streptococcus (fig. 15). Cocci in chains; many varieties. Hæmolytic streptococci are grouped serologically by antigenic content.			
Str. pyogenes (Group A)	Lives in pharynx of 5–10% of population. **Spread** by contact, and airborne from respiratory tract and wound discharges.	Tonsillitis, otitis, scarlet fever. Dangerous in wounds, cellulitis, septicæmia. Infections tend to spread rapidly in tissues.	Sensitive to penicillins and erythromycin. Resistant to aminoglycosides and often to tetracycline.
Str. pneumoniæ (Pneumococcus). A capsulated diplococcus.	Lives in nasopharynx of many normal people.	Post-operative and other pneumonias, otitis media and sinusitis, septicæmia. In young girls, occasionally cause peritonitis perhaps entering *via* the Fallopian Tube.	As *Str. pyogenes*, but penicillin resistance has appeared in some countries.
Str. fæcalis and anaerobic streptococci.	Large bowel.	Abdominal sepsis, usually mixed with other organisms.	*Str. fæcalis* is usually moderately sensitive to ampicillin and cephalosporins. Anaerobic strains are sensitive to penicillins, erythromycin and metronidazole.

GRAM-NEGATIVE BACILLI (Contain endotoxins that accentuate pyrexia and shock in severe infections).

Bacteroides Anaerobic Gram-negative rods.	The predominant normal organisms of the colon, greatly outnumbering all others.	Peritonitis, intra-abdominal and pelvic sepsis (usually accompanied by aerobic intestinal bacteria).	Sensitive to metronidazole and usually chloramphenicol. Resistant to penicillins and aminoglycosides.
Escherichia coli Aerobic rods. (fig. 16).	Normal inhabitant of large bowel.	Wounds, especially abdominal; intra-abdominal sepsis, cholangitis etc. The commonest cause of urinary infection.	Some are sensitive to ampicillin and sulphonamide, most are sensitive to gentamicin. Hospital strains can become resistant to many drugs.
Klebsiella	As *E. coli*.	As *E. coli*.	As *E. coli*., but ampicillin resistant.
Proteus	Large bowel.	As *E. coli*. Splits urea into NH_3 making urine alkaline causing phosphate deposits.	Similar to *E. coli*.
Pseudomonas	Large bowel. Often colonises moist lesions, its principal habitat. Also found in contaminated lotions, sinks etc.	An opportunist that attacks damaged tissues, burns, tracheostomy wounds, urinary tract.	Usually sensitive to gentamicin and carbenicillin.

Other bacteria that often are important in surgical patients include:—
Hæmophilus influenzæ, a Gram-negative bacillus, often present in the healthy nasopharynx. With pneumococci, are the commonest causes of post-operative chest infection. Usually sensitive to ampicillin, tetracycline and co-trimoxazole.

Some Gram-negative bacilli, pathogenic in the intestine, cause infections that may be confused with surgical causes of pain and diarrhoea. They include *Salmonella* species and Campylobacter, that cause enteritis, and *Shigellae* that cause dysentery. Some cases of ileitis and mesenteric adenitis are caused by *Yersinia*.

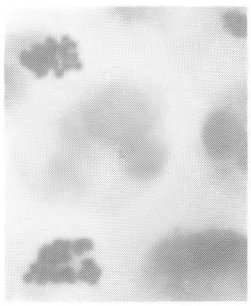


FIG. 14.—Streptococci.

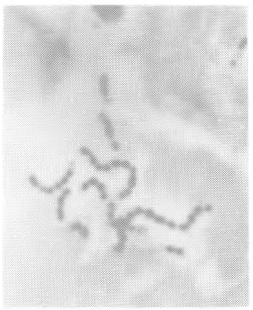


FIG. 15.—Staplylococci.

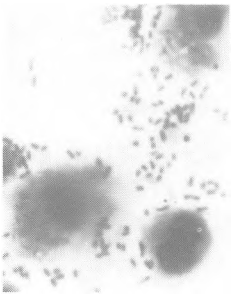


FIG. 16.—E. coli.

FIGS. 14, 15 and 16.—Principal Organisms of Sepsis.

Pseudomonas strains. It is very expensive. Like penicillin G, all these derivatives are useless against penicillinase-producing bacteria, staphylococci and others. *Cloxacillin* and *flucloxacillin* are insensitive to penicillinase and hence effective against most penicillin-resistant staphylococci. This is their only valid use. The main disadvantage of penicillins is their tendency to cause allergic reactions, e.g. rashes and occasionally anaphylaxis. Since they contain sodium, very large doses may cause sodium overload.

Cephalosporins act like penicillins. There are several modifications, some very expensive. They are best reserved for use in individual cases, after discussion with the bacteriologist.

Aminoglycosides, given only by injection and excreted by the kidney, include *gentamicin* and *tobramycin*, very similar in action. *Amikacin* is active against some bacteria that have become resistant to gentamicin; its use should be confined to these. Aminoglycosides are often effective in septicæmia and other life-threatening infections by various aerobic bacteria including most Gram-negative bacilli and most staphylococci (but *not* anaerobes nor streptococci). The chief problem in using aminoglycosides is that their therapeutic and toxic levels are quite close. Too high blood levels may damage the 8th cranial nerve and the kidney. In order to avoid toxicity, and the even greater risk of inadequately treating a dangerous infection, dosage should be related to body weight, age and renal function (preferably in consultation with the bacteriologist) and blood levels should be estimated periodically. Particular care is needed in the elderly and in renal insufficiency.

The tetracyclines are broad-spectrum antibiotics but many bacteria have acquired resistance to them. They are still valuable in *H. influenzæ* chest infection and in a variety of infections including lymphogranuloma venereum and non-specific urethritis. They are *dangerous in renal insufficiency* and should *never* be given in pregnancy nor to children under 8.

Chloramphenicol may dangerously damage the bone marrow and should be used only for some grave infections for which it is considered to be the most effective drug (e.g. typhoid fever).

Fusidic acid is a valuable drug for serious staphylococcal infections. Resistance develops readily and, to prevent this, it should always be given with another appropriate antibiotic (e.g. flucloxacillin or erythromycin).

Erythromycin is a valuable antibiotic against several Gram-positive bacteria and is a good substitute for penicillin in allergic patients. Erythromycin estolate may damage the liver and should not be used. Other preparations are non-toxic.

Clindamycin is highly active against similar organisms and also anaerobes but may cause pseudomembraneous colitis and has been largely replaced by metronidazole in the treatment of bacteroides infections.

Sulphonamides (e.g. Sulphadimidine) inhibit growth of many bacteria but are easily neutralised by inhibitors in blood and tissue. They are most useful in *E. coli* urinary infections (many non-hospital strains are still sensitive). Toxic effects sometimes occur; the most important are rashes, leukopenia and fever.

Co-trimoxazole ('Septrin', 'Bactrim') contains trimethoprim and sulphamethoxazole. It is used for several systemic infections as well as urinary infections.

Hans Christian Joachim Gram, 1853–1938. Professor of Pharmacology (1891–1900) and Professor of Medicine (1900–1923), Copenhagen. Described his method of staining bacteria in 1884 while engaged in post-graduate study in Berlin.

Nitrofurantoin (' Furadantin ' etc.) is given for *E. coli* urinary infection. (Avoid in renal insufficiency.) **Nalidixic acid** (' Negram ') is used similarly, though resistance readily develops.

Metronidazole (' Flagyl ') an anti-protozoal drug (amoebiasis, trichomoniasis and giardiasis), is active also against *anaerobic* bacteria. It is the drug of choice for bacteroides.

Drugs for tuberculosis and leprosy—see p. 37 and p. 41.

USE AND SELECTION OF ANTIMICROBIAL DRUGS

All these drugs have disadvantages or dangers and should be used with discretion and only when the clinical state of the patient requires them. They should not be used for localised infections that respond to surgical treatment e.g. superficial abscesses, and discharging wounds without cellulitis. If time permits, chemotherapy should be started after the organism has been identified and its sensitivity determined. However, in serious acute infections, treatment must start without delay, having first taken relevant specimens. The initial choice of drug must be based on the clinical diagnosis, and the bacteriologist's advice may be sought. Completely blind treatment may delay the diagnosis and do more harm than good. New drugs should be used with caution.[1] In general, if there is no improvement in a few days, the treatment should be stopped or changed. Treatment for more than 7–10 days is usually only needed in special circumstances e.g. tuberculosis.

Prophylactic antibiotics should *not* be given routinely but there are several situations in which chemoprophylaxis is advisable and sometimes even mandatory. These include penicillin to prevent gas-gangrene after thigh amputation in peripheral vascular disease, and also tetanus (p. 29); and metronidazole and gentamicin in colo-rectal surgery, to prevent post-operative sepsis by bacteroides and coliform bacilli. Prostatectomy or urethral instrumentation in an *already infected* urinary tract may cause Gram-negative septicæmia which usually can be prevented by an appropriate antibiotic. Protection is usually only conferred while the drugs are present in blood and tissues during operation and for a few hours afterwards. Hence, administration should start at premedication and usually continue for only a day or two afterwards (a week in thigh amputation). Other circumstances in which correctly planned chemoprophylaxis is usually employed include open heart surgery and total hip replacement operations. In general, the drugs are chosen according to the known or probable nature of the organisms likely to cause infection.

Administration of Antibiotics

Oral. Even with absorbable drugs, absorption may be incomplete, especially in very ill patients.

Intramuscular. The best site is usually the antero-lateral aspect of the thigh. If the buttock is used, it should be in the upper outer quadrant to avoid damaging the sciatic nerve (fig. 17). In shocked patients, absorption is uncertain and intravenous dosage is better as it also is when large volumes are injected.

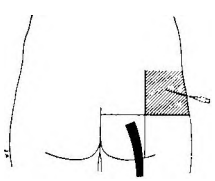


FIG. 17. — 'Safe' area for intramuscular injection.

Intravenous. Intermittent administration is usually more effective than continuous administration of the same daily dose. Several antibiotics are incompatible with others and also with other drugs in intravenous fluids. The pharmacist's advice and manufacturers' instructions should be followed.

HOSPITAL INFECTION

Patients may be infected by their own organisms (self-infection) or by organisms from other people (cross-infection). Cross-infecting organisms are often resistant to several antibiotics, especially in wards in which antibiotics are often used. The

[1] Sir Robert Hutchison, 1871–1960, Physician, London Hospital, used to warn that ' It is always well, before handing the cup of knowledge to the young, to wait until the froth has settled '.

principal cross-infecting organisms are staphylococci and Gram-negative bacilli. These may merely colonise discharging wounds and other lesions without necessarily causing sepsis. Wound discharges, infected urine, and fæces are important sources of cross-infection. Other sources, especially of staphylococci, include superficial lesions such as eczema, bedsores and the noses of carriers among patients and staff.

Staph. aureus causes many post-operative complications such as wound sepsis, bronchopneumonia and staphylococcal enterocolitis (now fortunately rare). Coagulase-negative staphylococci (*Staph. epidermidis* etc.), normally harmless, are dangerous causes of infection after open heart surgery and hip replacement operations.

Gram-negative bacilli.—Hospital infections by *E. coli*, *Klebsiella*, *Proteus* and *Pseudomonas* have increased recently because some other organisms have been diminished by antibiotics. They are common in abdominal wounds and burns, and in the sputum after antibiotic treatment of post-operative chest infection by *H. influenzæ* and pneumococci. Their presence in sputum is due more often to colonisation than infection and therefore does not necessarily require a change of antibiotic. These organisms are the commonest invaders of the urinary tract after catheterisation or operation, especially when introduced by inadequately disinfected cystoscopes, inexpert catheterisation and open draining indwelling catheters.

Clostridia. Tetanus spores are often present in soil and dust. Tetanus is an important danger from faulty heat sterilisers. Gas gangrene (p. 33) may be caused by the patient's own intestinal clostridia after lower limb amputations in diabetic and arteriosclerotic patients.

Hepatitis virus see Chap. 45.

Prevention of Hospital Infection

Prevention consists of reducing the *sources* of pathogens, restricting their *spread* and *protecting* susceptible tissues from infection. Proliferation of drug-resistant bacteria is reduced by avoiding unnecessary antibiotics and limiting the duration of treatment when they are given. Prophylactic chemotherapy, when justified, should usually be for short periods (p. 20). Local antibiotics are especially likely to breed resistant bacteria, and, in any case, are of limited value. Spread of infection is minimised by care when dressing contaminated wounds. Patients with drug-resistant staphylococcal infections of open lesions should be barrier-nursed. Infected urine is an important source of cross-infection. The spread of pathogens is restricted by general hygienic measures, including the avoidance of overcrowding and satisfactory ventilation. Hands of ward staff should be washed well with soap and water and dried with disposable towels (wet hands are dangerous). Contact with dangerous sources of infection e.g. discharging wounds, infected urine etc. should be avoided by wearing disposable gloves. Bedding should be disinfected by heat-laundering and baths by a cleaning agent containing hypochlorite (e.g. ' Vim ' with added bleach). Lotions and dilute aqueous disinfectants may become contaminated by Gram-negative bacilli and should only be used from single-use sealed containers issued sterile to wards. Bacteria should be excluded from susceptible tissues. Post-operative wounds should be left untouched as much as possible and a meticulous non-touch technique used when removing stitches; before removal, the skin and exposed portions of stitches should be disinfected with 70% alcohol.

The correct *heat-sterilisation of instruments and dressings* is sufficiently complicated to deserve skilled supervision in central supply departments and operating theatres. Unbleached cotton wool, sometimes used to pad splints may contain tetanus spores and should never be used unless satisfactorily sterilised. Instruments that are damaged at high temperature present special problems. Ethylene-oxide, used to sterilise heat-sensitive instruments, is only reliable if supervised by skilled staff, preferably in centralised departments. Cystoscopes may usually be pasteurised at 75° C in steam at sub-atmospheric pressure in special autoclaves; spores are not killed but are rarely important in the urinary tract. A chemical (glutaraldehyde) may also be used.

At operation the risk of infection is reduced by disinfecting the patient's skin with 1% iodine or 0·5% chlorhexidine in 70% alcohol; the latter is less likely to cause sensitisation. The maximum effect is achieved by allowing the preparation to dry

on the skin. The risk of infection from the surgeon's skin is reduced by hand-washing with hexachlorophane, povidone iodine or chlorhexidine and by rejecting pricked gloves. Prior to the incision the application of an adhesive film (Opsite[1]), through which the incision is made, helps to prevent contamination during operation from the surrounding skin, and fixes the drapes without the use of towel clips in the patients skin. Application of a fresh piece of film serves as an excellent dressing for clean stitched and non-oozing wounds (fig. 114).

Staff with septic lesions or sore throats should not enter the theatre. Air contamination is minimised by restricting unnecessary movement of staff and entry of unnecessary visitors and by replacing outer clothes by freshly laundered theatre garments and overshoes. Masks should *never* be handled while worn. Airborne bacteria may be further reduced by ventilation systems giving frequent changes of filtered air at a positive pressure.

Finally, since even the best precautions cannot exclude all bacteria from wounds, it is most important not to reduce resistance of tissues by rough handling, unnecessary drainage or inadequate hæmostasis.

Wound Infection.—On inspection there is redness and swelling of the wound edges. Small abscesses may appear in relation to the stitches. Infection may then spread into the surrounding subcutaneous tissues (cellulitis p. 27), and collections of pus may form in the layers of the wound. At this stage the patient is usually toxic and the temperature is raised. If infection is particularly virulent, invasion of the blood stream (septicæmia p. 28) may follow.

Treatment. 1. *Early stages.*—If infection is treated before pus is formed it may be possible to halt it by systemic antibiotic therapy. Any stitches which appear to be tight or causing tension should be removed.

2. *Stitch Abscess Formation.*—Any affected stitches should be removed.

3. *Deeper Pus Formation.*—The removal of stitches may result in sufficient separation of the edges of the wound to achieve satisfactory drainage, but otherwise the patient should be returned to the operating theatre, the wound opened and drained and allowed to heal by second intention. Occasionally, when all is beginning to heal cleanly, secondary suture or skin grafting is performed.

A Sharp Lesson from History.—It has often been said, and truly, that the practice of surgery can be divided into surgery before Lister and surgery after Lister (fig. 18). The meaning of this statement is to be found in an understanding of certain aspects of his research, by animal and human experiments. Armed with a splendid microscope, for his father had perfected the achromatic lens, and working all hours after a day's work in hospital, he was amongst the first to describe accurately the early vascular changes of inflammation[2] due to injury, and how increased blood flow (hyperæmia) was followed by intravascular coagulation. Had he prolonged his 'glorious' nights and got no sleep at all, he would have antedated the discovery of the cellular component of inflammation (migration of leucocytes by Cohnheim and phagocytosis by Metchnikoff).

FIG. 18.—Joseph Lister, FRCS, aged 28. 'The father of modern surgery' 'The man who made surgery safe'.

[1] *Opsite.* T. J. Smith and Nephew Ltd., Welwyn Garden City, England.

[2] For this work he was elected F.R.S. in 1860.

Julius Cohnheim, 1839–1884. Professor of Pathology, Leipzig.
Elie Metchnikoff, 1845–1916. A Russian biologist who worked in France.

Lister was puzzled and perplexed by inflammation which was then running amok in patients with wounds involving the skin, especially those with compound fractures. The sepsis and fulminating putrefaction were terrible indeed. Recovery after such an injury or an operation was considered by many surgeons to be a matter of luck. There was a mortality of up to 80% after amputation, reaching almost 100% in those wounded on the battle-fields of those days.

When his attention was drawn to the work of Louis Pasteur on fermentation indicating the presence of ' microbes in the air ', Lister immediately guessed why inflammation got so out of hand in surgical patients. By incorporating carbolic acid, already known as a disinfectant of sewage, in the dressing of wounds of hitherto unfavourable cases, sepsis was overcome and prevented. This work led him, in spite of disbelief by others in Pasteur's theory and his own clinical hypothesis and application, to an enunciation of his antiseptic principle—that every procedure of surgical technique should be against sepsis, *i.e.* anti-sepsis. Extending the use of carbolic acid and other chemicals, such as mercury perchloride and iodine, he produced, in effect, sterile dressings, catgut and silk ligatures, and metallic means for the open reduction of fractures. A carbolic acid spray was employed temporarily to act as another barrier to those (then) theoretical germs.

As a result, surgery was released from the shackles of sepsis and immediately most operations commonly practised today were thought out and put into practice. Other methods, based on the use of heat (e.g. the autoclave) were introduced to prevent infection during operation. Strictly speaking, the word ' asepsis ' was an unnecessary innovation. Lister had demonstrated that safe surgery was dependent upon surgeons committing themselves and whatever techniques they devised to being anti-sepsis.

The moral of this story is obvious. If we survey the everwidening surgical scene of today it is plain that the battle against sepsis (antisepsis) must continue. In the midst of successes we see occasional grim reminders that patients can still fester and die from whole-body inflammation, namely septicæmia, vascular paralysis and a disseminated form of the intravascular coagulation which Lister had observed in his early researches.

INCISIONS

The object of a surgical incision is to expose the part operated on to best advantage, and this should be combined with a desire to finish with a scar which is as good and as inconspicuous as possible. The creases and wrinkles in the skin are parallel with the lines of skin tension and it is in accordance with these lines that incisions should be made.

Incisions should not be made directly across skin creases unless there is some over-riding reason as the resulting scar will become hypertrophic. In no circumstances should an incision be made directly across the crease in the flexor surface of a joint. Adequate exposure can be obtained by making incisions ' S ' or ' Z ' shaped if a single straight incision will not suffice.

Louis Pasteur, 1822–1895. Chemist and Bacteriologist. Professor of Chemistry, Sorbonne, Paris.

Confusion has arisen in the past because of the interpretation of the significance of Langer's lines. These lines which were worked out by Langer in 1862, are associated with the distribution of the collagen and elastic fibres in the skin, and are not of importance with regard to skin incisions.

SCARS

Because of the final effect on function and appearance, it is the surgeon's aim to achieve a scar which is of good colour, narrow, soft and not contracted. All scars pass through a natural sequence of stages:—

Stage 1. 0–4 weeks. The scar is fine, soft, not contracted, not strong.
Stage 2. 4–12 weeks. The scar becomes red, hard, thick and strong. It tends to contract.
Stage 3. 12–40 weeks. The scar gradually becomes soft, supple, white and tends to relax.

The rate and extent to which these changes occur vary with (*a*) the position of the scar on the body, (*b*) the direction of the scar with regard to the lines of skin tension, (*c*) the age of the patient, (*d*) the race.

(*a*) *Position on the Body.*—The worst position on the body for scars is over the sternum. Scars in this area very regularly become severely hypertrophic and even truly keloidal (fig. 19). The region over the shoulder is also a poor one for making scars, as is the back.

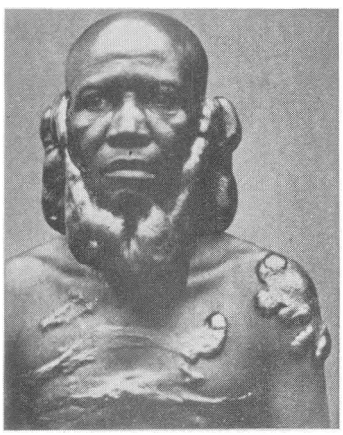


FIG. 19.—Extensive keloid in a West African. (*C. Bowesman, F.R.C.S., Kumasi, Ghana.*)

(*b*) *Direction with regard to lines of skin tension.*—Scars are better when they are in the lines of skin tension, i.e. lying in or parallel with the skin folds. Scars which directly cross skin folds, e.g. particularly the flexion creases of the joints, should be avoided.

(*c*) *Age.*—The age of the patient makes a considerable difference in the reaction of scars. The best scars are obtained in very old people with fine wrinkled skin. The skin scars in these people very quickly settle down and 9 months later may be almost invisible. Another favourable age is the first year of life, but after that the tendency to scar formation becomes more marked, and the worst period for scars likely to contract is in older children.

(*d*) *Race.*—The more highly pigmented the person the worse the scars will be.

Hypertrophic Scars.—This type of scar is common in young people and particularly after injuries such as burns (fig. 20). It is so common as to be considered the normal pattern of scar formation. Both the intensity and the duration of the active phase of scar formation are increased. However, even in cases where severe hypertrophy occurs the scar does not become worse after 12 weeks. By this time, the scar is very thick, red and often itchy, and this condition then persists in this severe state for a further 3 to 6 months. After this time, it gradually regresses. *Compression treatment.*—Fashioned elastic appliances are particularly useful in flattening and maturing these scars.

Karl Langer, 1819–1887. Professor of Anatomy, Vienna.

Stockings, armlets, gloves, body pieces, and face and neck helmets can be made.[1]

Keloid[2] (figs. 19 and 21).—True keloid in white-skinned people is a rare condition, and the only common site is over the sternum. It is said that the true keloid spreads into normal tissue not affected by the original injury or operation. However, in

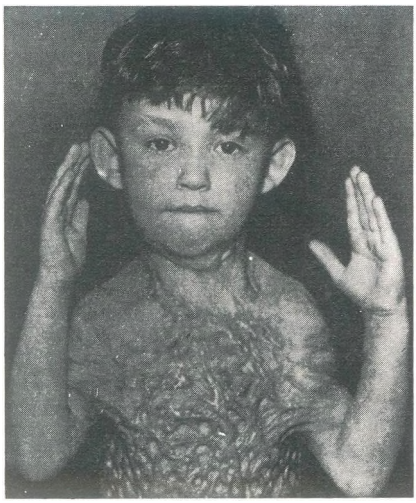


FIG. 20.—Contractures following burns (Children's Burns Unit, Guy's Hospital, London). (*The late Patrick Clarkson, F.R.C.S.*)

FIG. 21.—An unusual keloid, ten years after a laparotomy.

many scars this is difficult to determine, and the certain distinction between the true keloid and the hypertrophic scar is found not in the appearance, but in the time scale. The hypertrophic scar never gets worse after 6 months, but a true keloid continues to get worse even after a year, and some may even progress for 5 to 10 years. It would appear that the maturation and stabilisation of the collagen fibrils is inhibited (p. 3). Treatment is extremely difficult. Excision and resuture are certain to be followed by recurrence and although x-ray treatment was used in the hope that this would prevent recurrence the results of this are uncertain. Improved results have been obtained by shaving away the excess tissue, taking care not to carry the incision into normal tissue at any point and then resurfacing the area by a thin skin graft. Some excellent results have also been obtained by the injection of steroid preparations.

[1] Tubigrip pressure garments by Seton®, Tubiton House, Oldham, England, or Jobst anti burnscar™ supports by Jobst®, Toledo, Ohio, U.S.A.

[2] Keloid = Like a claw.

CHAPTER 4

ACUTE INFECTIONS

ACUTE ABSCESS

An abscess is a collection of pus. Bacteria which cause pus formation (pyogenic organisms) reach the infected area by one of three routes:

(i) Direct infection from without, e.g. penetrating wounds.
(ii) Local extension from some adjacent focus of infection.
(iii) Blood-stream (hæmatogenous) or lymphatic vessels.

The bacteria, having entered the tissues, multiply and cause inflammation. The area around the abscess is infiltrated with leucocytes and bacteria (the pyogenic membrane).

Pus.—Polymorphonuclear leucocytes contain a proteolytic enzyme which causes liquefaction of the tissues into pus, which is composed of dead leucocytes and bacteria. The tension in the abscess rises owing to the exudation of plasma and it may spread along the paths of least resistance to the surface of the body, or to a hollow viscus where the pus is eventually discharged.

Symptoms.—The patient feels ill, the degree depending upon the size of the abscess, the virulence of the organism, and the tension within the cavity. Throbbing pain is characteristic of suppuration, the pain becoming more severe if the affected part is dependent, e.g. an infected finger.

Signs.—(*a*) GENERAL.—The temperature is elevated. In severe cases, with bacteræmia or septicæmia, rigors may occur.

(*b*) LOCAL.—The five classical[1] local signs of inflammation are due to hyperæmia and inflammatory exudate (fig. 22):

1. *Heat*—the inflamed area feels warmer than the surrounding tissues.
2. *Redness* of the skin over the inflamed area. Both this sign and the previous one are due to hyperæmia.
3. *Tenderness*, due to the pressure of exudate on the surrounding nerves. If the exudate is under tension, e.g. a furuncle (boil) of the ear, pain is severe, whereas lax tissues (e.g. scrotum) may swell enormously with little discomfort.
4. *Swelling.*
5. *Loss of function*—an inflamed tissue does not perform its physiological function.

The degree to which these signs are evident depends on the extent of the inflammation and its proximity to the surface. The swelling is at first brawny

[1] Aulus Aurelius Cornelius Celsus, 25 B.C.–A.D. 50. A Roman medical writer described the first four of these signs as Calor (heat), Rubor (redness), Dolor (pain), and Tumor (swelling). The fifth sign Functio Læsa (loss of function) was added by Galen, A.D. 130–200, Roman Physician.

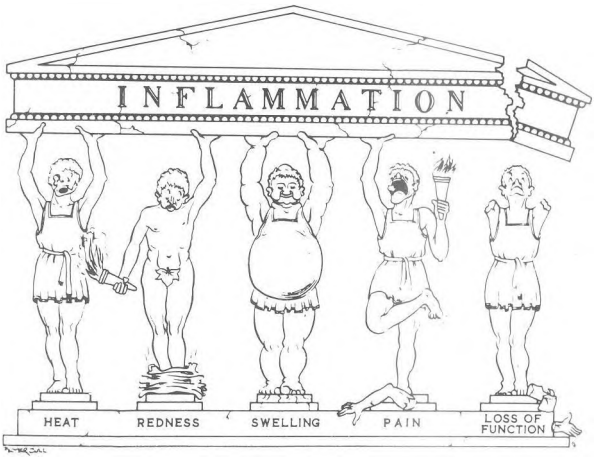


FIG. 22.—The Greeks had a word for it! *(With permission of Professors W. G. Spector and D. A. Willoughby. Artist, Mr. P. Cull.)*

and œdematous; later softening and fluctuation are manifest (fig. 23). In some cases increasing œdema is characteristic of deep pus, as in acute mastitis. If untreated, an abscess tends to point; the skin or membrane covering it gives way and the contents are discharged, usually with relief.

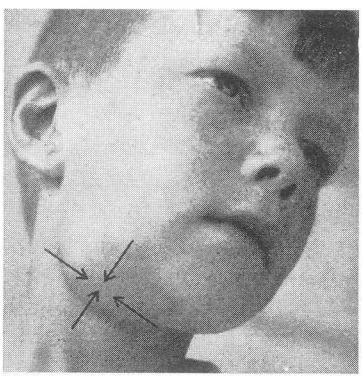


FIG. 23.—An abscess. The arrows indicate fluctuation in two planes.

Prevention.—An abscess can sometimes be aborted by antibiotics, e.g. tetracycline, co-trimoxazole (Septrin), rest and, if possible, elevation of the affected part.

Treatment.—As soon as pus is diagnosed, the abscess must be incised and dependent drainage instituted. In regions where incisions are fraught with danger to important anatomical structures, as in the neck or axilla, a modification of Hilton's[1] method should be used. This consists of incising the skin and superficial fascia, and opening the abscess by thrusting a pair of sinus forceps or a hæmostat into the cavity. By separating the blades, a sufficiently large opening can be made to insert, if necessary, a finger in order to convert loculi into a single cavity, followed by a drainage tube.

Pus from an abscess must be cultured so as to determine the sensitivity of the organisms to antibiotics.

Errors in treatment.—1. Once pus has formed antibiotics alone will not suffice. Indeed, the inflammation continues to smoulder and a hard, lump forms, called an ' antibioma '. In certain areas, e.g. the breast, it becomes difficult clinically to decide whether such a lump is malignant or inflammatory.

2. Incising an aneurysm in mistake for an abscess. It has been done on many occasions!

CELLULITIS

Cellulitis is the term given to a spreading inflammatory exudate along subcutaneous and fascial planes.

[1] *John Hilton, 1804–1878. Surgeon, Guy's Hospital, London.*

Streptococcus pyogenes, the commonest cause of severe cellulitis, may enter the tissues through an accidental wound, trivial in nature, such as a graze or scratch, or possibly as the result of an operation. If the general resistance of the patient is undermined by such conditions as diabetes, cellulitis is likely to spread rapidly and may lead to septicæmia.

The clinical condition depends upon the virulence of the organism and the extent of infection. Redness and itching or stiffness commence at the site of inoculation, to be followed by tenderness and swelling. The skin becomes shiny in appearance. Local gangrene may occur. The appearance of fine creases or wrinkles in the skin heralds the resolution of the condition.

Treatment consists of rest, elevation of the part, and above all the use of the appropriate antibiotic. Diabetes, if present, should be treated.

CELLULITIS IN SPECIAL SITUATIONS

Scalp.—This is due to infection of the sub-aponeurotic layer of areolar tissue. Pus may extend to the attachment of the epicranial aponeurosis, so that the whole scalp is lifted off the calvarium. Necrosis of bone, or thrombosis of emissary veins spreading to intracranial sinuses, may follow. Early incisions, parallel to the arteries, are necessary when pus is suspected. The condition may present as a Pott's puffy tumour (Chap. 26).

Orbit.—This follows wounds or spread of infection from air sinuses in the vicinity (Chap. 30). Proptosis and impairment of ocular movements follow, and infection may spread to the meninges, or thrombosis extend along the ophthalmic veins to the cavernous sinus (Chap. 30). The eyeball itself occasionally becomes infected (panophthalmitis). Owing to the risk of cellulitis, all wounds of the orbit demand careful attention. If infection supervenes, drainage is provided by incisions in the eyelids or conjunctival fornix. Panophthalmitis is treated by evisceration of the eye; this procedure is safer than excision, which is liable to be followed by meningitis from infection extending along the open sheath of the optic nerve.

Neck.—A complication of wounds, tonsillitis, or mastoiditis. Ludwig's angina is a term applied to submaxillary cellulitis (Chap. 34). The two main dangers of cervical cellulitis are œdema of the glottis with possible asphyxia, and mediastinitis.

Pelvic Cellular Tissue.—The common cause is a lateral tear of the cervix during parturition. Inflammation may extend upwards in the broad ligament so that an indurated swelling appears above the inguinal (Poupart's) ligament (parametritis, Chap. 51). Ultra short-wave therapy and the appropriate antibiotic will usually give relief. If the swelling appears above Poupart's ligament and deep œdema or softening is detected, the abscess is incised and drained extraperitoneally.

BACTERÆMIA, SEPTICÆMIA AND PYÆMIA

Bacteræmia and Septicæmia are terms used to describe the presence of organisms in the blood as diagnosed by blood culture.

François Poupart, 1661–1708. Surgeon, Hôtel Dieu, Paris.

The clinical features are those of severe infection and shock, sometimes preceded by a rigor. The pyrexia is commonly intermittent, and rigors may continue. Jaundice is due to liver damage. Acute tubular necrosis of the kidneys may occur. Peripheral circulatory paralysis of vasomotor tone and disseminated intravascular coagulation of blood forebode a fatal outcome.

Treatment must be prompt and aggressive. The causative focus must be found and treated, surgically if necessary. A blood culture is taken and adequate doses of broad spectrum antibiotics given, these being changed if necessary when the sensitivity of the organism is known. Transfusion with blood plasma expanders, and hydrocortisone injections are mandatory.

Pyæmia is due to the circulation in the blood-stream of infective emboli composed of masses of organisms, vegetations or infected clot.

Clinically, pyæmia is characterised by rigors, an intermittent fever and the formation of abscesses. Abscesses occur in any part of the body. Joints are occasionally affected, and may become quietly disorganised. Death may follow abscess formation in vital structures, e.g. heart or brain.

The *treatment* of pyæmia consists in endeavouring to prevent further emboli from reaching the blood-stream. Thus, if the condition is caused by thrombosis of the lateral intracranial venous sinus, then ligation and division of the internal jugular vein may interrupt the stream of emboli. Otherwise, abscesses are dealt with as they occur, and general treatment is instituted as for septicæmia. Portal pyæmia is discussed in Chap. 45.

Lymphatic Effects in Acute Infections

It should be remembered that most local acute infections give rise to lymphangitis and lymphadenitis in the affected area. These are discussed on p. 185.

ACUTE SPECIFIC[1] INFECTIONS—TETANUS

About 200 to 300 cases of tetanus occur annually in the United Kingdom, with a mortality of about 40%. This number is unlikely to diminish, as the majority follow a trivial or forgotten abrasion. Eradication can only follow universal active immunisation. Tetanus is relatively more common in other parts of the world.

The causal organism, *Cl. tetani*, is a Gram positive rod which forms a terminal spore giving a drum-stick appearance. It is an anaerobe; the growth requirements depend on other organisms in the wound, and on foreign bodies or dead tissue; and when it grows it forms a very powerful toxin. Although the organism is common, especially in manure and soil, the complex conditions for growth and toxigenesis can only occasionally co-exist and thus the disease is uncommon, but seems to occur when least expected (J. Macrae).

Tetanus is a wound infection; any wound will serve, but deep, contused, soil-contaminated injuries infected with pyogenic organisms and containing foreign bodies are most likely sources of tetanus toxæmia. The disease is wholly produced by the exotoxin. The organism itself does not damage tissues. The toxin travels along nerves to the central nervous system where it has a dual action: (1) it interferes with the acetyl-choline/cholinesterase balance at

[1] A specific disease is one which is produced by a single type of micro-organism.

James Macrae, Contemporary. Physician, Ham Green Hospital, nr. Bristol, England.

the peripheral motor end plates so that a continuous excess of acetyl-choline remains, producing clinically a sustained state of tonic muscular spasm, and (2) it causes extreme hyper-excitability of motor-neurones in the anterior horn cells which can result in explosive and widespread reflex spasm of muscle in response to even minor sensory stimuli. The toxin producing such effects is fixed in the tissues and cannot then be neutralised by antitoxin. Only free toxin can be neutralised. There is also a concomitant sympathetic overactivity.

FIG. 24.—Risus sardonicus.

Clinical Features.—Generally a short incubation period implies a serious attack, but an incubation period measured from the time of wounding until the first symptom is often fallacious since we cannot know when the organism started to grow or to produce toxin. Indeed, it is known that wounds containing tetanus organisms have healed and been forgotten for years and then some local change produced the correct combination of factors allowing the organism to multiply and produce toxin (latent tetanus). This should be remembered when re-opening an old accident or war wound.

A better prognostic index is the ' period of onset '. This is the time between the first symptom and the first reflex spasm. If this period has been less than forty-eight hours, death is very likely. As the period of onset lengthens so does the prognosis improve.[1]

The first symptoms are difficulty in swallowing and stiffness of the jaw, succeeded by pain in the neck, back, and abdomen. Then tonic muscle spasm develops, often sparing the limbs relatively. The sardonic smile of tetanus is evidence of this stage of the disease (fig. 24), and there is considerable difficulty in swallowing and reduction in breathing capacity. Thereafter, reflex convulsions can occur affecting all the muscles, causing great pain, opisthotonus and sometimes muscle rupture (psoas, rectus abdominis, pectoral) (fig. 25). These spasms are spontaneous or may be induced by even trivial stimuli of noise, injury, or movement. If severe, they stop respiration and the patient becomes cyanosed. A cyanotic tetanic convulsion threatens life. Tonic muscular spasm remains between the reflex attacks, thus distinguishing tetanus from strychnine poisoning.

FIG. 25.—A torn rectus abdominis muscle from a case of tetanus, which followed a trivial wound of a toe. (*R.C.S. Museum.*)

[1] Hippocrates, *circa* 460–377 B.C., is believed to have been the first to recognise this fact.

The temperature is usually a little elevated and the pulse is rapid. Without appropriate treatment, death occurs in a few days, usually from pneumonia, or from respiratory failure during a cyanotic attack.

Patients older than forty-five years fare badly with tetanus. In the newborn the infection enters the raw stump of the umbilical cord (tetanus neonatorum) and the disease tends to be rapidly fatal. It is unfortunately still common in certain parts of the world.

At an early stage, tetanus might be mistaken for tonsillitis, back strain, an acute abdominal condition, or hysteria (the latter, especially if dysphagia is the presenting symptom).

Treatment.—Prophylactic.—*Active immunity* is conferred by injections of absorbed toxoid; it is 100% safe but it takes time to develop. One ml. is administered and repeated at intervals of six weeks and six months, and followed by a ' booster ' dose every five years. If such an active immunity is present, another dose of toxoid should be given at the time of injury.[1] Long acting depot penicillin (e.g. Penidural) should also be included in the prophylactic treatment.

Antitetanus Sera.—The inherent disadvantages of equine antiserum (A.T.S.) (see Serum reactions, p. 32), can be avoided if human antitetanus globulin (A.T.G.) is available. This is an homologous antitoxin and its protective value is one hundred times that of the heterologous equine type. A dose of 250 units of A.T.G. will give adequate cover during the period needed to establish active immunity.

It is essential that every patient who has had antitetanic serum should be immunised at once with toxoid.

Treatment of Established Case.—All cases should be isolated in quietness and comfort. It is not necessary to attempt excision of the wound. If pus is present it should be drained but, in general, ordinary wound toilet will suffice. 100,000 I.U. of antitetanic serum are given at once, half intravenously and half intramuscularly, to limit the further fixation of toxin. If available, human antitetanus globulin (A.T.G.) is preferable to A.T.S.—only one dose is ever needed. In treatment, as in prophylaxis, tetanus antibody material, whether heterologous or homologous, is the only substance capable of neutralising the toxin which causes this serious disease. Further treatment resolves itself into three stages, dependent on the development of the disease.

Stage 1.—*A mild case* is one where there is tonic rigidity but neither swallowing nor respiration is noticeably affected. Initial sedation and some relaxant effect is obtained by such drugs as promazine (Sparine) 100 to 200 mg i.m., amylobarbitone 200 mg i.m., and diazepam 5–30 mg i.v. These drugs will be needed six-hourly.

Stage 2.—*A seriously ill* case is one where there is swallowing difficulty. There may be some reflex spasm but no major cyanotic episodes. A naso-gastric tube is passed (Chap. 8) and sedation continued. A liquid balanced diet containing 2000 Calories (approx. 8400 kJ)[2] and 50 g of protein is given by the tube, unless the blood urea rises, when protein must be temporarily discontinued. If the patient has difficulty in breathing, he will require a tracheostomy in order to maintain a clear airway. The operation can often be done under local anæsthesia and a cuffed tracheal tube must be inserted. Nursing must now include meticulous care of tracheostomy tube —posture, suction, and humidification (Chap. 39).

Stage 3.—*Dangerously ill.*—Should the patient have a major cyanotic convulsion, tubocurarine chloride in doses of 20–40 mg is given intravenously initially and after-

[1] Tetanus toxoid practically eliminated tetanus during the 1939–1945 war.

[2] The International Union of Nutritional Sciences accepted in 1969 the recommendation that the medical or kilo calorie be replaced by the Joule in nutritional sciences. (1 medical Calorie = 4186·8J!)

wards intramuscularly to maintain complete relaxation. Intermittent positive pressure respiration must be provided until such time as no further muscle spasms occur (usually twelve to thirty days). Sedation as given in Stages I and II is stopped. The treatment makes such demands on medical and nursing services that it can only be used safely in a special, experienced respiratory unit. A case requires over 350 individual acts of nursing service each day, and a special nurse continually, while the monitoring requirements of artificial respiration need special equipment and skill.

The plight of the '*dangerously ill*' case is often worsened by the development of complications made possible by the unnatural preservation of life brought about by artificial respiration. Such patients may develop severe negative nitrogen balance, myocarditis, vascular failure associated with extremes of blood pressure and pulse-rates, and severe damage to motor neurones in the cord and brain leading to major paralysis. These are all due to toxin. Thus, even if treatment prevents death by spasm or pneumonia, patients may still die from a lethal dose of toxin.

Although the major therapeutic effort is exerted in the control of spasm and respiration, the general care of the patient must not be forgotten. Antibiotics are used judiciously to prevent infection. The eyes must be protected while curare is used, or conjunctivitis develops. The curarised patient is unresponsive, but is conscious and sensitive: this must never be forgotten.

The treatment described, best carried out in critical or intensive care units, can reduce the tetanus death rate to about 15%.

SERUM REACTIONS

Patients who have had previous serum injections or who are prone to asthma or allergic diseases may have a local or general reaction when serum or other antigen is injected. Anaphylaxis is the term given to describe this reaction.

Prevention.—Utmost care must be taken before any intravenous or subcutaneous injection is given. *When using serum, the best procedure is to dilute it ten times in normal saline and to inject it subcutaneously in small amounts.* If anaphylaxis develops it will be mild and respond easily to treatment. If trouble is anticipated, intravenous Piriton (chlorpheniramine) 10 mg should be given ten minutes beforehand. With antitetanic serum 35 per cent of patients get some sort of reaction. The usual routine is to give 0·2 ml of the serum, to wait half an hour, then give the remaining 0·8 ml and to wait for a further half-hour. Adrenaline (1:1 000) must be immediately available.

Acute Anaphylaxis.—The symptoms may appear within a few minutes. They are most alarming and may rapidly lead to death. In general the treatment is to give adrenalin (1:1 000) 1 ml subcutaneously or Piriton 10 mg intravenously, and to apply a tourniquet. There are four main types of acute reaction.

(1) *Respiratory distress* with urticaria, bronchospasm, and œdema of the glottis. Treatment is to introduce a large needle through the crico-thyroid membrane in order to give endotracheal oxygen. Calcium gluconate 20 ml of 10 per cent intravenously will help. If pulmonary œdema occurs, hydrocortisone succinate 100 mg intravenously must be given.

(2) *Generalised Convulsions.*—This requires 50 mg 2·5% thiopentone intravenously repeated in five minutes if necessary.

(3) *Peripheral Vascular Failure.*—This requires phenylephrine 1 mg. intravenously and intravenous hydrocortisone. If this fails, a noradrenaline drip should be started.

(4) *Sudden Cardiac Arrest.*—This requires immediate cardiac massage: the heart must be restarted within three minutes (Chap. 41).

Local anaphylaxis is occasionally seen if further injections are given at the same site after the interval of a week. An acute inflammatory reaction occurs, which in rare cases progresses to sloughing of tissues.

Serum sickness is the commonest serum reaction and may occur in any patient. Urticaria, œdema, pains or effusions in joints, and elevated temperature are usual features, which frequently occur seven to ten days after the injection. Cold applications applied locally, minimise the discomfort. Oral anti-histamines must be given until the condition responds.

INFECTIVE GANGRENE

Gas Gangrene

Gas gangrene (clostridial myositis), although uncommon, is a regular contributory cause of death in the case of accidents. It may complicate thigh amputations in patients with arterial disease.

Clostridia are introduced into the wound from clothing more commonly than by soil, hence wounds of the thighs and buttocks are especially liable to infection owing to fæcal contamination.

The causative organisms present in the normal intestine and in soil are *Clostridium welchii* (fig. 26) (*Cl. perfringens*) and, less common, *Cl. septicum* and *Cl. œdematiens*. They invade and multiply in damaged muscle, form gas bubbles and produce toxins.

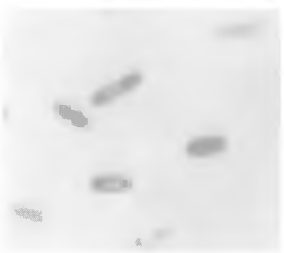


FIG. 26.—Smear of muscle from gas gangrene with *Clostridia* (some with spores).

Clostridia are often found in wounds without causing disease.

The diagnosis of true gas gangrene, i.e. gas in the tissues combined with gangrene, is essentially clinical. Pain, swelling, œdema, and toxæmia, usually develop within forty-eight hours. There is a rapid increase in the pulse-rate, a slight rise, or, in severe cases, a fall of temperature, and vomiting. The mental condition usually remains clear, and death occurs suddenly. (We have seen a patient die while engaged in completing a football forecast.) On examination of the wound, stitches are seen to be under tension, and, through the pouting edges, thin brownish fluid exudes, which possesses a sickly foul odour. The limb is tense and swollen, and palpation often reveals crepitus. An X-ray will demonstrate the presence of gas in intramuscular planes, and is very useful in the diagnosis of early cases. Gas and foul fluid form in the cellular spaces; contractability of the muscles is soon lost, and their colour changes successively from dull red to green and then black. If septicæmia occurs, gas is produced in many organs, notably the liver, which at necropsy drips with frothy blood, and is well named a ' foaming liver '.

Treatment.—Prevention.—The essential prophylactic measure is early and meticulous excision of dead and dying tissue. Partial suturing is sufficient, and secondary suture can be undertaken at a later date (p. 6). Prophylactic *penicillin* should be administered.

Treatment of Established Disease.—The patient's life depends upon:

(i) Preparation for immediate operation, with blood transfusion (or plasma if necessary).

(ii) Injection of 8 mega units of penicillin immediately, followed by at least 4 mega units four-hourly for 4–8 days. Streptomycin (1–2 g) daily may be used in addition to the large doses of penicillin.

(iii) Adequate excision of all affected groups of muscles, through *long* incisions in the limb (on both sides if necessary). If total clearance is not

William Henry Welch, 1850–1934. Professor of Pathology, Johns Hopkins University, Baltimore, Maryland, U.S.A. Discovered the causative organism of gas gangrene in 1892.

possible, amputation must be performed (e.g. disarticulation through the hip-joint for gas gangrene in an above-knee amputation stump).

(iv) Anti-gas gangrene serum (3 ampoules of polyvalent serum immediately, and repeated six-hourly i.v.).

(v) If available, oxygen drenching in a pressure chamber (hyperbaric oxygen, p. 85) reduces the amount of toxin produced by the organisms, and when the condition is under control, surgery is then confined to the excision of sloughs and skin grafting.

Gas Infections

In less severe forms of infection gas bubbles occasionally are formed:

(i) *Local Type.*—Invasion is sometimes limited to a single muscle, which becomes necrotic while adjacent muscles escape.

(ii) A *gas abscess* frequently occurs if a contaminated foreign body is present in a wound (fig. 27). It subsides when the abscess is adequately opened ('uncapped'), and the foreign body removed.

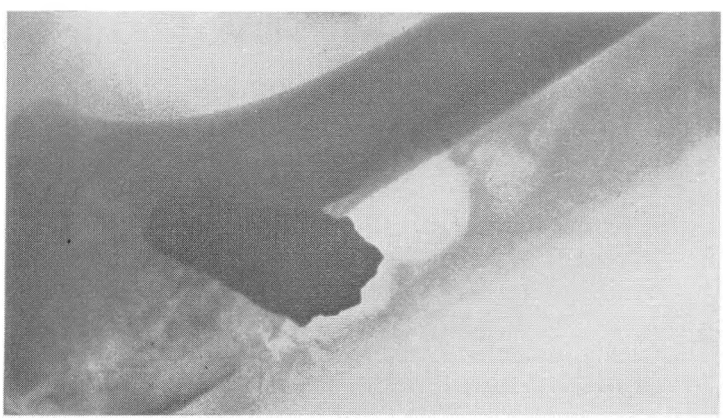


FIG. 27.—Comminuted fracture of humerus due to a fragment of a high-explosive missile; three days after injury at Dunkirk (1941). Bubbles of gas are present under the triceps muscle (shown here as rounded 'white' areas).

(iii) *Subcutaneous infection* is sometimes seen spreading for a considerable distance around a wound. Crepitus is easily palpable, and the skin becomes khaki-coloured as a result of hæmolysis. *Unless this condition is recognised a needless amputation may be performed.* The surgical treatment required is thorough drainage of the wound by multiple incisions into the affected subcutaneous tissues, down to, but not including, the deep fascia, otherwise infection may be carried into the underlying fascial planes or muscles. Removal of subcutaneous necrotic fat and connective tissue is carried out. Penicillin must be given and, if available, hyperbaric oxygen can be most helpful.

Cancrum oris et noma is discussed in Chap. 32.

Carbuncles and boils are considered on p. 113.

ERYSIPELAS

Erysipelas is a spreading inflammation of the skin and subcutaneous tissues due, to an infection by *Str. pyogenes*. The general health of the patient is usually below par, and debilitating conditions and the extremes of life are predisposing causes.

Symptoms.—The skin in the vicinity of a scratch or abrasion becomes irritable and feels stiff. After a few hours symptoms of toxæmia supervene, which rapidly increase in severity.

Signs.—If the infection commences in a wound, a rose-pink rash extends over the adjacent skin. The edge of the rash is raised, a feature which is often more easily appreciated with the finger than the eye. The colour of the rash and its obvious edge distinguish erysipelas from cellulitis. As the rash extends, vesicles appear, which burst and discharge serum. Considerable œdema occurs when lax tissues are involved, particularly the orbit and the scrotum. The rash gradually fades, and for some weeks a brown discoloration of the skin remains, due to pigment set free as a result of destruction of red corpuscles.

Complications.—*Gangrene.*—Sloughing of skin and subcutaneous tissues occasionally occurs, particularly of lax tissues in patients with poor resistance.

Lymphatic Obstruction.—A severe attack of erysipelas is sometimes followed by fibrosis of the lymphatic vessels and nodes, so that lymphatic drainage is impaired. The eyelids are not uncommonly affected, greatly to the detriment of the patient's appearance and comfort (fig. 28).

Treatment.—Fortunately it is a relatively uncommon disease and is completely controlled by penicillin.

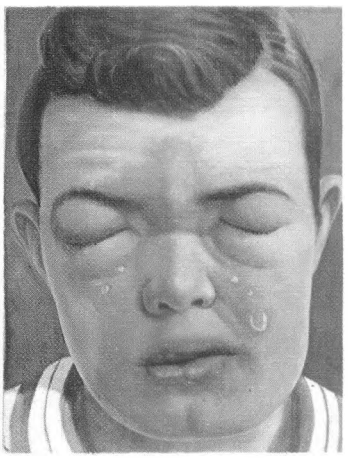


FIG. 28.—Erysipelas with lymphatic œdema of face and eyelids. The patient was unable to open his eyes.

ANTHRAX

B. anthracis is a large Gram-positive aerobic spore-forming rod. It is very resistant to heat and antiseptics. The disease occurs in domestic animals in some parts of the world and is likely to appear in men who handle imported carcases, wool, hides, hair, and bone-meal.

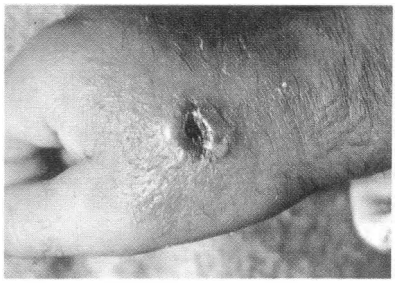


FIG. 29.—Anthrax pustule.
(Dr. W. D. Paterson, Carlisle.)

The **cutaneous type** is the commonest human variety; the incubation period is from three to four days. The lesion usually commences on an exposed portion of the body, such as the hands, forearms, or face. An itching papule occurs, around which a patch of induration soon becomes evident. The papule suppurates and is replaced by a black slough, and a ring of vesicles appears on the surrounding indurated area. This stage comprises the typical 'malignant pustule'. A brawny, congested area of induration develops around the site of infection. The regional lymph nodes are involved. Toxæmia is always in evidence. A smear of vesicle fluid is used to confirm the diagnosis by culture and animal inoculation.

Treatment.—Penicillin is the treatment of choice.

Prevention.—Must include precautions to sterilise potentially infected animal products and wool from countries where the disease is endemic.

Differential Diagnosis.—The condition easily mistaken for it is a severe furuncle (Chap. 30) and accidental vaccinia. (The recently vaccinated child with the vaccinia pustule on its arm in full activity, while being carried by the mother or nurse, places the vaccinated area against her cheek.)

Other forms of anthrax are rarely, if ever, now seen, e.g. **Woolsorter's Disease**, a pneumonia due to inhalation of spores and an **Alimentary Type,** following ingestion of spores.

CANDIDIASIS

Candida albicans (formerly called **monilia**) is a yeast, frequently present in small numbers in the healthy mouth and bowel. It may cause primary infection in the newborn, or superinfection when flora are disturbed by antibiotic treatment. In **Thrush** (candidal stomatitis) white patches are seen in the mouth; it may occur in infants, in post-operative patients, and with ill-fitting dental plates. **Vaginitis** is common in pregnancy and diabetes. Candida may infect moist skin under breasts, and the nail folds.

Administration of broad-spectrum antibiotics often results in proliferation of candida in the respiratory tract and bowel, and may be responsible for digestive upsets. Systemic candidiasis with invasion of lung and blood-stream is a complication of immuno-suppression in transplantation surgery and in the chemotherapy of malignant disease.

Candida infections are treated by the topical antibiotic nystatin or by gentian violet.

CHAPTER 5

CHRONIC SPECIFIC INFECTIONS[1]

(TUBERCULOSIS, ACTINOMYCOSIS, LEPROSY, SYPHILIS)

TUBERCULOSIS

THIS disease derives its name from the characteristic lesion called a tubercle (a collection of inflammatory cells surrounding the organism, and later progressing to caseation) which is the response of the body to invasion by tubercle bacilli (*mycobacterium tuberculosis*). When stained they are difficult to decolorise with acid (hence the term 'acid-fast bacilli'). *Myco. tuberculosis* (fig. 30) may be human or bovine in type and is spread by airborne infection by the human type from open tuberculous cases, and by milk from infected cows in the case of the bovine bacillus. The three main routes of primary infection are.—(1) By direct spread to the lungs where a primary focus occurs and thence by lymphatics to the hilar lymph nodes. There is nearly always some spread by the blood stream to other organs where the bacilli may lie dormant for many years. (2) Via the tonsils to the lymph nodes of the neck. (3) Via the lower ileum, probably in Peyer's patches, to the lymph nodes of the ileocæcal angle where they later show as calcified areas on X-ray.

Fig. 30.—*Mycobacterium tuberculosis* in human sputum.

Tuberculosis of the various organs is described in the appropriate chapters of this book.

Guidelines of Treatment.—The second half of this century has witnessed dramatic advances in the treatment of this notorious and age-old disease. Previously recovery depended upon waiting for the requisite level of immunity to be built up within the patient. This meant bed rest, prolonged for 3–4 years, and adequate nutrition in a decent environment free from overcrowding (*i.e.* in a sanatorium). Surgery, designed to aid rest and healing, was used only when the patient became well enough to withstand operative trauma, examples being extra-articular arthrodesis of joints using bone grafts, and extrapleural compression of the apices of the lungs by selective filleting of ribs (thoracoplasty). By 1950 recovery and supportive surgery was hastened by the introduction and use of streptomycin (1 g i.m. daily), para-amino-salicylic acid—PAS 1 (12–15 g daily), and iso-nicotinic acid—Isoniazid—(300 mg daily). These drugs are bacteriostatic, and the first and last bactericidal as well. Isoniazid has a small molecule, penetrates macrophages and destroys bacilli in the middle of a tubercle. It has

[1] A chronic specific disease can be recognised by clinical and pathological features specifically caused by a particular organism.

Robert Koch, 1843–1910. Professor of Hygiene and Bacteriology, Berlin (1885–1910). Discovered Mycobacterium Tuberculosis whilst working in the Imperial Health Office, Berlin in 1882.
Johann Conrad Peyer, 1653–1712. Professor of Logic, Rhetoric and Medicine, Schaffhausen, Switzerland. Described the Lymph Follicles in the Intestine in 1677.

been usual to give the 3 drugs together for the first 3 months of treatment to avoid development of drug resistance. By then the sensitivity of the organism has been determined, and then the streptomycin may be omitted. The whole course may last for 2 years.

Today, new drugs have been introduced and shorter courses (e.g. 9 months) are being advocated. The front-line drugs include rifampicin, which is given for 4–8 weeks, and ethambutol (dosages being related to body weight). These have to be given in conjunction with INAH. Streptomycin is kept as the heavy artillery. For an example see renal tuberculosis Chap. 56.

All these drugs must be given under careful supervision. Streptomycin has toxic effects on the vestibular branch of the auditory nerve, and on the kidneys. INAH causes peripheral neuritis, ethambutol visual impairment, and rifampicin is hepatotoxic.

It should be remembered that it is nigh impossible to eradicate every tubercle bacillus from the body. Lying dormant and enveloped in fibrous tissue any remaining bacilli are still able to cause a flare of the disease, particularly after trauma, after gastrointestinal operations resulting in nutritional deficiency, and in old age.

Amyloid Disease primary or secondary. The primary form is either idiopathic or a complication of multiple myelomatosis. The secondary form may be seen in cases of chronic osteomyelitis, bronchiectasis, tuberculosis, rheumatoid arthritis and lepromatous leprosy. Abnormal protein, related to immunoglobulins and lymphocytes, is deposited in the walls of the arterioles and later in the larger vessels. Microscopically, congo-red stains the infiltrated tissues. The kidneys, intestine, liver, and spleen are specially affected. Polyuria occurs due to infiltration of the glomeruli of the kidneys. Diarrhœa ensues owing to infiltration of the mucosal villi. The spleen and liver become enlarged and palpable. It is most important to bear in mind that this disease is a possibility in anyone who has prolonged suppuration from whatever cause. The diagnosis can be made on rectal biopsy—this method is simple, safe, and reliable. If the source of the infection can be eradicated, early amyloid disease will resolve.

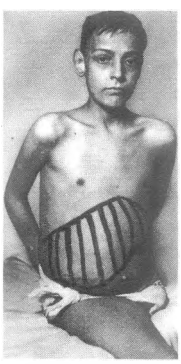


FIG. 31.—Enlargement of the liver and spleen due to amyloid disease. Amputation was performed through the left hip joint for osteomyelitis of the upper end of the femur, but sinuses persisted. He succumbed six months later.

ACTINOMYCOSIS

This disease is caused by *Actinomyces israelii*, an anaerobic Gram-positive branching filamentous organism (fig. 32) which sometimes lives as a harmless parasite in the tonsillar crypts and dental cavities of the otherwise normal mouth. It is popularly supposed that it occurs in corn and grasses, but the pathogenic organism does not do so. If the organism invades tissue, it causes a subacute pyogenic inflammation with considerable induration and sinus formation. Trauma and the presence of carious teeth are important predisposing factors in the development of lesions in the mouth.

James Israel, 1848–1926. Urologist, Germany. Discovered Actinomyces Bovis in man in 1878.

Diagnosis depends on finding the organism in pus or in tissue section. Pus should be collected in a sterile tube (a swab is usually insufficient) and inspected in a good light for the presence of pin-head sized 'sulphur granules'. On microscopy the granules are seen to consist of Grampositive mycelia. The peripheral filaments radiate[1] from the central part of the granule and may be surrounded by Gram-negative tissue clubs. The identity of the organisms may be confirmed by culture but the presence of secondary organisms often makes this difficult.

FIG. 32.—*Actinomyces israelii* in tissue section.

Actinomycotic lesions are characterised by the formation of a firm, indurated mass, the edges of which are indefinite, and infection spreads by direct invasion of adjacent tissues. Lymph nodes are not affected, but if a vein is invaded, pyæmic infection is likely to follow.

There are four main clinical forms of actinomycosis:

(i) *Facio-cervical* is the commonest form. The lower jaw is more frequently affected, often adjacent to a carious tooth. The gum becomes so indurated that it simulates a bony swelling. As extension occurs, nodules appear, which soften and burst. The overlying skin of the face and neck is indurated and bluish in colour, softening occurs in patches, and eventually abscesses burst through the skin (fig. 33). The characteristic features of the condition are chronicity, dense induration and sinuses surrounded by bluish skin.

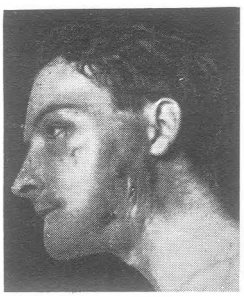


FIG. 33.—Actinomycosis. Indurated sinuses extend from the neck to the orbit.

(ii) *Thorax.*—The lungs and pleura are infected either by aspiration of the fungus or, occasionally, by direct spread downwards from the pharynx or neck, or upwards through the diaphragm.

The disease extends through the lungs to the pleura and chest wall, which, in the late stages, is riddled with sinuses (fig. 34). An empyema is not uncommon, and the infection sometimes spreads through the diaphragm to the liver or subphrenic spaces. Clinically, the condition resembles tuberculosis, and in the early stages is only distinguished by the discovery of the organism in the sputum and confirmation by culture.

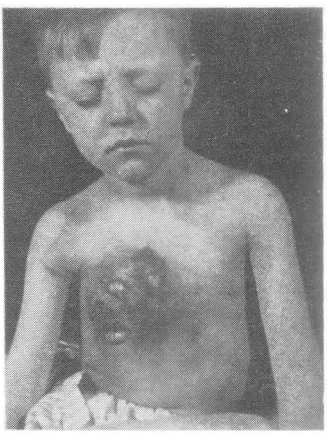


FIG. 34.—Actinomycosis of the lung involving the chest wall.

(iii) *Right iliac fossa* (Chap. 49).

(iv) *Liver* (Chap. 45).

Treatment.—Actinomyces is usually sensi-

[1] Actinomyces = Ray fungus.

tive to penicillin, tetracycline and some other antibiotics (e.g. Lincomycin), but the sensitivity should be checked in the laboratory. A prolonged intensive course of penicillin (10 mega units reducing to 4 mega units daily) is usually the best treatment until all signs of the disease have disappeared.

LEPROSY (HANSEN'S DISEASE[1])

There are probably from ten to fifteen million leprosy sufferers in the world today.

Leprosy is an infectious disease widely spread throughout the tropical and subtropical areas of the world. It is caused by *Mycobacterium lepræ*, an acid-fast bacillus morphologically like the tubercle bacillus. It is mainly, but not entirely, contracted in childhood and late adolescence. While the mode of transmission regarding the portal of entry of *M. lepræ* is not known, recent studies have established that the source of infection is mainly from nasal secretions of leprosy patients and not from their skin. Leprosy is no longer endemic in Northern Europe, as it was in the middle ages and in Norway until the late 19th century; neither is it now spread by immigrants in Europe. These facts suggest that leprosy requires for its transmission some factors associated with poverty or lack of hygiene that is common in the areas where it is still endemic. A vast change in the outlook for this disease has occurred in the last 30 years. The condition was formerly regarded as hopeless, but in spite of the fact that it is now curable only 25% of the cases of this widely spread disease are under treatment. It is probably true to say that leprosy causes more paralysis, deformity, and misery than any other disease, but that in many cases these could now be prevented by modern therapy, given an adequate medical service for early diagnosis.

Although leprosy is a systemic infection, it presents predominantly as an infection of the skin, upper respiratory tract and dermal and peripheral nerves. Leprosy must always be considered in a patient presenting with a combination of skin *and* neural disorder, particularly because of the variation in the preponderance of these two manifestations and the tremendous variation in the appearance and histopathology of the dermal lesions, among individual patients with leprosy. These diverse manifestations led to a plethora of classification systems until Ridley and Jopling found that

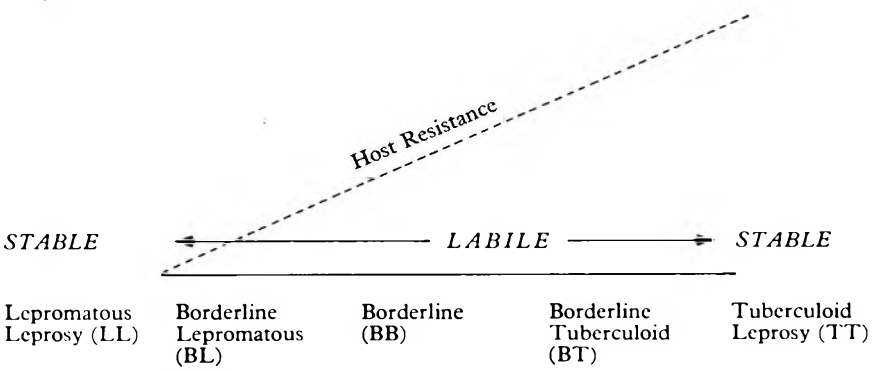


FIG. 35.—Ridley-Jopling classification of leprosy based on immunity of host.

there was a spectrum of disease in leprosy determined by the resistance of the host (fig. 35). The spectrum ranges from polar lepromatous (LL) to polar tuberculoid (TT) leprosy, denoting patients with minimal and maximal irreversible capacities to mount an immunological response against *M. lepræ*. The majority of patients are more labile in having some residual immunological capacity against *M. lepræ*, and thus are represented as borderline (BB), or borderline-lepromatous (BL) or borderline-tuberculoid (BT), if they veer more towards lepromatous or tuberculoid leprosy.

[1] After Hansen who first showed leprosy was a bacterial infection caused by *M. lepræ*. Because of the stigma attached to leprosy, Dr. R. G. Cochrane and others recommended that it should be referred to as Hansen's disease.

Gerhard Henrik Armauer Hansen, 1841–1912. Physician in Charge of a Leper Hospital near Bergen, Norway.
Robert Greenhill Cochrane, Contemporary. Formerly Medical Superintendent, Kola Ndoto Leprosarium, Shinyange, Tanzania.
William Henry Jopling, Contemporary. Formerly Consultant Leprologist, Hospital for Tropical Diseases, London.
Dennis Snow Ridley, Contemporary. Consultant Pathologist, Hospital for Tropical Diseases, London.

If left untreated, patients anywhere within the borderline spectrum can deteriorate towards lepromatous leprosy, whereas under treatment they will shift towards polar tuberculoid leprosy.

Lepromatous Leprosy

There is little or no resistance, the bacilli multiply with little cellular response, until the subcutaneous tissues may be loaded with masses of bacilli, many of them distending macrophages as large ' globi '. The cellular infiltrate is mainly of macrophages with a few lymphocytes.

Tuberculoid Leprosy

There is a strong tissue response, the bacilli are not numerous and are seldom seen except by special concentration methods. The histology consists of an epitheloid granuloma, many lymphocytes and a few giant cells.

Characteristically *tuberculoid* leprosy causes sharply localised lesions often affecting only one part of the body, while *lepromatous* leprosy is symmetrical and extensive. Since the damage in leprosy is mainly due to the response of the host cells, *tuberculoid* leprosy causes early, severe but localised deformity, while *lepromatous* leprosy causes deformity late, and more mildly and widely spread. The most severely deformed patients are those affected by some of the border-line forms where the disease may be both widespread through the body and also rather violent in its reactions.

A unique feature of the disease is not only its predilection for the surface of the body, but also for the cool parts of the surface. Warm areas like the axilla and gluteal cleft are spared, while the parts of the upper respiratory tract, like the lining of the nose, are severely involved. The testis is affected, while the ovary, and other deeply placed glands and organs, are unaffected. Since leprosy does not affect the vital organs of the body, it rarely causes death, and patients do not even feel ill for most of the time they have the disease.

During treatment, many patients manifest acute episodes which are referred to as ' reactions ' of which there are two distinct types:

The ' lepra or Type 1 reaction ' occurs in the borderline form of leprosy (BT, BB and BL), in which the skin lesions become erythematous, warm to touch and may break down, and the nerves swell and become painful and tender to touch. It is usually caused by a rapid increase in cell-mediated immunity by the host with an outpouring of lymphocytes into the lesions giving rise to acute inflammation with associated œdema. For this reason, the lesions have been referred to as ' reversal reactions ', advantageous by resulting in destruction of *M. lepræ*, but causing irreversible destruction of axons when the inflammation and œdema occur in nerves.

The other type of reaction is referred to as erythema nodosum leprosum (ENL or Type 2 reaction), usually occurring during treatment but confined to patients with lepromatous type leprosy (BL, LL). Here there are no changes in the established leprosy lesions, but new crops of very small erythematous lesions in the skin appear associated with systemic symptoms such as malaise, fever and nerve and joint pain. Occasionally, rhinitis, acute iridocyclitis, swollen and tender lymph glands, acute epididymo-orchitis and proteinuria occur. This is an Arthus type reaction, due to the deposition of immune complexes in and around blood vessels, locally or generalised throughout the body, and more akin to chronic serum sickness. This reaction may come and go or be persistent, and can in its severest form be fatal.

One of the most characteristic features of leprosy is its effect on nerves (fig. 35). Histologically, the cellular infiltrate may be seen localised around nerve fibres in and under the skin and, on clinical examination, superficial nerves such as the ulnar and posterior auricular may be observed to be swollen and tender. The anæsthesia that results from nerve involvement is an important point in diagnosis, and is also a cause of secondary damage and deformity. Much of the loss and disfigurement of hands and feet which has always been associated with leprosy is now known to be due not to leprosy itself, but to the damage and misuse which follows loss of pain sensation.

Medical Treatment.—Diaminodiphenylsulphone (Dapsone, DDS) (50–100 mg/daily) is now the standard treatment for all forms of leprosy. It is a bacteriostatic drug and experience has shown that in leprosy it has to be given for a long time—at least 3 years for all forms of tuberculoid leprosy and probably for life for all forms of lepromatous leprosy—for even when it is given to lepromatous patients until they

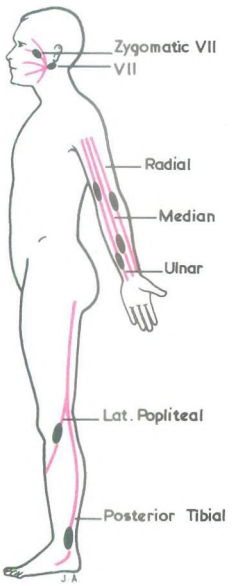


FIG. 36.—Sites of motor paralysis in leprosy.
(1) Ulnar nerve just above elbow joint or wrist joint.
(2) Median nerve just above wrist joint.
(3) Lateral popliteal at knee joint.
(4) Posterior tibial above ankle joint.
(5) Facial nerve in the bony canal or the zygomatic branch.
(6) Radial nerve at the elbow (rarely).
(After Paul Brand, F.R.C.S.)

become bacteriologically negative (5–10 years), they can still relapse when treatment is stopped. For many years it was considered that reactional episodes occurred more commonly when the full dose of DDS was given from the beginning of treatment and that reactions, once developed, persisted if dapsone treatment was continued. Both these ideas have now been proved to be fallacious. Since 1964 DDS resistance has been recognised, in ever increasing frequency throughout the world, among patients with lepromatous leprosy, and that it occurs much more commonly in those patients who have received smaller doses of DDS in the past or have had their treatment interrupted. Since DDS resistance occurs only among patients with lepromatous type leprosy, it is now recommended that new patients with lepromatous leprosy must be initiated with DDS plus another drug (in line with the successful use of combined therapy for the prevention of drug resistance in tuberculosis). The other antileprosy drugs of proven value are rifampicin, thiacetazone and clofazimine. Rifampicin, the first bactericidal drug against *M. lepræ*, has been shown to act more rapidly than any of the other antileprosy drugs. Unfortunately, the very high cost of rifampicin excludes its routine use by many leprosy control services, but it should be the drug of choice for combining with DDS in the initial treatment of all new lepromatous patients. Therefore rifampicin, in combination with DDS, should be given as a single initial dose of 1,200 mg, or as a daily dose of 600 mg fo rone week to three months, depending on funds available.

For reactions, symptomatic treatment should be given for the milder cases, including analgesics and tranquillisers. For the more severe reactions various proprietary antimonial drugs, such as Stibophen and Fouadin, etc., have proved beneficial as has Chloroquine. For a still more persistent and severe lepra and ENL reactions it may be absolutely essential to give steroids to tide the patients over and particularly to protect the nerves. For patients with the most severe ENL reactions, but not lepra reactions, thalidomide has proved to be as beneficial as steroids. If thalidomide is available it must be confined to male patients or females outside the child-bearing age. Clofazimine in high doses can suppress all but the most severe ENL reactions.

Surgery.—**The Deformities of Leprosy** are divided into *primary*—those which are caused directly by leprosy and its reactions, and *secondary*—those which result from anæsthesia and consequent misuse. The stigma of leprosy is the stigma of deformity, and a wide-open field awaits the plastic surgeon in this disease.

The Face.—*Primary deformity:* the skin of the face becomes thickened and sometimes nodular in lepromatous leprosy (fig. 37); the forehead, cheeks, nose, and ears are especially affected. The result in the acute phase is referred to as ' leonine facies '.

This infiltration subsides under medical treatment, but may leave the skin wrinkled and without its normal support, producing, in a young person, a caricature of old age. The hair of the eyebrows falls out and the lateral cartilages and septum of the nose may be destroyed leaving collapse of the centre of the nose and lifting of the tip towards the bridge (fig. 38). The upper branches of the facial nerve may be paralysed, giving rise to lagophthalmos; the lower branches are sometimes partially paralysed.

The patient with the above deformities usually finds it quite impossible to return to normal social relationships even though his leprosy may be cured. Plastic surgery can completely transform such a face using a post-nasal inlay to the nose (fig. 38),

FIG. 37.—Nodular lepromatous leprosy.

FIG. 38.—(*Left*) Extreme case of nasal collapse. (*Right*) After post-nasal epithelial inlay. (*N. H. Antia, F.R.C.S.*) (*By permission from the Annals of the Royal College of Surgeons of England.*)

and an ' island flap ' for the eyebrows. A temporalis muscle segment reactivates the eyelids and a face-lift may restore more normal contours to the skin.

Eyes.—Some of the blindness of leprosy is simply due to exposure following paralysis of the eyelids. This is correctable by plastic surgery. Other causes of loss of vision are lepromatous infiltration of the anterior segment of the eye and acute allergic changes of the tissues associated with reaction. Acute plastic irido-cyclitis is one of the commonest manifestations of this allergic reaction. Any redness of the eye or loss of visual acuity in leprosy demands full examination and prompt treatment if the sight is to be saved.

Hands[1].—*Primary Deformity.*—In the upper limbs, leprosy causes paralysis frequently in the ulnar nerve at the elbow and in the median nerve at the wrist (fig. 36) but rarely in the motor part of the radial nerve (1%). Thus the patient commonly loses the use of all the small muscles of the hand but very few forearm muscles (fig. 39). The fingers are clawed and the thumb lies beside the palm, unable to oppose the fingers.

Treatment. — The extensor carpi radialis brevis muscle is extended into the hand with free grafts which run along

FIG. 39.—Clawed left hand in leprosy. Note that the thumb lies completely unopposed. (*Dr. R. G. Cochrane, London.*)

[1] The work on reconstruction of the hand in Hansen's disease was started by Professor Paul Brand at the instigation of Dr. R. G. Cochrane (p. 40).

Paul Wilson Brand, Contemporary. Chief of the Rehabilitation Branch, U.S. Public Health Service Hospital, Carville, Louisiana, U.S.A.

the lines of the lumbrical tendons to correct the clawing of the fingers. The flexor sublimis tendon to the ring finger is withdrawn in the forearm and rerouted to oppose the thumb along the line of the abductor brevis. In this way the fingers and thumb may be balanced and function almost normally. Before attempting operation it is important to make sure that the fingers are made mobile by massage and exercise.

Secondary Deformity.—Since the hand is often totally anæsthetic the patient frequently burns himself, or damages himself by the uninhibited strength which he uses through his fingertips. His hands become scarred and progressively absorbed until only stumps remain. It takes patience and perseverance to teach every patient that his hands can be preserved only by constant alertness to foresee possible dangers, and constant gentleness to his own tissues which are not protected by pain. Once he is convinced that it is not leprosy that is destroying his fingers he may be willing to accept the discipline of caring for himself.

Feet.—*Primary Deformity.*—In the lower limbs the posterior tibial nerve is often involved at the ankle, giving rise to ' clawing ' of the toes and anæsthesia of the sole of the foot. The lateral popliteal nerve may also be destroyed, giving rise to foot-drop. The medial popliteal nerve is never involved, so the tibialis posterior muscle can be safely used to correct foot-drop.

Secondary Deformity.—The anæsthesia of the sole of the foot is very serious because almost every patient with insensitive feet sooner or later develops trophic ulceration. If he then continues to walk on his ulcers, the condition progresses and the infection spreads until, after a few years, the foot is contracted and distorted, and destroyed to the point where amputation must be advised.

It is important for the patient to understand the pathology of his ulcers and to realise that they are not due directly to leprosy. These ulcers heal readily with rest in a plaster cast and their recurrence can be prevented by the regular use of special footwear designed to spread the weight evenly over the whole foot.

SYPHILIS[1]

For a detailed description of venereal syphilis reference should be made to a text book on venereal disease.

Acquired syphilis is a venereal infection caused by *Treponema pallidum* (fig. 40), a delicate spiral organism (spirochæte), 6–15 μm in length. A dramatic decline in incidence after the introduction of penicillin has been followed by a gradual but significant increase throughout the world.

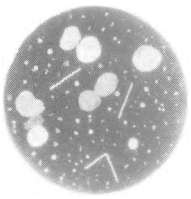


FIG. 40.—*Treponema pallidum* in the exudate from a chancre.

Transmission is by direct contact with a surface lesion containing treponemes which penetrate the skin or mucosa at the point of contact. Since treponemes are present only in the surface lesions of EARLY Syphilis, *i.e.* primary, secondary and occasionally the first year or two of latency, syphilis is only infective in its early stages. After 2–5 years acquired syphilis is rarely communicable and the ulcerative cutaneous lesions of tertiary syphilis are not regarded as infective as they contain few, if any, treponemes. The organism dies rapidly on drying, hence infective early lesions are predominantly sited in moist areas, *e.g.* genitals and mouth, and infection almost always takes place by sexual contact.

[1] Syphilis derives its name from a poem by a Physician, Girolamo Fracastoro (1478–1553), published in Venice in 1530. The poem tells of the shepherd, Syphilus, who was struck down by the disease as a punishment for insulting Apollo.

On his return from Haiti in 1493, Christopher Columbus (1451–1506) brought back syphilis, parrots and rare plants. The King and Queen of Spain received him with the highest honours.

Clinical Features.—The disease is divided into 4 stages.

Primary Syphilis.—A primary sore or chancre (figs. 41 and 42) develops at the site of entry of the treponemes in about 3–4 weeks. It may resolve at any stage of its development and thus be quite atypical. Starting as an indurated papule, it becomes eroded and when fully developed will present the following signs of a classical Hunterian chancre:—a shallow, indurated,

FIG. 41.—Penile primary chancres.

FIG. 42.—Vulval chancre in a female.

painless, non-bleeding ulcer, usually single, oval or round, with a raised hyperæmic margin. A painless enlargement of the associated lymph nodes occurs. There are no constitutional symptoms and a female patient will be unaware of the presence of an intravaginal chancre. Extragenital chancres of the lip,[1] tongue, nipple, *etc.* are now rare, but rectal and perianal primaries occur in homosexuals and are usually atypical.

Diagnosis is by finding *Treponema pallidum* in the clear exudate from the lesion by dark-field microscopy. The serum tests do not become positive for a week or two after the appearance of the chancre, hence initial negative serology must never be interpreted as excluding primary syphilis. The tests should be repeated for up to three months where doubt persists. Chancres must be distinguished from traumatic ulcers, chancroid (p. 49), herpes, granuloma inguinale (Chap. 59), early carcinoma or any genital lesion following risk.

Secondary Syphilis

Signs usually appear in 6–12 weeks as a reaction to spirochætæmia. The commonest sign is a dull red or coppery rash, generalised, symmetrical, indolent and non-irritant. Often inconspicuous, the rash is characteristically pleomorphic, being roseolar or macular at first, with papular, papulo-squamous, or other elements appearing later. Papules on contiguous moist sites, e.g. vulva, perineum may enlarge to form condylomata lata, fleshy,

[1] Primary sores on the lips usually result from kissing, and a case is recorded in which a gentleman with secondary ulceration of the mouth infected five young ladies at a dance, each of whom developed a chancre on the lip.

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London. To further his knowledge of venereal disease he is said to have inoculated himself with syphilis in 1767.

wart-like growths teeming with treponemes. Small, round, superficial erosions may occur in the mouth where they may coalesce to form the so-called snail-track ulcers. A generalised, painless lymphadenopathy often occurs and less common signs include sore throat, hoarseness, ' moth-eaten ' alopecia, iritis and bone and joint pains. Constitutional symptoms, malaise, headache, pyrexia, if present, are usually mild.

Latent Syphilis

Follows the untreated secondary stage and lasts from 2 years to a life-time. There are no signs but the serum tests are positive.

Late Syphilis (*Syn.* Tertiary Syphilis).—Syphilis in all its stages is essentially a vascular disease. Following spirochætæmia, treponemes cause inflammatory reactions in the perivascular lymphatics of terminal vessels with subsequent obliterative endarteritis, tissue necrosis and fibrosis. Since almost any structure may be involved, the signs can be extraordinarily variable and will be referrable to the site or system involved. Only about 40% of untreated syphilitics will develop signs of tertiary syphilis about 5–15 years after infection, 8–10% will develop *neurosyphilis*, 10–12% *cardiovascular syphilis* and 15–20% *late benign syphilis* involving less vital structures. The three types are not mutually exclusive. The typical lesion of late benign syphilis is the localised *gumma* or diffuse gummatous infiltration. The gumma is a syphilitic hypersensitivity reaction consisting of granulation tissue with central necrosis. Sloughing of a subcutaneous gumma may produce the typical, painless, punched-out gummatous ulcer with ' wash-leather ' base (fig. 43). On healing it leaves a silvery, ' tissue-paper ' scar. Alternatively the gummatous process may be nodular or infiltrative without ulceration and slow peripheral spread occurs with central healing. The individual lesions are round and indurated and grouped lesions are circinate in outline with sharply defined, hyperpigmented margins.

FIG. 43.—A painless, punched-out gumma of the shoulder.

Serological Tests for Treponemal Diseases are of two types :

1. *Non-specific* (*syn. Reagin, Non-treponemal, Lipoidal antigen tests.*)

Examples are Cardiolipin Wassermann (C/WR), Kahn, Ven. Dis. Ref. Lab. slide test (V.D.R.L.). These tests detect the presence of an antibody *reagin* in the serum of patients with treponemal infections, but *false positives* may occur since reagin may be present temporarily in some non-treponemal conditions; e.g. malaria, vaccinia, glandular fever, typhus, Weil's Disease. Stronger and more persistent false positives may occur in leprosy and collagen diseases. A diagnosis of syphilis is never justified on serological grounds alone without confirmation from one or more of the specific tests—

2. *Specific Tests.*—(*syn. Treponemal antigen tests*)

Examples are Reiter's Protein Complement Fixation Test (R.P.C.F.T.)
Treponemal Hæmagglutination Assay (T.P.H.A.)

Absorbed Fluorescent Antibody Test (F.T.A.Abs.)
Treponema Pallidum Immobilisation Test (T.P.I.)

After curative treatment for early syphilis the reagin tests should become negative within a year but the specific tests may remain weakly positive for life, giving rise to doubts as to the continued activity of the disease. This problem can be resolved by performing the tests on the IgM fraction of the patient's serum since antibodies associated with the IgM fraction cease to be produced within a few months of curative treatment whereas the production of IgG class antibodies may continue for life. Thus, negative tests on the IgM fraction indicates arrest or cure.

Treatment.—No local treatment should be applied to a suspected chancre until dark-field examinations have proved negative on 3 successive days and no treponemicidal antibiotics should be prescribed until syphilis is confirmed or excluded. Penicillin has supplanted all other forms of treatment. A high cure rate is achieved in early syphilis with intramuscular Procaine Penicillin G, one mega unit daily for 10–14 days. This dosage is prolonged to 14–21 days for latent and late syphilis. Clinical and serological observation should be continued for two years after treatment. For penicillin sensitive patients tetracycline, erythromycin, cephaloridine and chloramphenicol are all treponemicidal but experience with them is limited.

Herxheimer Reaction.—About 6 hours after the first injection 40% of early syphilitics will develop pyrexia, malaise and possibly rigors lasting a few hours only. The reaction is infrequent in late syphilis, but may be more serious.

Prognosis is excellent after standard treatment for early syphilis. In late syphilis, particularly of the cardiovascular system, cure of the underlying disease may not significantly improve the condition of the patient.

Congenital Syphilis

Transmission.—Infection occurs when treponemes from an infected expectant mother cross the placental barrier to the fœtal circulation. The more recent the mother's infection, the more likely is this to occur and the more serious the effects on the child. The results of fœtal infection vary from death in late fœtal life or early infancy to the birth and normal development of an apparently healthy child whose only abnormality is positive serology.

Early Congenital Syphilis

Signs in the newborn, which may be delayed for a few weeks, include a generalised rash and mucous erosions as in secondary syphilis. The ' snuffles '—a syphilitic rhinitis with nasal discharge which interferes with suckling causing loss of weight; epiphysitis, periostitis, osteochondritis, hepatosplenomegaly and basal meningitis. Signs may be so slight as to escape notice or so severe as to cause death in early infancy.

Late Congenital Syphilis

The extraordinary variety of clinical manifestations which can occur in acquired tertiary syphilis can also occur in childhood or puberty in late congenital syphilis; *e.g.* congenital neurosyphilis, cutaneous, visceral or skeletal gummata, but congenital cardiovascular syphilis is very rare. In addition some manifestations, the *Stigmata*, occur in congenital, but never in acquired syphilis.

The *stigmata* of late congenital syphilis—Hutchinson's classical triad consists of:

1. *Interstitial Keratitis.*—The most frequent of the stigmata, is a syphilitic hypersensitivity reaction with onset between 5–15 years. The cornea becomes inflamed causing pain, lacrimation and photophobia. It tends to be bilateral and recurrent. Prolonged, severe recurrent attacks result in a hazy ' ground-glass ' appearance of the cornea, with yellowish-red corneal patches in severe cases (salmon patches).[1] It is uninfluenced by anti-syphilitic treatment but can be controlled by local cortisone treatment.

2. *8th Nerve Deafness.* A progressive, bilateral, perceptive deafness, onset about puberty, uninfluenced by treatment.

[1] Not to be confused with the salmon-patch birthmark.

Karl Herxheimer, 1861–1944. Dermatologist, Frankfurt, Germany.

3. *Hutchinson's Teeth.* A peg-shaped deformity of the upper central incisors, second dentition. *Moon's Molars* (mulberry molars), the 6-year molars erupt with dwarfed cusps. Other classical signs include saddle nose (fig. 44), perforation of the palate (fig. 45), sabre tibia and Clutton's joints (painless effusions, commonly in the knee joint).

Prevention and Treatment.—One mega unit of Procaine Penicillin G given to the mother for 14–21 days as early as possible in pregnancy will not only protect her from the ravages of late syphilis but will prevent infection of the fœtus, or even cure

FIG. 44.—Saddle nose of congenital syphilis.

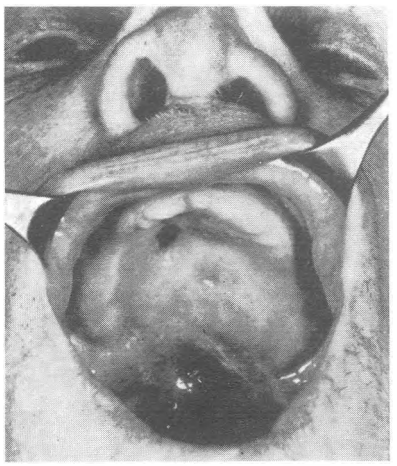


FIG. 45.—Perforation of palate.

it *in utero*, if already infected. An infected neonate whose mother received no treatment during pregnancy should be treated as for late acquired syphilis with the dosage adjusted to weight.

Neonatal Serology.—An expectant mother who has received treatment during pregnancy, or who is an untreated latent syphilitic may produce a child free from syphilis but seropositive due to passive transfer of maternal IgG antibodies. This serological problem can be solved by performing tests on the IgG and IgM fractions of the infant's serum. In the case of passive transfer the IgG tests will be positive and the IgM tests negative. Positive IgM tests indicate active disease in the child since the IgM fraction of the maternal serum proteins does not cross the barrier of the normal placenta. In all cases careful clinical and serological follow-up of mother and child is essential.

YAWS[1] (FRAMBŒSIA)

An endemic disease of rural areas in tropical countries of high humidity. The causative organisms, *Treponema pertenue*, appears indistinguishable from *Tr. pallidum* and produces identical serological reactions. Direct contact with an early lesion is the usual mode of transmission, but a vector may sometimes be responsible. It is not sexually transmitted. The primary lesion is most frequently seen on the legs of children. About one month after infection a papule appears at the site of entry of the treponemes, this ulcerates giving the lesion a pink, raised, raspberry like (frambœsia[2]) appearance. Secondary lesions, usually papillomatous, appear some weeks later. After 5 or more years of latency a minority of patients develop late gummatous-like lesions of soft tissue or bone similar to those of tertiary syphilis. The cardiovascular and nervous systems are not involved and congenital yaws does not occur. Treatment as for syphilis.

[1] The possible answer to ' What's Yaws? ' is ' Syphilis '. [2] Framboise (Fr.) = Raspberry.

Sir Jonathan Hutchinson, 1828–1913. Surgeon, The London Hospital (1859–1883). Described Hutchinson's Teeth and the triad in 1858.

Henry Moon, 1845–1892. Dental Surgeon, Guy's Hospital, London (1870–1887). Described Moon's Molars in 1876.

Henry Hugh Clutton, 1850–1909. Surgeon, St. Thomas's Hospital, London, described these joints in 1886.

CHANCROID (SOFT SORE)

This venereal disease is rare in western countries.

It is caused by the strepto-bacillus Ducrey (*Hæmophillus ducreyi*). Two to five days after infection, vesicles, often multiple, appear on the genitals. They become pustular and ulcerate, forming rounded, painful, soft, readily bleeding ulcers with undermined edges. Inguinal adenitis follows, the swollen nodes being hard and tender causing a feeling of stiffness in the groin. Resolution may occur at this stage, but suppuration may follow, the nodes becoming matted together forming a fluctuant unilocular abscess (bubo) with red overlying skin, in one or both groins. The bubo should never be incised since healing is very slow. Evacuation of the abscess by repeated aspirations is preferable. Phagedæna[1] sometimes occurs.

Treatment.—Any antibiotic which may prevent the identification of *T. pallidum* in a case of concomitant syphilitic infection, or when the ætiology of the lesion is in doubt, is contraindicated. Sulphonamides 4 g daily for 7–10 days is the treatment of choice.

Gonorrhœa.—This venereal disease is an acute specific disease and is discussed in relation to the Genitourinary system, Chapters 59 and 60.

Lympho-granuloma Inguinale is described in Chapter 59.

Granuloma Inguinale is described in Chapter 59.

PARASITIC DISEASES

Filariasis is a disease widely spread through tropical and sub-tropical countries. It is due to a nematode worm, *Filaria sanguinis hominis* which is transmitted by a mosquito (*Culex fatigans*). Once in the human body, the female worm finds its way to the lymphatics and lymph nodes (especially the inguinal group). It attains sexual maturity in six to eighteen months and produces vast numbers of microfilariæ, so that there may be 50 millions in the blood at one time. Obstruction of the lymph vessels ensues and this is manifested in (1) varicosities of the lymphatic vessels producing chylous ascites and hydroceles and, sometimes, chyluria, and (2) solid œdema (elephantiasis) often affecting the legs, scrotum, and arms, though it may occur anywhere. The best method of treatment known at present is Hetrazan (diethylcarbamazine-citrate), which appears to sterilise or kill the adult female worm. Prevention by anti-mosquito measures is, of course, vitally important. Elephantiasis may require surgery (p. 186).

Hydatid disease is discussed in Chapters 40 and 45.
Bilharziasis is discussed in Chapter 57.
Amœbiasis is discussed in Chapter 45.
Dracunculus mediensis (Guinea worm) fig. 70.

[1] Phagedæna = A rapidly spreading destructive ulceration.

Augusto Ducrey, 1860–1940. Professor of Dermatology, Pisa, Italy. Described the causative organism of chancroid in 1889.

CHAPTER 6

TUMOURS. CYSTS. ULCERS. SINUSES

THIS chapter concerns the general surgical pathology of tumours. Tumours arising in the various organs are considered in their appropriate chapters.

A tumour is a new formation of cells of independent growth usually arranged atypically, which fulfils no useful function and has no typical termination. The term ' tumour ' should be reserved for new-growths; its loose application to inflammatory swellings, such as Pott's puffy ' tumour ' (Chap. 26), or to enlargement of an organ due to hypertrophy, should be abandoned.

Causation

Over 300 years ago it was stated that ' any kind of external irritation, whether from motion, heat, or acrimony, may cause cancer '. Natives of Kashmir are prone to cancer of the skin of the thighs and lower abdomen (McNeill Love). This is due to their habit of keeping warm by squatting and hugging earthenware pots containing glowing charcoal (the pot being termed a *kangri*), with the result that the adjacent skin is irritated by heat and fumes. It is common knowledge that women can swallow in comfort fluids at a considerably higher temperature than men can tolerate, which fact may explain the greater incidence of post-cricoid carcinoma in females. ' Chimney-sweeps' ' cancer (Chap. 60), and ' countryman's lip ' (Chap. 30), and tar workers cancer (fig. 46) are other examples of carcinoma due to chronic irritation.

FIG. 46.—Squamous-celled carcinoma on the back of the hand of a tar worker.

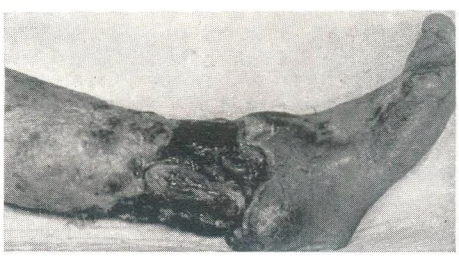


FIG. 47. — Squamous-celled carcinoma in a varicose ulcer (note everted edge).

Regeneration of tissue, with possibly the awakening of erstwhile dormant embryonic cells, may encourage malignant changes. Primary carcinoma of the liver is sometimes seen in cases of cirrhosis, and arises from the liver cells which are endeavouring to regenerate. Similarly, squamous-celled carcinoma occasionally occurs in a chronic ulcer (fig. 47), also Marjolin's ulcer, p. 120), and a fibrosarcoma arising in a scar is not uncommon. Viruses cause cell instability and are therefore implicated. A wart is the commonest example of a virus causing a tumour (see also Burkitt's tumour, p. 195).

Carcinoma is not hereditary. However experience shows that it appears to ' run in families '.

Tumours reproduce cells which are similar to those from which they arise, although

if the tumour grows rapidly the resemblance becomes less obvious (*anaplasia*). It sometimes happens that the epithelium from which the tumour grows has already changed its characteristics. The gall-bladder is normally lined by columnar epithelium, but the presence of gall-stones may result in the epithelium undergoing squamous-celled *metaplasia*.

Classification

Teratomas arise from ' totipotent ' cells, containing representative cells from all three embryonic layers: ectoderm, endoderm, mesoderm. Teratomatous ' dermoids ', for example, contain hair, teeth, muscle, gland tissue. An unusual type is the sacrococcygeal teratoma (fig. 48) which can be considered as *fœtus in fœtu* (an ' included ' fœtus).

Blastomas develop from unipotent cells, and arise from any one of the three embryonic layers (e.g. neuroblastoma, p. 56).

Dermoid Cysts.—'Dermoid' is a loose term given to cysts lined by squamous epithelium occurring in various parts of the body. Sebaceous cysts are lined by superficial squamous cells and should more accurately be called ' epidermoids '.

(i) *Teratomatous dermoids* (see above) are found in the ovary, testis, retroperitoneum, superior mediastinum, and the presacral area. Malignant change (carcinomatous or sarcomatous) can occur.

(ii) *Sequestration dermoids* (p. 62) are not teratomas but are formed by the inclusion of epithelium beneath the surface at places where lines of developing skin meet and join (forehead (fig. 49), external angular process, Chap. 31, root of nose, Chap. 31, branchial cysts, Chap. 34).

(iii) *Implantation dermoids* (p. 63) may follow puncture wounds, commonly of the fingers.

FIG. 48.—This sacrococcygeal teratoma had taken the shape of a leg (a third leg!). It had rudimentary toes and nails. It was successfully operated on. (*A. C. Bose, M.S., F.A.C.S., Senior Consultant (Surgery) Armed Forces, India*).

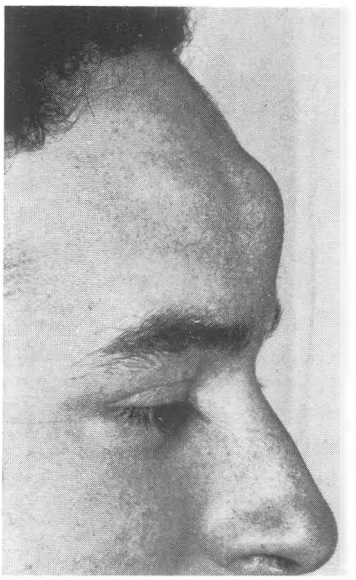


FIG. 49.—Dermoid cyst of forehead.

(iv) **Seeding.**—Implantation of carcinoma has been observed in situations where skin or mucous membrane is closely in contact with a primary growth. Examples of this '*kiss cancer*' are carcinoma of the lower lip affecting the upper, and carcinoma of the labium majus giving rise to a similar growth on the opposite side of the vulva. Recurrence after operation is occasionally due to *implantation* of malignant cells in the wound. Examples of this mischance are the appearance of a malignant deposit in the bladder scar after suprapubic removal of a primary growth, and nodules of carcinoma in the scar of the incision after mastectomy. When the peritoneum is involved, cells from a carcinoma may spread like snowflakes all over its serous surface. This *transcœlomic spread* is specially notable when cells from a colloid carcinoma of the stomach gravitate on to an active ovary and give rise to malignant ovarian tumours (Krukenberg's tumour, Chapter 43), which may be the first manifestation of gastric malignancy.

FIG. 51. — Melanomatous deposits in subcutaneous lymphatics of the abdominal wall. (*R.C.S. Museum.*)

GRADING AND STAGING OF MALIGNANT TUMOURS

Grading and staging are used to assess the degree of malignancy of the tumour as an indication of the prognosis, and may be used as a guide to determine the type and the extent of the treatment which is required.

Dukes' Staging.—This is a method of classifying the spread of carcinoma of the rectum and is described in Chapter 52.

TNM Classification.[1]—This is a detailed clinical staging which is arrived at simply by the clinician ascertaining the following points during his examination of the patient: What is the extent of the primary **T**umour? Are any lymph **N**odes affected? Are there any **M**etastases? The information so obtained is scored, *for example in carcinoma of the breast*, as follows:—

Tumour	**N**odes	**M**etastasis
T_1 = 2 cm. or less. No skin fixation.	N_0 = No nodes.	M_0 = No metastasis.
T_2 = More than 2 cm., but less than 5 cm. Skin tethered or dimpled. No pectoral fixation.	N_1 = Axillary nodes movable (a) not significant, (b) significant.	M_1 = Metastases are present including involvement of skin beyond breast, and contralateral nodes.
T_3 = More than 5 cm., but less than 10 cm. Skin infiltrated or ulcerated. Pectoral fixation.	N_2 = Axillary nodes fixed.	
T_4 = More than 10 cm. Skin involved but not beyond breast. Chest-wall fixation.	N_3 = Supraclavicular nodes. Œdema of arm.	

[1] Adopted by the International Union against Cancer. TNM Classification has been extended to many sites of cancer.

Cuthbert Esquire Dukes, 1890–1977. Consulting Pathologist, St. Mark's Hospital, London.

INNOCENT OR MALIGNANT

An **innocent or benign tumour** is usually encapsulated, and does not disseminate nor recur after removal. Symptoms, which can be dangerous, are due to its size, position, and pressure, and certain adenomas secrete a *hormone* which may affect bodily functions. Frequently innocent tumours are multiple.

Malignant Tumours.—The characteristics of malignancy are: (*a*) Invasion of surrounding tissues, (*b*) Pleomorphism (variable shapes) of cells, (*c*) Rapid growth, (*d*) the tendency to metastasise to other parts of the body by the lymphatics and the blood-stream, (*e*) general weight loss and a rapid downhill path.

It has been suggested that the division of tumours into these two major groups imposes a concept which is too rigid (Walter). A third group of intermediate tumours exists which includes carcinoid tumours, ' adenoma ' of the bronchus, ' mixed ' salivary tumours, basal cell carcinoma, etc.

METHODS OF SPREAD OF MALIGNANT TUMOURS

(i) **Local Extension** (**direct spread**).—This feature is the most reliable evidence of malignancy when the tumour is examined microscopically. Invasion takes place most readily along connective-tissue planes.

(ii) **Blood-stream.**—This is the most common method of dissemination of a *sarcoma*, as the venous clefts, so typical of a sarcoma, readily permit malignant cells to enter the blood-stream. These malignant emboli are liable to be arrested in the lungs, where they form secondary deposits. Large veins are sometimes extensively invaded by sarcoma (fig. 50).

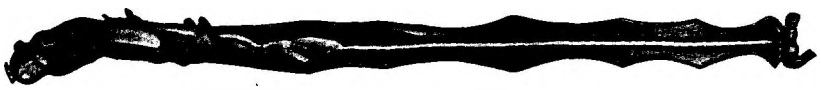


FIG. 50.—Intravenous spread of sarcoma.

Carcinoma disseminates less commonly by the blood-stream, although a carcinoma of the kidney may invade the renal vein and inferior vena cava. Cancer cells may be detected in the venous blood draining the region of a malignant tumour.

(iii) **Lymphatics.**—Carcinoma spreads by permeation and by embolism.

Permeation.—The malignant cells grow along the lymphatic vessels from the primary growth. This may even occur in a retrograde direction. The cancer cells stimulate perilymphatic fibrosis, but this does not stop the advance of the disease.

In some instances, notably malignant melanoma (p. 123), groups of cells may so overcome the surrounding fibrosis, as to give rise to intermediate deposits between the primary growth and the lymph nodes (fig. 51).

Embolism.—Cancer cells invade a lymphatic vessel and are carried by the lymph circulation to a regional node, so that nodes comparatively distant from the tumour may be involved in the early stages.

John B. Walter, Contemporary. Associate Professor of Pathology, University of Toronto, Canada.

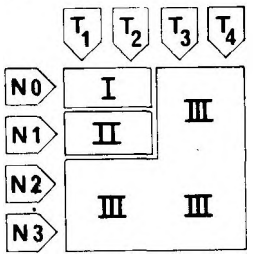


FIG. 52.—TNM scoring related to clinical stage grouping of breast cancer when there are no metastases.

Thus, for example, one patient may have a carcinoma which is $T_1N_0M_0$, while in another the extent of the disease may be $T_3N_2M_1$.

This scoring can be applied quite easily to the commonly used method of *clinical stage grouping* of carcinoma of the breast (described in Chap. 37), by the use of fig. 52. Metastases present is equivalent to Stage IV.

BENIGN TUMOURS

There are many types of benign tumours and in this section only the main groups will be considered.

Papilloma.—A papilloma consists of a central axis of connective tissue, blood-vessels, and lymphatics; the surface is covered by epithelium, either squamous, transitional, cuboidal or columnar, according to the site of the tumour. The surface may be merely roughened, or composed of innumerable delicate villous processes, as in the case of those occurring in the kidney, bladder and rectum. In these situations papillomas resemble malignant tumours, as secondary growths arise by implantation, and, sooner or later, the tumour becomes frankly malignant (Chapter 57). Other common sites for papillomas are the skin, the colon, the tongue and lip, the vocal cords and the walls of cysts, particularly those in connection with the breast and ovary.

Fibroma.—A true fibroma (containing only fibrous connective tissue) is rare. Most fibromas are combined with other mesodermal tissues such as muscle (fibromyoma), fat (fibrolipoma), and nerve sheaths (neurofibroma), etc. Multiple tumours are not uncommon, as, for example, in neurofibromatosis (von Recklinghausen's disease, p. 57).

Fibromas are either *hard* or *soft*, depending on the proportion of fibrous to the other cellular tissue. Soft fibromas are common in the subcutaneous tissue of the face, and appear as soft brown swellings.[1]

Desmoid.—This unusual type of fibroma occurs in the abdominal wall (Chap. 53).
Keloid (p. 24).

Lipoma.—A lipoma is a slowly growing tumour composed of fat cells of adult type. Lipomas may be encapsulated or diffuse.

Encapsulated lipomas are among the commonest of tumours. The characteristic features are the presence of a definite edge and lobulation. A sense of fluctuation may be obtained. These tumours can occur in any part of the body where fat is found. As would be expected, a lipoma deeply situated is liable to be mistaken for other swellings. Most lipomas are painless, but some give rise to an aching sensation which may radiate.

Multiple lipomas are not uncommon. The tumours remain small or moderate in size, and are sometimes painful, in which case the condition is probably one of *neurolipomatosis. Dercum's disease* (*adiposis dolorosa*),

[1] Oliver Cromwell (1599–1658), Lord Protector of England from 1653 until his death, was disfigured by one of these tumours which he called a wart. He insisted that his portraits should show him 'wart and all'.

Francis Xavier Dercum, 1856–1931. Neurologist, Jefferson Medical College, Philadelphia, Pennsylvania, U.S.A. Described Adiposis Dolorosa in 1888.

characterised by tender deposits of fat, especially on the trunk, is an associated condition.

Should the lipoma contain an excessive amount of fibrous tissue it is termed a *fibrolipoma*. In other cases considerable vascularity is present, often with telangiectasis of the overlying skin, in which case the tumour is a *nævolipoma*. A retroperitoneal lipoma, or one in the thigh (fig. 53), occasionally undergoes sarcomatous changes.

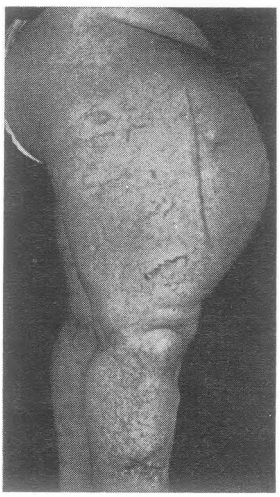


FIG. 53.— Liposarcoma of thigh which had been present for over twenty years. The scar indicates a previous attempt at removal.

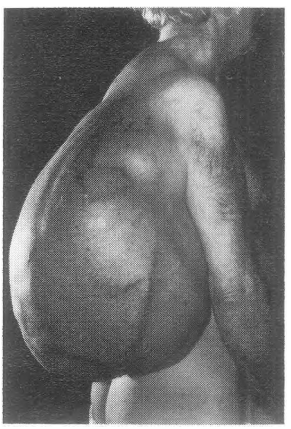


FIG. 54.—A giant sub-cutaneous lipoma, carried around for years. (*P. Roberts, F.R.C.S., The London Hospital.*)

Myxomatous degeneration, *saponification* and *calcification* sometimes occur in lipomas of long duration.

Clinically, circumscribed lipomas are classified according to their situation:

(i) *Subcutaneous* are most commonly found on the shoulders (fig. 54) or back, although no part of the body is immune. It must be remembered that a lipoma is occasionally present over the site of a spina bifida. A careful examination should distinguish such conditions as a tuberculous abscess or a sebaceous cyst, which may superficially resemble a lipoma. Subcutaneous lipomas occasionally become pedunculated (lipoma arborescens), or the influence of gravity may cause the tumour gradually to change its position.

FIG. 55.—Erosion of the skull due to a subaponeurotic lipoma.

(ii) *Subfascial.*—Lipomas occurring under the palmar or plantar fascia are liable to be mistaken for tuberculous tenosynovitis, as the tough, overlying fascia masks the definite edge and lobulation of the tumour. However, the swelling is circumscribed, and wasting of muscles is negligible. Difficulty is encountered in complete removal as pressure encourages the tumour to ramify. Subfascial lipomas also occur in the areolar layer under the epicranial aponeurosis, and if of long duration they erode the underlying bone, so that a depression is palpable on pushing the tumour to one side (fig. 55).

(iii) *Subsynovial* arise from the fatty padding around joints, especially the knee. They are apt to be mistaken for Baker's cysts (Chap. 20) but are easily distinguished as, in distinction to a cyst or bursa, their consistency is constant whether the joint is in extension or flexion.

(iv) *Intra-articular.*

(v) *Intermuscular.*—These occur particularly in the thigh or around the shoulder. Owing to transmitted pressure the tumour becomes firmer when the adjacent muscles are contracted. Weakness or aching results, owing to mechanical interference with muscular action. The condition is often difficult to distinguish from a fibrosarcoma, and exploration may be necessary.

(vi) *Parosteal* occasionally occur under the periosteum of a bone.

(vii) *Subserous* are not common, but are sometimes found beneath the pleura, where they constitute one variety of innocent thoracic tumour. A retroperitoneal lipoma may grow to enormous dimensions, and simulate a hydronephrosis or pancreatic cyst. A lipomatous mass is frequently found at the fundus of the sac of a femoral hernia, but this is a condensation of extra-peritoneal fat.

(viii) *Submucous* occur under the mucous membrane of the respiratory or alimentary tracts. Very rarely a submucous lipoma in the larynx causes respiratory obstruction. A submucous lipoma occasionally occurs in the tongue (fig. 595). One situated in the intestine is likely to cause an intussusception, which is the first indication of its presence.

(ix) *Extradural.*—A lipoma is a rare variety of spinal tumour. Owing to the absence of fat within the skull intracranial lipomas do not occur.

(x) *Intraglandular.*—Lipomas have been found occasionally in the pancreas, under the renal capsule and in the breast (see Chapter 37).

As will be noted, lipomas are very widely distributed in the body. The ' universal tumour ' or the ' ubiquitous tumour ' are appropriate terms, which should be remembered when a swelling causes diagnostic difficulty.

Treatment.—If a lipoma is causing trouble on account of its site, size, appearance, or the presence of pain, removal is indicated.

During operation, any finger-like projections of the tumour into the surrounding tissue must also be removed. Although the tumour is relatively avascular, care is needed to obtain complete hæmostasis in the resulting cavity—drainage often is necessary—otherwise a hæmatoma is common, which may be followed by infection and delay in wound healing.

Diffuse lipoma occasionally occurs in the subcutaneous tissue of the neck, from which it spreads on to the preauricular region of the face. The tumour is not obviously encapsulated, and gives rise to no trouble beyond being unsightly.

Adenoma.—Adenomas arise in secretory glands, and resemble the structure from which they arise. They are encapsulated, and sometimes they profoundly influence metabolism, as in the case of the thyroid, parathyroid and pancreas. Occasionally an adenoma contains a large proportion of fibrous tissue, e.g. the hard fibro-adenoma in the breast, while in other situations, notably the pancreas and thyroid gland, cystic degeneration is common. Those arising from secretory glands of mucous membrane are liable to pedunculation, as in the case of rectal ' polypus '.

Neuroma.—True neuromas are rare tumours, and occur in connection with the sympathetic system. They comprise the following types:

(*a*) *Ganglioneuroma* (Chap. 36) which consists of ganglion cells and nerve fibres. It arises in connection with the sympathetic cord, and therefore is found in the retroperitoneal tissue, or in the neck or thorax.

(*b*) *Neuroblastoma*, which is less differentiated than the ganglioneuroma, the cells being of an embryonic type (p. 51). The tumour somewhat resembles a round-celled sarcoma, and disseminates by the blood-stream. It occurs in infants and young children. It may occasionally undergo spontaneous remission.

(*c*) *Myelinic neuroma*, is very rare, being composed only of nerve fibres, as the ganglion cells are absent. They arise in connection with the spinal cord or pia mater.

Neurilemmoma (*syn.* Schwannoma).—These lobulated and encapsulated tumours arise from the neurilemmal cells. They are soft and whitish in appearance. They displace the nerve from which they arise and can be removed (fig. 56).

Neurofibroma arise from the connective tissue of the nerve sheath. The following varieties are described.

Local.—A single neurofibroma is usually found in the subcutaneous tissue. The 'painful subcutaneous nodule' forms a smooth firm swelling which may be moved in a lateral direction, but is otherwise fixed by the nerve from which it arises. Paræsthesia or pain is likely to occur from pressure of the tumour on the nerve fibres which are spread over its surface. Cystic degeneration or sarcomatous changes occasionally occur.

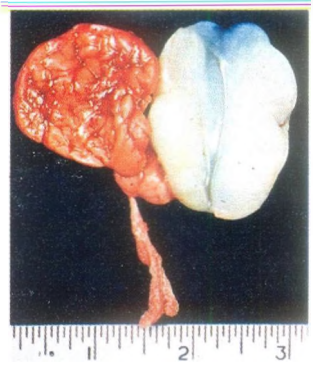


FIG. 56.—Neurilemmoma of the lingual nerve attached to submandibular salivary gland.

Neurofibromas may also grow from the sheath of a peripheral nerve or a cranial nerve (e.g. the acoustic tumour, Chap. 26). As the nerve fibres are 'part and parcel' of the tumour they are difficult to remove without removal of the nerve itself. In major nerves recurrence is known, also malignant (sarcomatous) change.

Generalised Neurofibromatosis (*syn.* von Recklinghausen's Disease of Nerves).—In this inherited (autosomal dominant) disease, any cranial, spinal or peripheral nerve may be diffusely or nodularly thickened (fig. 57). The overgrowth occurs in connection with the endoneurium. Associated pigmentation of the skin is common, and sarcomatous changes may occur.

FIG. 57.—Neurofibromatosis.

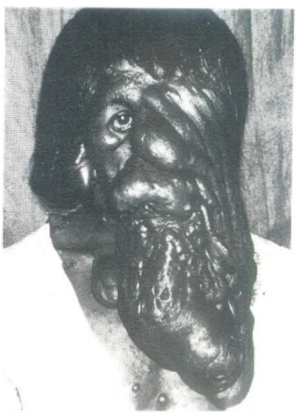


FIG. 58.—Plexiform Neurofibromatosis of the 5th cranial nerve. (*Dr. N. G. Nayar, Kerala*)

Friedrich Daniel von Recklinghausen, 1833–1910. Professor of Pathology, Strasbourg, France (1872–1906). Described Generalised Neurofibromatosis in 1882.

Plexiform Neurofibromatosis.—This rare condition usually occurs in connection with branches of the fifth cranial nerve (figs. 58 and 59), although it may occur in the extremities (fig. 59). The affected nerves become enormously thickened as a result of myxofibromatous degeneration of the endoneurium. If occurring in the scalp, the underlying skull may be eroded, and in other situations the involved skin sometimes hangs down in pendulous folds with grotesque effects (figs. 58, 59 and 60). Plexiform neurofibromatosis is sometimes associated with the generalised type of neurofibromatosis. Sarcoma rarely develops.

Elephantiasis Neuromatosa is a rare and congenital condition. The skin is coarse, dry, and thickened, resembling an elephant's hide, and the subcutaneous tissues become greatly thickened. If a leg is affected, the patient finds walking increasingly difficult.

False Neuroma.—Arises from the connective tissue of the nerve sheath after injury to a nerve (lacerations or amputation). These swellings consist of fibrous tissue and coiled nerve fibres (p. 170).

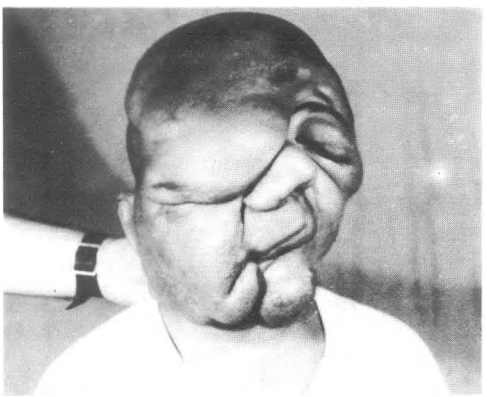


FIG. 59.—Plexiform neurofibromatosis of the 5th cranial nerve. (*Dr. P. Nayak, M.S., Bombay.*)

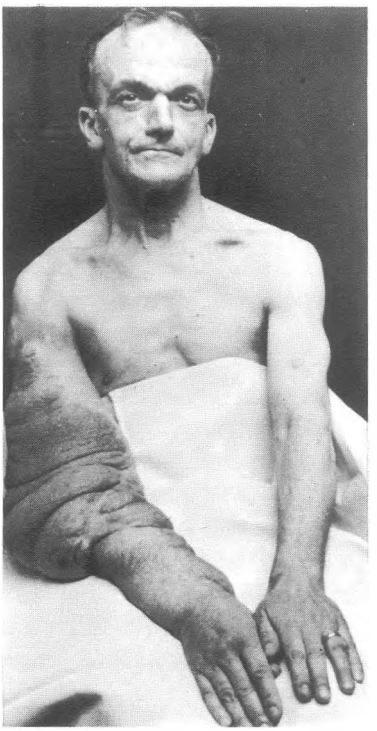


FIG. 60.—Plexiform neurofibromatosis affecting the right arm.

Hæmangiomas are described in Chapter 9 on p. 118.

Glomangioma (*syn.* Glomus Tumour).—These tumours arise from a cutaneous glomus composed of a tortuous arteriole which communicates directly with a venule, the vessels being surrounded with a network of small nerves. These specialised organs regulate the temperature of the skin, and are found in the limbs, especially the nail-beds. The tumour is compressible. The associated pain is out of all proportion to the size of the tumour, which may be only a few millimetres in diameter. The pain is burning in nature and radiates peripherally, and is often more noticeable when the limb is exposed to sudden changes of temperature.

On section the tumour consists of a mixture of blood spaces, nerve tissue and muscle fibres derived from the wall of the arteriole (angiomyoneuroma). Large cuboidal cells are frequently seen (glomal cells). Glomal tumours grow very slowly, and do not become malignant. They should be excised.

Hamartoma.—The term hamartoma is roughly translated from the Greek[1] as a 'fault' or 'misfire' or 'error' tumour, and its original meaning was 'missing the mark in spear throwing'. It is a developmental malformation consisting of a tumour-like overgrowth of tissue or tissues proper to the part. The possible range

[1] Hamartano (Greek) = I miss.

therefore is very wide, and the lesions are often multiple. Common lesions that are hamartomas are benign pigmented moles, and the majority of angiomas and neurofibromas. On rare occasions a malignant change occurs in a hamartoma, but for practical purposes the lesion is benign (Peters).

MALIGNANT TUMOURS

CARCINOMA

Carcinomas are conveniently classified according to the type of cell from which they arise, i.e. glandular, squamous, or basal-celled.

(i) **Glandular** commonly occurs in the alimentary tract, breast and uterus, and less frequently in the kidney, prostate, gall-bladder, and thyroid. The three types of glandular carcinoma are as follows:—

(*a*) *Carcinoma simplex*, in which the cells are arranged in circumscribed groups, no glandular structure being recognisable. This type commonly occurs in the breast, and the majority of cells are spheroidal in shape.

(*b*) *Adenocarcinoma*, so called from the tendency of the cells to form acini, which resemble those of the gland from which they are derived. The alveoli are ductless, and the walls are composed of layers of cells which invade the surrounding tissues. The cells of the primary growth, and even of the metastases, sometimes retain secretory powers.

(*c*) *Colloid* (mucoid) is a degenerative process which develops in tumours arising from mucin-secreting cells. The mucin permeates the stoma of the growth, which appears as a gelatinous mass and is typically seen in the colon and stomach.

Glandular carcinoma is also subdivided into various types, e.g. encephaloid, scirrhus and atrophic scirrhus. These distinctions depend clinically on their rate of growth, and pathologically on the relative proportions of fibrous tissue and gland elements. Examples occur in the breast (Chap. 37).

(ii) **Squamous** arises from surfaces covered by squamous epithelium, particularly as a result of chronic irritation. Also chronic irritation of transitional cells (e.g. by a stone in the renal pelvis) or columnar cells (e.g. the gall bladder) will cause a change in these cells to a squamous type (*squamous metaplasia*) and so lead on to carcinoma. The regional lymph nodes are likely to be invaded, and may also be infected from the sepsis attendant upon the primary growth. Blood-borne metastases occur, but rarely from skin carcinoma.

Macroscopically, squamous-celled carcinomas are either papilliferous or ulcerative. On section solid masses of polyhedral cells are seen, which invade the deeper structures. ' Cell-nests ' are usually apparent in slowly growing cases, and are due to deeper cells becoming flattened and undergoing keratinisation. ' Prickle ' cells are characteristic, and resemble the prickle cells present in the epidermis.

(iii) **Basal-celled** (*syn.* Rodent Ulcer, p. 120).

SARCOMA[1]

Sarcomas occur in connection with structures of mesoblastic origin. They differ from carcinomas, not only in their derivation, but also in their

[1] Sarkos (Greek) = Flesh.

Philip Michael Peters, 1916–1977. Pathologist, Royal Northern Hospital, London.

age incidence, as sarcomas are most common during the first and second decades. Sarcomas often grow with rapidity, and dissemination occurs mainly by the blood-stream.

The macroscopic appearance of a sarcoma varies considerably. As the word implies, most tumours appear as a fleshy mass, but their consistency depends on the relative proportion of fibrous and vascular tissue. Hæmorrhage commonly occurs owing to the very thin walls of the veins, which in some places are represented merely by venous spaces.

Sarcomatous cells may reproduce tissue similar to that from which the tumour originated, e.g. osteosarcoma or chondrosarcoma. In some cases a sarcoma develops in pre-existing benign tumours, such as a fibroma or a uterine fibroid, and also in bones which are affected by osteitis deformans (p. 300).

Sarcomas of separate organs are considered in their appropriate chapters.

Fibrosarcoma is composed of spindle cells of varying lengths (the rounder they are the more malignant they are), and occurs in muscle sheaths, scars and as a fibrous epulis (Chap. 31). A fibrosarcoma of a muscle sheath presents as an elastic or firm and slowly growing swelling. Dilated veins over the tumour suggest malignancy, and if not obvious they may be demonstrated by infra-red photography. On palpation the tumour often feels warm and pulsation may even be detected.

Recurrent Fibroid.—Fibrosarcomas not uncommonly arise in scar tissue, sometimes many years after the scar developed.[1]

Treatment of Fibrosarcoma.—Spread of a sarcoma is hastened by incomplete efforts at removal. *The moral is that wide excision with surrounding healthy tissues should be practised in all cases.* This may mean amputation in the case of a limb. If untreated, or if wide local excision is unsuccessful, a fibrosarcoma eventually fungates through the skin. Metastases are widely scattered, and, unfortunately, radiotherapy has but little effect on either the primary growth or on the secondary deposits.

Reticulosarcoma (syn. lymphosarcoma) arises in lymph nodes, tonsils, Peyer's patches or lymph nodules in the intestines. Lymph nodes of the neck or mediastinum are most commonly affected (p. 190).

Synovioma.—This rather uncommon tumour may arise in any synovial joint or tendon sheath, especially those of the hand. It appears as a soft, painless swelling, and sarcomatous changes may develop. Diagnosis can only be established by extirpation and biopsy of the tumour.

Nævus and Melanoma are described in Chapter 9.

Endothelioma. Mesothelioma[2].—The endothelial linings of blood-vessels, lymphatic spaces and serous membranes occasionally give rise to neoplasms. They can be malignant. They arise from the pleura (Chapter 40) and rarely from the pericardium or peritoneum. The original cells are flattened, but they become spheroidal or cuboidal when neoplastic changes occur. The ' endothelioma ' (meningioma)

[1] Sir James Paget, 1814–1899, Surgeon, St. Bartholomew's Hospital, London, described this type as a ' recurrent fibroid '.

[2] ' Blue ' asbestos fibres have been shown to be a cause.

Johann Conrad Peyer, 1653–1712 (*see p. 37*).

of the dura mater is thought to arise from the arachnoid membrane, which is *not* an endothelial structure (Chap. 26).

Peritheliomas are tumours arising in the endothelial lining of small blood-vessels or lymphatics. Carotid body tumours are probably of this nature (Chapter 34).

BENIGN ⟶ MALIGNANT

Certain innocent neoplasms are prone to undergo malignant changes, and it is important, both for treatment and prognosis, to realise when this occurs. Some or all of the following changes may be recognised:

(i) *Increase in size*—comparatively rapid enlargement is always suspicious, e.g. a neurofibroma which is becoming sarcomatous.

(ii) *Increased vascularity*—dilated cutaneous veins, expansile pulsation of the tumour, or ulceration and bleeding in the case of a superficial growth.

(iii) *Fixity*—due to invasion of surrounding structures, e.g. carcinomatous changes in a prostatic adenoma may cause ' tethering ' of the rectal mucosa.

(iv) *Involvement of adjacent structures*—carcinomatous changes in an adenoma of the thyroid should be suspected if the recurrent laryngeal nerve is implicated. Facial palsy suggests a malignant parotid tumour.

(v) *Dissemination*—discovery of secondary deposits is occasionally the clue to malignancy. Examples.—The true nature of a doubtful lump in the breast is rendered evident if involved lymph nodes or osseous metastases are detected. The discovery of a secondary deposit in a bone may lead to detection of, say, a carcinoma of the kidney.

CYSTS

The word ' cyst ' is derived from the Greek word meaning ' bladder '. The pathological term ' cyst ' means a swelling consisting of a collection of fluid in a sac which is lined by epithelium or endothelium.

True cysts are usually lined by epithelium or endothelium. If infection supervenes, the lining may be composed of granulation tissue. The fluid is usually serous or mucoid and varies from brown-staining by altered blood to almost colourless. In epidermoid, dermoid, and branchial cysts the contents are like porridge or toothpaste, as a result of the accretion of desquamated cells. Cholesterol crystals are often found in the fluid.

False Cysts.—Certain collections of fluid are not regarded as true cysts. They are usually exudation and degeneration cysts. A pseudo-cyst of the pancreas is an encysted collection of fluid in the lesser sac. In tuberculous peritonitis fluid may be walled off in cystic form by adherent coils of intestine. Fluid may collect in the centre of a tumour (cystic degeneration), due to hæmorrhage or colliquative necrosis. This can also happen in the brain as a result of ischæmia, and an ' apoplectic cyst ' is formed.

A Classification of Cysts:-

1. CONGENITAL
   - Sequestration dermoids.
   - Tubulo-embryonic (tubulo-dermoid).
   - Cysts of embryonic remnants.

2. ACQUIRED — Retention. Distension. Exudation. Cystic swellings. Implantation. Trauma. Degeneration.

3. PARASITIC — Hydatid. Trichiniasis. Cysticercosis.

Congenital Cysts.—*The sequestration dermoid* is due to dermal cells being buried along the lines of closure of embryonic clefts and sinuses by skin fusion (fig. 61). The cyst is therefore lined by epidermis and contains paste-like desquamated material. The usual sites are (*a*) the midline of the body—especially in the neck, (*b*) above the outer canthus (external angular dermoid) (Chap. 31), (*c*) in the anterior triangle of the neck (branchial cyst, Chap. 34).

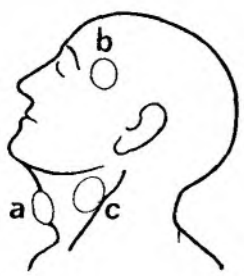


FIG. 61.—Sequestration dermoids—see text.

Tubulo-embryonic (*tubulo-dermoid*) *cysts* occur in the track of an ectodermal tube used in development, e.g. a thyroglossal cyst from the thyroglossal duct; a post-anal dermoid from the post-anal gut. In the brain, ependymal cysts arise from the sequestration of cells of the infolding neurectoderm.

Cysts of Embryonic Remnants.[1]—These arise from embryonic tubules and ducts which normally disappear or are only present as remnants. There are many examples in the urogenital system, e.g. in the male from remnants of the paramesonephric duct (Müllerian)—the hydatid of Morgagni, or from the mesonephric body and duct (Wolffian) (Chapter 56). Cysts of the urachus, and the vitello-intestinal duct are other examples of cysts of embryonic remnants.

Acquired Cysts.—*Retention cysts* are due to the accumulated secretion of a gland following obstruction of a duct. Examples are seen in the pancreas, the parotid, the breast, the epididymis, and Bartholin's gland. A sebaceous cyst starts with the obstruction of a sebaceous gland, but this is followed by the downgrowth and the accumulation of desquamated epidermal cells, thus making it an epidermoid cyst.

Distension cysts occur in the thyroid from dilatation of the acini, or in the ovary from a follicle. Lymphatic cysts and cystic hygromas are distension cysts. *Exudation cysts* occur when fluid exudes into an anatomical space already lined by endothelium, e.g. hydrocele, a bursa, or when a collection of exudate becomes encysted. These are false cysts (p. 61).

Cystic Tumours.—Examples are cystic teratomas (dermoid cyst of the ovary (p. 51)) and cystadenomas (pseudomucinous, and serous cystadenoma of the ovary).

[1] These cysts are not to be confused with teratomatous cysts (e.g. Dermoid, p. 51).

Johannes Peter Muller, 1801–1858. Professor of Anatomy and Physiology, Berlin.
Giovanni Battista Morgagni, 1682–1771. Professor of Anatomy, Padua, Italy.
Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad), U.S.S.R.
Caspar Bartholin, Secundus, 1655–1738. Professor of Medicine, Anatomy, and Physics, Copenhagen, Denmark.

Ganglia (see p. 311).

Implantation dermoids arise from squamous epithelium which has been driven beneath the skin by a penetrating wound. They are classically found in the fingers of women who sew assiduously (fig. 62). The contents are therefore desquamated cell-debris which may undergo mucoid degeneration.

FIG. 62.—Implantation dermoid.

Trauma.—A hæmatoma may resolve into a cyst. This sometimes happens to hæmatomas of muscle masses in the loin and antero-lateral aspects of the thigh or the shin. They are located between muscle, fascial, or subcutaneous planes and contain straw- or brown-coloured fluid containing cholesterol crystals. They become lined by endothelium and calcium salts may be laid down. Aspiration is only of temporary value, and a cure depends upon complete excision of the lining.

Degeneration Cysts.—These have already been discussed under false cysts.

Parasitic Cysts.—These are encysted forms in the life-cycle of various worms.

Hydatid Cyst of the *Tænia Echinococcus.*—This is described later according to the organ involved, e.g. liver, Chapter 45; lung Chapter 40.

Trichiniasis.—Cysts of the *Trichina spiralis*, affecting muscle.

Cysticercosis.—Cysts of *Tænia solium.* A disease of the pig, man being rarely affected. Eosinophilia is present. The cysts occur in any organ. They calcify and may cause clinical effects according to their situation, especially in the brain. Only those cysts which are actually causing symptoms should be excised.

Clinical Features of a Cyst.—The swelling usually has a smooth spherical appearance. Fluctuation depends upon the pressure of fluid within; a tense cyst feels like a solid tumour, though careful palpation between two fingers may elicit a characteristic elasticity. In addition, a solid tumour is most hard at the centre; a cyst is least hard at the centre. If fluctuation is present, a cyst may be confused with a cold abscess or a lipoma. A cold abscess usually has a peculiar rim of thickening surrounding the soft centre. A lipoma may well test clinical acumen. *Transillumination*, while brilliantly clear in cysts containing serous fluid, does not really distinguish between a lipoma and a dermoid or branchial cyst. There is even an old axiom that ' when in doubt hedge on fat '. According to circumstances, a test aspiration or excision reveals the true nature of the swelling.

Cysts may be painful, especially when infection or hæmorrhage causes a sudden increase in the intracystic tension. Sometimes they change in size for no apparent reason. Occasionally they diminish due to rupture through a fascial plane.

Effects of a Cyst.—These are according to site and size. As with benign tumours, a cyst may compress ducts and blood-vessels. For example, the common bile duct may be obstructed by a choledochal cyst, a renal cyst, or a hydatid cyst. The pelvic veins may be obstructed by an ovarian

cyst, the patient presenting for treatment of her varicose veins. The sheer size of an ovarian cyst may so increase intra-abdominal tension as to bring the patient to hospital with symptoms of a hiatus hernia.

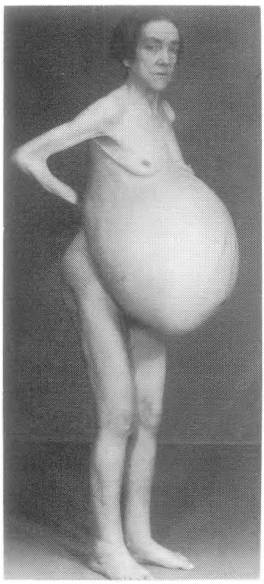


FIG. 63.—Cachexia ovarica.

Complications.—*Infection.*—The cyst becomes tense and painful, and adherent to surrounding tissues. An abscess may form and discharge on the surface and result in an ulcer or a sinus (viz. Cock's peculiar tumour, p. 114). Healing will not occur until the whole lining of the cyst or the embryonic track is excised.

Hæmorrhage.—Sudden hæmorrhage, as may occur in a thyroid cyst, causes a painful increase in size. In this particular case breathing may be difficult because of pressure on the trachea.

Torsion may occur in cysts which are attached to neighbouring structures by a vascular pedicle. Ovarian dermoids are sometimes brought to notice in this way as acute abdominal emergencies. The cyst (or cysts—they may be bilateral), turns to a purple or black colour as the venous, and then the arterial supply is cut off.

Calcification follows hæmorrhage, or infection, and may be the result of reaction to a parasite, e.g. hydatid cyst.

Cachexia Ovarica.—Enormous cysts are rarely seen nowadays; however fig. 63 shows a patient who successfully underwent an operation for a giant ovarian cyst containing 15 litres of fluid. The site, size, and weight of the cyst combine to cause cachexia, lordosis, œdema of the legs, and an anxious appearance!

ULCERS

An ulcer is a discontinuity of an epithelial surface. There is usually progressive destruction of surface tissue, cell by cell, as distinct from death of macroscopic portions (e.g. gangrene or necrosis). Ulcers are classified as *non-specific*, *specific* (e.g. tuberculous or syphilitic), or *malignant*.

Non-specific ulcers are due to infection of wounds, or physical or chemical agents. Local irritation, as in the case of a dental ulcer, or interference with the circulation, e.g. varicose veins (p. 178), are predisposing causes.

Trophic Ulcers[1] are due to an impairment of nutrition, which normally depends upon an adequate blood supply and a properly functioning nerve supply. Ischæmia and anæsthesia will therefore cause these ulcers. Ulcers of the tips of the fingers occur in chronic vasospasm (Raynaud's disease, p. 163)

[1] Trophē (Greek) = Nutrition.

and in syringomyelia. In the leg, ischæmic ulcers occur around the ankle or on the dorsum of the foot (p. 137). They are very painful and resistant to local treatment. Ulcers due to anæsthesia (diabetic neuritis, spina bifida, tabes dorsalis, leprosy, or a peripheral nerve injury) are often called *perforating ulcers* (fig. 64). Starting in a corn or bunion, they penetrate the foot, and the suppuration may involve the bones and joints and spread along fascial planes upwards, even involving the calf.

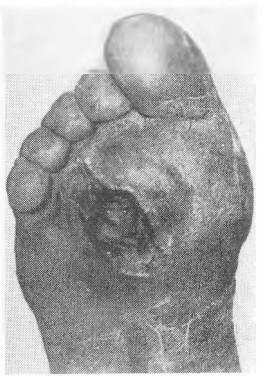


FIG. 64.—Perforating ulcer in a diabetic.

The life-history of an ulcer consists of three phases; these are extension, transition, and repair. During the *stage of extension* the floor is covered with exudate and sloughs, while the base is indurated. The edge is sharply defined, and discharge is purulent and even blood-stained. The *transition stage* is occupied in preparation for healing. The floor becomes cleaner, the sloughs separate, induration of the base diminishes, and the discharge becomes more serous. Small reddish areas of granulation tissue appear on the floor, and these link up until the whole surface is covered (fig. 6). The *stage of repair* consists of the transformation of granulation to fibrous tissue, which gradually contracts to form a scar. The edge of the ulcer becomes more shelving, and epithelium gradually extends from it to cover the floor (at a rate of 1 mm per day). This healing edge consists of three zones—the outer of epithelium, which appears white, the middle one bluish in colour (where granulation tissue is covered by a few layers of epithelium), and the inner reddish zone of granulation tissue covered by a single layer of epithelial cells.

Local (Topical) Treatment of Non-specific Ulcers.—Any underlying cause is treated (e.g. varicose veins (Chap. 11) diabetes, arterial disease). Many lotions and non-adhesive applications are used to aid the separation of sloughs, hasten granulation, and stimulate epithelialisation. Hypochlorite solution (e.g. Eusol[1]) and 0·5% silver nitrate are popular in the early stages, and 1% zinc sulphate solution (e.g. Lotio rubra) later[2]; ointments incorporating zinc oxide, 1% hydrocortisone cream; silver foil; household vinegar (1:6) (efficacious against pyocyaneus). Excessive granulations, commonly known as ' proud flesh ', need to be discouraged by excision, curettage, or by the application of a caustic, such as silver nitrate. Tetracycline (10%) in propylene glycol solution helps to clean an ulcer prior to skin grafting. Chronic or indolent ulcers often respond to infra-red radiation, short-wave therapy, or ultra-violet light.

Large ulcers in the healing phase should be covered by a free split skin graft as soon as the granulations are healthy (p. 131). Healthy granulations are flat, are below the level of the surrounding skin, are pink in colour, and do not discharge pus.

Amnion has returned to fashion as an effective application to promote healing. Fresh from the delivery room, it is cleaned thoroughly by sterile gauze or brush, washed in a suitable hypochlorite solution, cut in to size and applied like a skin graft. The rough chorion side helps to stimulate granulations, the smooth amnion side epithelialisation. Excess amnion is stored at 4°C in saline and used when replacement is necessary every 2 to 5 days (usually determined by infection).

[1] Eusol = *E*dinburgh *U*niversity *SOL*ution.
[2] Zinc sulphate capsules (200 mg) thrice daily by mouth have been recommended.

CLINICAL EXAMINATION OF AN ULCER

This should be conducted in a systematic manner. The following are, with brief examples, the points which should be noted:

Site, e.g. 95 per cent. of rodent ulcers (p. 120) occur on the upper part of the face. Carcinoma typically affects the lower lip, while a primary chancre of syphilis is usually on the upper.

Size, particularly in relation to the length of history, e.g. a carcinoma extends more rapidly than a rodent ulcer, but more slowly than an inflammatory ulcer.

Shape, e.g. a rodent ulcer is usually circular. A gummatous ulcer is typically circular (fig. 65), or serpiginous, due to the fusion of multiple circles.

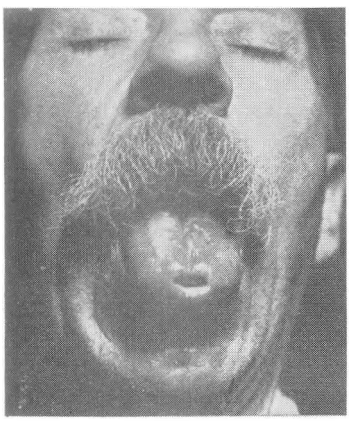


FIG. 65. — Syphilitic gumma of the tongue. Note the punched-out appearance of the ulcer.

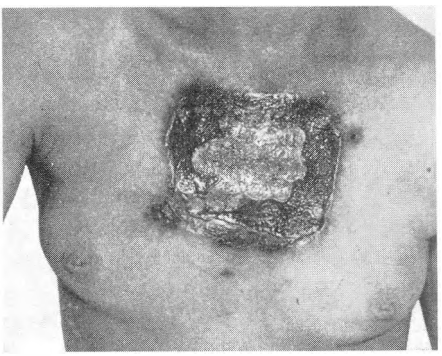


FIG. 66.—Dermatitis artefacta. This condition is due to self-mutilation, e.g. by the application of irritants, such as corrosives. The patient usually has a hysterical temperament, or litigation may be involved. The ulcer will begin to heal if protected by a dressing which cannot be disturbed by the patient.

An ulcer with a square area or straight edge is suggestive of ' dermatitis artefacta ' (fig. 66).

Edge (fig. 67).—A healing non-specific ulcer has a shelving edge. It is undermined and often bluish if tuberculous, vertically punched-out if syphilitic (fig. 65), rolled or rampart if a rodent ulcer, and raised and everted if malignant (e.g. epithelioma).

Floor.—The floor is that which is seen by an observer, e.g. watery or apple-jelly granulations in a tuberculous ulcer, a wash-leather slough in a gummatous ulcer.

Base.—The base is what can be palpated. It may be indurated as in a carcinoma or attached to deep structures, e.g. a varicose ulcer to the tibia.

Discharge.—A purulent discharge indicates active infection. A blue-green coloration suggests infection with *Pseudomonas pyocyaneus*. A watery

FIG. 67.—1. Healing ulcer. 2. Tuberculous ulcer. 3. Rodent ulcer. 4. Epithelioma.

discharge is typical of tuberculosis. It is blood-stained in the extension phase of a non-specific ulcer. *Bacteriological examination* may reveal colonisation by coagulase positive staphylococci. Spirochætes are found in a primary chancre (p. 45).

Lymph nodes are not enlarged in the case of a rodent ulcer, unless due to secondary infection. In the case of carcinoma, they may be enlarged, hard, and even fixed. The inguinal nodes draining a syphilitic chancre of the penis are firm and ' shotty ', but the submandibular nodes draining a chancre of the lip are greatly enlarged.

Pain.—Non-specific ulcers in the extension and transition stages are painful (except the anæsthetic trophic type). Tuberculous ulcers vary, that of the tongue being very painful. Syphilitic ulcers are usually painless, but the anal chancre of the homosexual may be painful (cf. anal fissure, Chap. 52).

General Examination.—Evidence of debility, cardiac failure, all types of anæmia, including sickle cell anæmia, or diabetes must be sought.

Pathological examinations, e.g. biopsy, may confirm carcinoma. The serological and Mantoux tests may be of value.

Marjolin's Ulcer. See p. 120.

ORIENTAL SORE (*syn.* DELHI BOIL, BAGHDAD SORE, ETC.)

This disease is due to infection by a protozoal parasite, *Leishmania tropica*, and is a common condition in Eastern countries which is occasionally imported to Western zones. An indurated papule appears on an exposed surface, usually the face. If untreated this breaks down to form an indolent ulcer (fig. 68), which eventually leaves an ugly, pigmented scar. The condition readily responds to intravenous injections of antimony tartrate, but very small lesions can be treated by carbon dioxide snow.

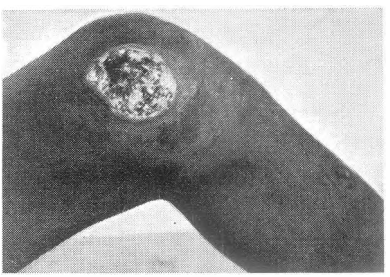


FIG. 68.—Oriental sore, boy aged eight years. *(Professor Riyaz-Qadeer, F.R.C.S., Lahore.)*

FIG. 69.—Bazin's disease.

Bazin's disease (*syn.* erythema induratum) is due to localised areas of fat necrosis and particularly affects adolescent girls. Symmetrical purplish nodules appear, especially on the calves, and gradually break down to form indolent ulcers, which leave in their wake pigmented scars (fig. 69). Tuberculosis may be a cause in many instances, the ulcers responding to antituberculous drugs (p. 37). Sympathectomy also may be curative.

SINUSES AND FISTULÆ

A sinus[1] is a blind track lined with granulations leading from an epithelial surface into the surrounding tissues. *A fistula*[2] is an abnormal communication between the lumen of one viscus and the lumen of another, or

[1] Sinus (Latin) = A hollow; a bay or gulf. [2] Fistula (Latin) = A pipe or tube.

Charles Mantoux, 1877–1947. Physician, Le Cannet, France.
Sir William Leishman, 1865–1926. Professor of Pathology, Royal Army Medical College.
Pierre Antoine Ernest Bazin, 1807–1878. Dermatologist, Hôpital St. Louis, Paris.

the body surface. Sinuses and fistulæ may be congenital or acquired. Forms which have a congenital origin include pre-auricular sinuses (p. 530), branchial fistulæ (Chap. 34), and tracheo-œsophageal fistulæ (Chap. 42). The acquired forms often follow inadequate drainage of an abscess. Thus a perianal abscess may burst on the surface and lead to a sinus (erroneously termed a blind external ' fistula '). In other cases the abscess opens both into the anal canal and on to the surface, resulting in a true fistula-in-ano (Chap. 52).

Persistence of a Sinus or Fistula.—The reason for this will be found amongst the following:

(1) A foreign body or necrotic tissue is present (e.g. a suture, sequestrum, a fæcolith or even a worm (see below)).

(2) Inefficient or non-dependent drainage.

(3) Unrelieved obstruction of the lumen of a viscus or tube distal to the fistula. Irritating discharges, such as urine or fæces, maintain continuous inflammation.

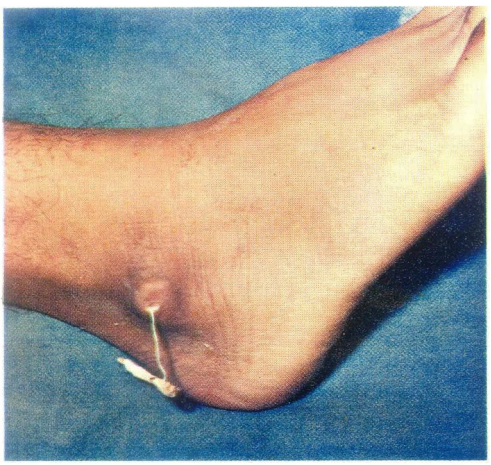


FIG. 70.—?Ulcer ?Sinus—presenting in a U.K. outpatient department! The 30-cm female Guinea worm is carefully extricated by being wound on a matchstick, a few turns a day to prevent breakage and retraction.

(4) Absence of rest, such as occurs in fistula-in-ano due to the normal contractions of the sphincter.

(5) The walls have become lined with epithelium.

(6) Dense fibrosis prevents contraction and healing.

(7) Type of infection, e.g. tuberculosis or actinomycosis.

(8) The presence of malignant disease.

Treatment. — The remedy depends upon the removal or specific treatment of the cause.

The treatment of sinuses and fistulæ affecting different sites and organs will be found on the appropriate pages.

Guinea Worm (Syn. Dragon worm, *Dracunculus mediensis*) (fig. 70). The larval form enters through the wall of the stomach or duodenum in drinking water containing a tiny cyclops crustacean which has consumed the larvæ. In the abdominal connective tissue male and female mate, the pregnancy lasts about a year, and the female wanders in the subcutaneous tissues to select a part of the anatomy likely to be submerged in water (containing the cyclops), usually the lower leg. Cellulitis, abscesses, ulcers and sinuses follow through which the embryos are discharged, hopefully to be eaten by the cyclops. Baid travelled the interior of India and in 500 cases discerned a syndrome of the infestation, presenting with conjunctivitis (allergic) in 11%, fibrous contracture of joints in 19%, periostitis with osteomyelitis in 21% and acute arthritis in 65%.

J. C. Baid, Contemporary. Surgeon, Jodhpur, India.

CHAPTER 7

HÆMORRHAGE. BLOOD TRANSFUSION. SHOCK

HÆMORRHAGE

TYPES OF HÆMORRHAGE

1. Arterial. Venous. Capillary.

Arterial hæmorrhage is bright red. The blood spurts as a jet which rises and falls in time with the pulse. In protracted bleeding, particularly when quantities of intravenous fluids other than blood are given, it can become watery in appearance.

Venous hæmorrhage is a darker red and escapes as a steady flow. The colour darkens still further from excessive oxygen desaturation when blood loss is severe, or in respiratory depression or obstruction.[1]

Venous bleeding can be under increased pressure as in asphyxia, or from ruptured varicose veins (Chap. 11). Also portal vein pressures (Chap. 41) are high enough to cause rapid blood loss, especially in portal hypertension with œsophageal varices. Pulmonary artery hæmorrhage is dark red (venous blood) at around 30 mmHg* (≈4kPa), and bleeding from the pulmonary veins is bright red.

Capillary hæmorrhage is a bright red, often rapid ooze. If continuing for many hours blood loss can become serious (as in hæmophilia, p. 80).

2. Primary. Reactionary. Secondary.

Primary hæmorrhage is that which occurs at the time of injury or operation.

Reactionary hæmorrhage may follow primary hæmorrhage within 24 hours (usually 4–6 hours) and is mainly due to rolling (' slipping ') of a ligature or dislodgement of a clot. The precipitating circumstances are (a) the rise of blood pressure and the refilling of the venous system on recovery from shock, (b) restlessness, coughing and vomiting which raise the venous pressure (e.g. reactionary venous hæmorrhage within a few hours of thyroidectomy).

Penetrating wounds involving main veins in the thigh or groin are potentially fatal, as exsanguination may follow the removal of a first-aid dressing which has apparently controlled the bleeding (butcher's thigh, p. 147). Such a wound should never be treated in a perfunctory manner; it requires careful examination and closure in an operating theatre. Venous hæmorrhage, whether primary or reactionary, can tax the skill of even an experienced surgeon.

Secondary hæmorrhage occurs after 7–14 days, and is due to infection and sloughing of part of the wall of an artery. Predisposing factors are pressure

[1] Occasionally dark blood is due to the presence of sulph- or met-hæmoglobin.

* 1 mmHg ≈ 133 Pascals in the S.I. system. Since blood pressure is commonly measured by a column of mercury, the Editors find themselves in sympathy with the Society for the Preservation of the mmHg.

of a drainage tube, a fragment of bone, a ligature in an infected area, or cancer. It is also a complication of arterial surgery and amputations. It is heralded by ' warning ' hæmorrhages, which are bright red stains on the dressing, followed by a sudden severe hæmorrhage which may be fatal. A warning hæmatemesis may occur in the case of a peptic ulcer and is a danger signal which it is imprudent to ignore. In advanced cancer the erosion of a main vessel (e.g. carotid, uterine) by a locally ulcerating growth is a swift and merciful termination to the patient's suffering.

3. Internal (concealed) and External (revealed) Hæmorrhage.

Internal bleeding may be *concealed* as in ruptured spleen or liver, fractured femur, ruptured ectopic gestation, or in cerebral hæmorrhage. Concealed hæmorrhage may become *revealed* as in hæmatemesis or melæna from a bleeding peptic ulcer, as in hæmaturia from a ruptured kidney, or via the vagina in accidental uterine hæmorrhage of pregnancy.

SIGNS OF HÆMORRHAGE. PHYSIOLOGICAL AND PATHOLOGICAL EFFECTS (OLIGÆMIC SHOCK)

Acute Blood Loss.—*Signs.*—External bleeding is obvious, but a serious internal hæmorrhage must be recognised without delay by the general signs of blood loss:— *increasing pallor and pulse rate, restlessness* and, in protracted bleeding, deep sighing respirations (*air hunger*). These signs are accompanied by a cold and clammy skin, empty veins, and later, by thirst, tinnitus and blindness (see also fig. 74).

The pulse volume will be low (thready pulse) as the blood-pressure falls, though it is important to remember that this is *not* a sign to be relied upon. A normal or raised blood-pressure can occur in the face of other signs of hæmorrhage in an apparently healthy person, and collapse and death can occur suddenly. This phenomenon was noticed in the first world war (1914–18) by front-line research.

Effects.—In short, blood loss threatens the oxygen supply to, and therefore the life of, tissue cells. Pending the arrest of hæmorrhage and the replacement of blood, the function of vital structures such as the heart and brain stem is largely preserved by the increasing pulse rate and peripheral vasoconstriction (even though vasoconstriction may have deleterious effects on the liver, kidneys and also the adrenals). Unchecked or untreated blood loss results in failure of the heart and vasomotor centre to maintain a sufficient perfusion of oxygen for their own purposes and death follows (see death cycle—below).

Hæmorrhage results in loss of circulating blood volume from the whole of the vascular system. 60–70 per cent. of the blood volume is accommodated in the low pressure venules and veins and in the splanchnic vessels, and a loss of up to 10 per cent. (500–600 ml.) is adequately compensated for by venoconstriction and therefore the contraction of this large venous reservoir. (The spleen contributes only about 50 ml.) Further blood loss results in a diminished venous return and therefore a fall in cardiac output. The blood pressure, and therefore the perfusion of oxygen, is threatened, so a fall is prevented by increased heart rate and vasoconstriction. This is effected via sympathetic nervous activity, adrenaline, noradrenalin and the adrenal corticoids—which probably sensitise the vasoconstrictor nerve endings to noradrenalin.

Clinically this compensation is shown as pallor, cold clammy skin, empty veins and increased pulse rate. Restlessness comes with the effects of hypoxia on the midbrain (mesencephalic reticulum) while changes in the rate and depth of respiration are due partly to effects on the brain stem (chemoreflex), and partly in response to an increase in the airway dead space and metabolic acidosis.

The brain and the myocardium, which are the structures vital to the preservation of life in this emergency, are spared from vasoconstriction. The kidneys and even the liver may suffer irreparable damage from the tissue anoxia caused by the intense and prolonged vasoconstriction in severe blood loss, with acute renal failure or liver failure claiming victims, even though the hæmorrhage is successfully treated (e.g. in ruptured abdominal aneurysm).

Vasoconstriction fails to maintain the blood pressure when blood loss increases beyond 20–30 per cent. Hypotension becomes severe with a 50 per cent. loss, and the perfusion of myocardium and the brain stem is affected. Thus the heart fails and vasoconstriction of the venous reservoir fails—each affecting the other in a vicious circle which ends in death (the death cycle).

Failure of vasoconstriction will occur sooner if any conditions causing vasodilation are present (alcohol, anæsthesia, infection, heating the patient by too many blankets or a heat cradle).

Infants are intolerant of blood loss because a small amount may represent a considerable percentage of the total blood volume, and also because the peripheral circulatory resistance is normally high and so cannot be so effectively increased. In the elderly, arteriosclerosis affecting brain, heart and the peripheral circulation will hinder an adequate response.

Natural Blood Volume and Red Cell Recovery.—The recovery of blood volume begins immediately by the withdrawal of fluid from the tissues into the circulation. There is hæmodilution. Plasma proteins are replaced by the liver. Red cell recovery takes some five to six weeks. The iron content will be less than normal if stores are depleted or absorption is impaired (e.g. after gastrectomy, Chap. 43).

Chronic Hæmorrhage.—Examples of causes in surgical practice are bleeding hæmorrhoids, fibroids, carcinoma of the caecum, peptic ulcer. There is no diminution of the blood volume as there is time for plasma replacement, but red cell replacement lags behind (microcytic hypochromic anæmia) resulting in a state of anæmic hypoxia, requiring an increased cardiac output. These patients develop high-output cardiac failure; they must not be transfused with normal blood, but require packed cells instead. Acute hæmorrhage in such cases is poorly compensated, as oxygen carriage is already depleted.

CLINICAL AND LABORATORY MEASUREMENT OF HÆMORRHAGE

1. **Bedside observation** of the appearance of the patient is essential (see signs of hæmorrhage, p. 70). The severity of blood loss and shock can be gauged by feeling the temperature of the nose. A cold nose usually means that shock is severe and peripheral failure is imminent.

2. **Record keeping** of the pulse rate and the blood pressure are also essential (in spite of the fact that the blood pressure can be misleading). Readings are taken at ¼ or ½ hourly intervals during an emergency and thereafter 4-hourly. The patient is not roused from sleep for a blood pressure reading if the pulse is not raised.

3. **Hæmoglobin level.**—This is estimated in g/100 ml. (g/dl), normal values being 12–16 g/100 ml. (12–16 g/dl). There is no immediate change in

hæmorrhage, but within the space of a few hours the level is lowered by the hæmodilution caused by the movement of tissue fluid into the vascular compartment in order to restore the blood volume. The degree of dilution can be gauged by the hæmatocrit reading (P.C.V.).

4. The hæmatocrit (fig. 71) is a stout glass tube 110 mm long with a 2·5–3·00 mm bore and it is graduated 0–100. It is filled up to the 100 mark with a small quantity (about 0·8 ml) of oxalated or heparinised freshly drawn blood, and centrifuged at 3,000 rev/min for 30 minutes. The packed cell volume (P.C.V.) is then read from the millimetre reading gradations. The normal range for venous blood is: men, 40–56 per cent; women, 35–48 per cent; children, 32–44 per cent. The P.C.V. can also be measured on capillary blood, using very small amounts, in the microhæmatocrit centrifuge. This is particularly useful in shocked patients where it is often difficult to obtain a sample from a vein.

FIG. 71.— A hæmatocrit.

As explained above, some hours after the start of hæmorrhage, the P.C.V. will be lowered by a hæmodilution. In dehydration states and the plasma loss in burns, the P.C.V. will be increased (hæmoconcentration), because the plasma volume is diminished without a concomitant loss of red cells and hæmoglobin.

5. Measuring Blood Loss

(*a*) *Blood Clot.*—Blood clot the size of a clenched fist is roughly equal to 500 ml.

(*b*) *Swelling in Closed Fractures.*—Moderate swelling in closed fracture of the tibia equals 500–1,500 ml blood loss. Moderate swelling in a fractured shaft of femur equals 500–2,000 ml blood loss (Ruscoe Clarke).

(*c*) *Swab Weighing.*—In the operating theatre, blood loss can be measured by weighing the swabs before and after use and adding the total so obtained (1 g = 1 ml) to the volume of blood collected in the suction or drainage bottles. In extensive wounds and operations, the blood loss estimated in this way is much less than the total amount of blood, plasma and water actually lost from the body. For operations such as radical mastectomy or partial gastrectomy it is necessary to multiply the swab weighing total by a factor $1\frac{1}{2}$.[1] For larger wounds, as in abdomino-thoracic or abdomino-perineal operations, multiply by a factor of 2. *Washing swabs* and estimating the Hb content of the washings is another method.

(*d*) *Blood Volume Determinations.*—Either the volume of circulating plasma, or the volume occupied by the red cells, must be measured first. The hæmatocrit reading will give the ratio of plasma to red cells, and thus the total blood volume can be arrived at. *Methods.*—The measurement of the circulating plasma or red cell volume involves the injection of a known quantity of a substance which will be evenly diluted as it mixes with, and circulates in, the blood. Radioactive iodinated serum albumin (RISA) is used to give plasma volume, and red cells ' tagged ' with radiochromium or radioiron are used for red cell volume measurement. After allowing time for the substance to mix, a sample of blood is withdrawn. A measured volume of this sample is examined in the scintillation counter to give the dilution of the amount originally injected, and from this the whole of the diluting volume (plasma or red cells) can be arrived at. The normal blood volume is 70–85 ml. of whole blood per kilogram body weight, or 35–50 ml. plasma/kg. Thus it varies between a few hundred ml. in a

[1] These important factors (Rains factors) were established in a series comparing swab-weighing with the measurement of blood volume.

Alan Ruscoe Clarke, 1908–1959. Surgeon, Birmingham Accident Hospital, Birmingham, England.

neonate, up to 5–6 litres in an adult (more in certain pathological conditions, e.g. high output cardiac failure from chronic anæmia or arteriovenous fistula).

(*e*) *Measurement of Central Venous Pressure* (*C.V.P.*) (figs. 72, 74 and 75).

An immediate definitive diagnosis of a collapsed patient is often difficult. Hæmorrhage, pulmonary embolism, cardiac infarction, mesenteric thrombosis, acute pancreatitis, also pleural, peritoneal, intestinal and bacteræmic shock may present similarly. Delay in giving blood or other intravenous fluids (too little as well as too late) to those in need of it, denies them the chance of survival, while on the other hand, a patient's condition may be worsened by infusion if there is right or left ventricular failure, or cardiac tamponade. Measurement of the C.V.P. is of practical value because it indicates the state of the venous reservoir, the venous return, the cardiac efficiency, and therefore whether a transfusion should be given, continued, or discontinued.

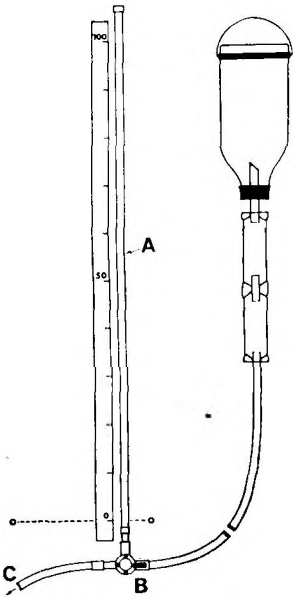


FIG. 72.—Saline manometer for measuring central venous pressure. A = The sterile glass or plastic tube manometer against a centimetre scale; a spirit level is used to set zero (0) to the manubriosternal angle or the mid axillary point. B = The three-way stop-cock allows (i) saline to run via C into the vein from the reservoir, (ii) to fill the manometer, and (iii) to exclude the reservoir and to allow the fluid in A to fall to the level of the C.V.P.

Method.—A commercially available intravenous catheter, made to proper standards and of requisite length (20 cm), is passed into the right internal jugular vein. The surface marking of the vein is a line drawn between the lobe of the ear and the medial end of the clavicle. With the patient in the head-down position, and using full aseptic precautions, the catheter needle, mounted on a syringe, is either inserted (a) downwards in the line of the vein after entrance through the skin some 3–4 finger breadths above the sternoclavicular joint, usually through the anterior fibres of the sternomastoid muscle, or (b) through a skin puncture 3 cm above the medial end of the clavicle in the hollow between the two heads of the sternomastoid muscle and beneath which lies the lower bulb of the vein, the needle being first aimed in the direction of the suprasternal notch. The head-down position is used to distend the vein and to prevent air being sucked in (air embolism p. 156). Insertion (a) is best avoided in the unanæsthetised patient.

The catheter tip should be positioned in the superior vena cava (confirmed radiologically at the first opportunity). Preceding every measurement the patency of the catheter is confirmed by lowering the saline reservoir briefly to check the free reflux of blood in the connecting tubing (fig. 72C). It must be emphasised that the use of this method requires supervised training, skill, practice and patience, also reference to the special manuals, because the complications can be serious (e.g., pneumothorax, hæmothorax, brachial plexus and major artery damage). The catheter must be removed when not required for C.V.P. measurement, and should not be kept in position as a matter of convenience for electrolyte or parenteral infusions (the latter entering the pleural cavity or the mediastinum can be lethal).

The infra-clavicular route tends to be reserved for parenteral nutrition (p. 109).

An alternative, though perhaps less accurate method, is the insertion of a longer catheter (60 cm) via the median basilic vein in the antecubital fossa. It may be difficult to position the tip so as to be certain that central pressure is being measured.

The catheter is connected to the saline manometer* (fig. 72). Readings with the zero reference point at the manubriosternal angle (angle of Louis) are 0–2 cm = low, 10 + = high, and at the mid axillary point are 0–5 cm = low, 15 + = high. Therefore, if the C.V.P. is low the venous return should be supplemented by intravenous infusion, but not if the pressure is high.

THE TREATMENT OF HÆMORRHAGE

This can be summarised:—

1. Stop the blood loss by—Pressure and Packing. Position and Rest. Operation. Ligation, Repair, Excision.
2. Restore blood volume by—Blood Transfusion (p. 76): Saline; Dextran (p. 81); Plasma (p. 81).

Methods of Stopping Hæmorrhage

Pressure and Packing.—*The first-aid* treatment of hæmorrhage from a wound is a pressure dressing, this being made from anything handy which is soft and clean (linen), but even a rolled up forage cap stuffed into a wound has been effective in battle (Drummond). The dressing or pack should be bound on tightly.

Other examples of pressure being used to control hæmorrhage include: the use of forefinger and thumb or a clothes-peg for epistaxis (Chap. 31); the use of a double balloon in the œsophagus and the stomach to control the bleeding from œsophageal varices (Chap. 45).

Packing by means of rolls of wide gauze is an important standby in operative surgery. If several rolls are used the ends must be tied together to ensure complete removal later.

N.B.—If on removal of pressure or packing, bleeding appears to have ceased completely, do not assume that all is well, especially when dealing with deep wounds involving large veins. Continued close observation is required.

Tourniquets are to be avoided in *first aid.* They can only be used around the thigh or upper arm; they are difficult to apply tightly enough to interrupt arterial blood flow, and only cause venous congestion and increased blood loss. There have been many examples of tourniquets of some kind being used for ruptured varicose veins causing exsanguination by only occluding venous return. (For example, a policeman was brought into a casualty department with seven tourniquets in place. The venous bleeding ceased as soon as they were removed!) Tourniquets tight enough to obstruct arterial flow may cause contusion of the artery wall and thrombosis, especially if atheroma is present. Nerve conduction can also be seriously impaired, while to leave a tourniquet on for over an hour is to invite muscle death and the risk of renal failure (crush syndrome, p. 86). To sum up, it can be said that tourniquets are dangerous because they may be applied too loosely, too tightly, or for too long.

In the operating theatre tourniquets are used (a) to control hæmorrhage temporarily while exploration and repair are being carried out: (b) for some amputations (not for atherosclerotic gangrene); (c) in order to obtain a bloodless field for orthopædic and soft tissue operations (cartilage, tendon, nerves, ganglia of tendon sheaths). The sphygmomanometer cuff is very satisfactory as a pneumatic tourniquet; the cuff is inflated after elevation of the limb for five minutes has materially reduced the vascularity. Alternatively, a bloodless operation field can be obtained by an Esmarch

* Since C.V.P. is measured in centimetres using a column of saline, no alterations to S.I. units of pressure will be used.

Antoine Louis, 1723–1792. Surgeon, Paris.
Sir William Alexander Duncan Drummond, Contemporary. Formerly Director-General, Army Medical Service (1956–1961).
Johann Friedrich August von Esmarch, 1823–1908. Professor of Surgery, Kiel, Germany (1857–1899). Developed the 'Esmarch Bandage' whilst working as a Military Surgeon during the Franco-Prussian War (1870–1871), and reported on its use in 1873.

bandage applied spirally to the elevated limb commencing distally, and then bound on tightly. The distal spirals are unwound to expose the limb for operation.

Removal of a Tourniquet.—The fact that a tourniquet is in place and the time of application must be written large on the theatre blackboard and deleted only when it is removed. It is a wise precaution to attach the tourniquet to the operating table, so that it cannot be overlooked when the patient is lifted on to the trolley.

Venous Tourniquet.—A light rubber or a pneumatic tourniquet is applied to the upper arm prior to intravenous injections and sampling. It is also used in the diagnosis of varicose veins (p. 180).

Position and Rest.—*Elevation of limbs* (in ruptured varicose veins) employs gravity to reduce bleeding. Elevation also causes vasoconstriction (Lister). *A bed elevator* is often used to raise the foot of the bed as an aid to vasoconstriction in reducing the size of the vascular compartment, so facilitating blood flow to the brain and the restoration of a satisfactory blood pressure. Gravity is also used in certain operations, as in thyroidectomy when the patient is tilted feet downwards (anti-Trendelenburg position) or as in stripping of varicose veins when a head-down tilt is used (Trendelenburg).

In the ward as well as the theatre, Esmarch bandages may be applied temporarily to the limbs in order to reduce the vascular compartment in shock, so aiding the heart to maintain blood flow to vital structures.

Absolute rest is vital. This means nursing the patient in a comfortable recumbent position, and he should be relieved of unnecessary exertion. He does not wash or feed himself, and assistance is given for turning in bed, which must be at least two-hourly to prevent pressure sores.

Sedation also brings rest. Morphine (10–20 mg) relieves pain, calms restlessness, and aids coronary and cerebral blood flow. Though it is contraindicated where there is respiratory depression (as in head injuries), its use is invaluable where respiratory movements and coughing are inhibited because of chest pain. Morphine is best given intramuscularly, or intravenously in hæmorrhage and shock. A subcutaneous dose is not easily absorbed because of vasoconstriction, and if repeated doses are given in this way an excessive amount is liable to be absorbed when the circulation improves. Morphine does not in itself produce sleep, and the patient, though relieved of pain, may remain wakeful and anxious. Amylobarbitone Sodium (120 mg) should be used if there is no respiratory depression, otherwise chloral hydrate (1–2 g).

Examples of Operative Techniques in Hæmorrhage.—Artery forceps (e.g. Spencer Wells) or ' hæmostats ' are mechanical means of controlling bleeding by pressure. The clamped vessel can be ligated with catgut, cotton, thread or silk, or coagulated with diathermy. When an incision is made through the scalp for craniotomy, the profuse bleeding is not easily arrested by direct forcipressure, so the cranial aponeurosis is picked up by a series of forceps which are everted together, thus exerting pressure. Silver clips (Cushing) may be applied to cerebral vessels.

Suturing may be employed. The vessel can be underrun or transfixed by needle and suture and then ligated, while if the continuity of a main vessel is to be restored, 4/0 silk or polypropylene is used (p. 146) on a 20-mm atraumatic needle.

Pressure by packing, using rolls of wide gauze has been previously mentioned, but light pressure with a ' peanut ' of gauze held by forceps aids the sealing of an arterial suture line after reconstruction following trauma, embolectomy, or in artery grafting. About five minutes is required for the platelets to seal the join.

Patches of vein or Dacron mesh may be used to repair a vascular defect. A patch of muscle, lightly hammered, provides thrombokinase to stop a troublesome ooze.

Joseph Lister (Lord Lister), 1827–1912 (see footnote, p. 1).

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Rostock (1875–1882), Bonn (1882–1895), and Leipzig (1895–1911). The Trendelenburg Position was first described in 1885.

Sir Thomas Spencer Wells, 1818–1897. Surgeon, Samaritan Free Hospital for Women and Children, London. Introduced the improved version of his Pressure Forceps, used today, in 1879.

Harvey Williams Cushing, 1869–1939. Professor of Surgery, Johns Hopkins University, Baltimore, Maryland, U.S.A. (1903–1912), and Harvard Medical School, Boston, Massachusetts U.S.A. (1912–1932).

Other topical applications for oozing include gauze or sponge which is absorbed by the body. 'Oxycel' or gelatin sponge provides a network upon which fibrin and platelets can be deposited. This is the modern counterpart of the use of cobwebs by our forefathers, or sphagnum moss by our neolithic ancestors.

Gauze soaked in adrenalin soln. (1:1,000) or 'Stypven' (Russell viper venom) are other topical applications. Bone wax (Horsley) is used for oozing bone.

Excision of a bleeding viscus may be necessary, e.g. a ruptured spleen must be excised. However, a ruptured kidney is treated conservatively if possible (Chap. 56).

BLOOD TRANSFUSION

The indications for transfusion in surgical practice are as follows:—

1. Following traumatic incidents where there has been severe blood loss, or hæmorrhage from pathological lesions, *e.g.* from gastrointestinal tract.

2. During major operative procedures where a certain amount of blood loss is inevitable, *e.g.* abdomino-perineal or cardiovascular surgery.

3. Following severe burns where, despite initial fluid and protein replacement there may be associated hæmolysis.

4. Post-operatively in a patient who has become severely debilitated and anæmic as the result of infection, septicæmia, *etc.*

5. Pre-operatively in cases of chronic anæmia where surgery is indicated urgently, i.e. where there is inadequate time for iron or other replacement therapy (see Blood Fractions p. 77 and Complications p. 79), or where the anæmia is unresponsive to therapy, e.g. aplastic anæmia.

6. To arrest hæmorrhage or as a prophylactic measure prior to surgery, in a patient with a hæmorrhagic state such as thrombocytopenia, hæmophilia or liver disease. (See Blood Fractions below.)

Preparation of Blood Products for Transfusion.—It is important that blood donors should be fit and with no history of serious disease, in particular hepatitis which is transmitted in donor blood.

Blood is collected into a sterile commercially prepared plastic bag with needle and plastic tube attached in a complete, closed sterile unit.

With the donor lying on a couch, a sphygmomanometer cuff is applied to the upper arm and inflated to a pressure of 70 mmHg (9·3 kPa) or 80 mmHg (10·6 kPa). After introducing 0·5 ml of local anæsthetic, the 15 gauge needle is introduced into the median cubital vein and 410 ml of blood allowed to run into the bag containing 75 ml of anticoagulant solution (ACD).[1]

During collection the blood is constantly mixed with the anticoagulant to prevent clotting and at the end of the procedure the tube is clamped and the needle removed. Specimens for use in blood grouping and cross-matching procedures may be obtained by clamping off small sections of the plastic tubing containing the donor blood.

Blood Storage.—It is essential that all blood for transfusion be stored in special blood bank refrigerators controlled between temperatures of 2°C and 7°C. Blood allowed to stand at higher temperatures for more than 2 hours is in danger of transmitting infection, whereas at temperatures below 2°C there is liable to be hæmolysis of the red cells resulting in a severe pyrexial reaction and possibly renal damage in the recipient.

Red blood cells remain suitable for transfusion for at least 3 weeks. The cells from blood stored for this length of time, however, show some changes in metabolism resulting in a temporary reduction in their ability to release oxygen to the tissues of the recipient. This defect is restored in the body in about 72 hours and is therefore

[1] (ACD. Acid citrate dextrose solution containing 2·2 g anhydrous dextrose, 2·2 g disodium citrate monohydrate, 800 mg citric acid to 75 ml water. The solution is placed in the bag and sterilised.)

Patrick Russell, 1727–1805. Physician who practised for a time at Aleppo, Syria, and later became naturalist to the East India Company. He produced a memoir on Poisonous Snakes in 1787.

Sir Victor Alexander Haden Horsley, 1857–1916. Surgeon, University College Hospital, London. Horsley's Wax consists of seven parts of Beeswax to one part of Almond Oil. (Died of heatstroke or paratyphoid fever in Mesopotamia. One of the original Authors (McN. L.) attended his funeral.)

probably not clinically significant except in a patient requiring massive transfusion. In this case it is wise to give at least one or two units of blood which are less than 7 days old.

White blood cells are rapidly destroyed in blood stored by these procedures. *Blood platelets* similarly do not remain functional for more than 72 hours.

Blood Fractions.—Whole blood may be divided into various fractions. This is not only more economical of blood donors but certain fractions are more appropriate than whole blood transfusion for certain clinical conditions. Fractionation procedures are relatively safe and simple, using sealed sterile plastic bag units.

1. *Packed red cells.*—These are prepared by centrifugation of whole blood at 3,000 rev/min and removal of the supernatant plasma. Packed red cell transfusion is especially advisable in patients with chronic anæmia, in the elderly, in small children, and in patients in whom introduction of large volumes of fluid may cause cardiac failure. Packed red cells are suitable for most forms of transfusion therapy including major surgery especially in association with clear fluids.

2. *Platelet rich plasma.*—This is prepared by centrifugation of freshly donated blood at 1,000 rev/min for 20 minutes. The supernatant plasma containing the majority of the platelets is separated and is suitable for transfusion to patients with thrombocytopenia who are either bleeding or require surgery.

3. *Platelet concentrate.*—This is prepared by centrifugation of platelet-rich plasma at 3,000 rev/min, the supernatant plasma is removed, and the precipitate forms a very rich source of platelets for transfusion to patients with thrombocytopenia.

4. *Plasma.*—This is removed after centrifugation of whole blood at 3,000 rev/min and it may be further processed or fractionated in various ways.

(a) *Freeze-dried plasma.*—Plasma from 8–10 units of blood may be pooled and lyophilised. The resulting product may be stored for many years in the dried form and reconstituted by dissolving in sterile distilled water. Owing to its potential danger of transmitting serum hepatitis its use has been largely superseded by purified protein fraction (see below).

(b) *Fresh frozen plasma.*—Plasma removed from fresh blood obtained within 4 hours is rapidly frozen by immersing in solid carbon dioxide and ethyl alcohol mixture. This is stored at −20°C and is a good source of all the coagulation factors. It is the treatment of choice when considering surgery in patients with abnormal coagulation due to severe liver failure. It may also be given in any of the congenital clotting factor deficiency diseases especially *Christmas disease* (Factor IX deficiency) or hæmophilia (Factor VIII deficiency).

(c) *Cryoprecipitate.*—When fresh frozen plasma is allowed to thaw at 4°C a white glutinous precipitate remains and if the supernatant plasma is removed, this cryoprecipitate is a very rich source of Factor VIII. It is stored at −20°C and immediately available for treatment of patients with hæmophilia (Factor VIII deficiency). The advantage of cryoprecipitate treatment in hæmophilia is the simplicity of administering large quantities of Factor VIII in relatively small volumes by intravenous injection. It is also a rich source of fibrinogen, of value in hypofibrinogenæmic states.

(d) *Purified protein fraction.*—Repeated fractionation of plasma by organic liquids followed by heat treatment results in a plasma fraction rich in protein but free from the danger of transmission of serum hepatitis. This may be stored for several months in liquid form at 4°C and is suitable for replacement of protein, e.g. following severe burns.

(e) *Fibrinogen.*—This is prepared by organic liquid fractionation of plasma and is stored in the dried form. When reconstituted with distilled water it is suitable for transfusion in patients with severe depletion of fibrinogen, e.g. disseminated intravascular coagulation or congenital afibrinogenæmia.

Blood Grouping and Cross-Matching

Human red cells have on the cell surface many different antigens. For practical purposes, there are two groups of antigens which are of major importance in surgical practice:

(1) **Antigens of the ABO Blood Groups.**—These are strongly anti-

genic and are associated with naturally occurring antibodies in the serum. Individuals show four different ABO cell groups viz:

Red cell group	*Serum contains*
A	Anti B antibody
B	Anti A antibody
AB	No ABO antibody
O	Anti A and Anti B antibody

(2) **Antigens of the Rhesus Blood Groups.**—The antigen of major importance in this group is Rh(D) which is strongly antigenic and is present in approximately 85% of the population in the United Kingdom. Antibodies to the D antigen are not naturally present in the serum of the remaining 15% of individuals but their formation may be stimulated by the transfusion of RhD positive blood. Such acquired antibodies are capable, during pregnancy, of crossing the placenta and if present in a RhD negative mother may cause severe hæmolytic anæmia and even death (hydrops fœtalis) in a Rhesus D-positive fœtus *in utero*. The other minor blood group antigens may be associated with naturally occurring antibodies or may stimulate the formation of antibodies on relatively rare occasions.

Incompatibility.—If antibodies present in the recipient's serum are incompatible with the donors cells a transfusion reaction will result. This is the result of agglutination and hæmolysis of the donated cells leading in severe cases to acute renal tubular necrosis and renal failure. For this reason therefore, it is essential that all transfusions should be preceded by:—

(1) *ABO and Rhesus grouping* of the recipient's and donor's cells so that only ABO and RhD compatible blood is given.

(2) Direct matching of the recipient's serum with the donor's cells to confirm ABO compatibility and to test for any minor blood group antibody in the serum of the recipient.

Blood grouping and cross-matching require full laboratory procedures, each technique requiring 2 hours. In emergencies it may be necessary to reduce this time but the risk of undertaking urgent techniques must be weighed against the danger to the patient by the delay in transfusion entailed by the full laboratory procedures. In such emergencies it may be advisable to restore the patient's blood volume by dextran, plasma or saline, until blood has been made available. Alternatively donor blood, group O Rhesus D-negative, which is compatible with the majority of individuals should be given and this should always be available in acute emergency situations.

Giving Blood.—Blood transfusion is commenced by:—

(a) selection and preparation of the site (p. 96).

(b) careful checking of the donor blood:—this should bear a compatibility label stating the patient's name, hospital reference number, ward and blood group.

(c) Insertion of the needle or cannula:—the latter may be valuable if intravenous therapy is required for any length of time.

(d) Giving detailed written instructions as to the rate of flow (e.g. 40 drops per minute allows one 540 ml unit of blood to be transfused in 4 hours).

In acute emergencies it may be necessary to increase the rate of flow and it is possible to give 1–2 units in 30 minutes by a pressure cuff around a plastic bag of blood.

Warming Blood.—During cardiopulmonary operations the blood may be warmed before reaching the patient by passing it through a blood warming unit thus reducing the risk of cardiac arrest from large volumes of cold blood direct from the refrigerator.

Filtering Blood.—See fig. 73.

Autotransfusion.—This is an old, well-tried method of immediately restoring a patient's blood volume, transfusion with his or her own blood. For example, in a case of ruptured ectopic gestation, the blood is collected from the peritoneal cavity and put into a sterile container suitable for connecting to transfusion tubing. The classical method of filtration of this blood to prevent the transfusion of any small clots is to place a piece of sterile gauze within the container. Nowadays special autotransfusion apparatus is being marketed.

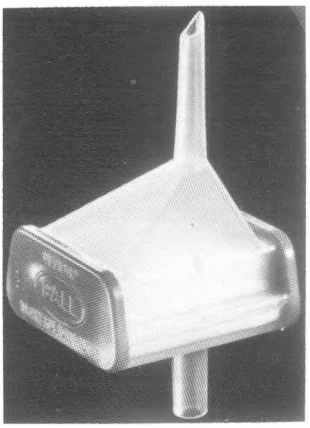


FIG. 73.—This Pall® filter has an absolute filtration rating of 40 μm (i.e. this the largest opening in the filter element). It will filter off platelet aggregates and leucocyte membranes in stored blood. *(Pall Biomedical Limited, Portsmouth, PO6 1TD, England.)*

Complications of Blood Transfusion

(1) **Congestive Cardiac Failure.**—This is especially liable to occur in the elderly or where there is cardiovascular insufficiency and may result from too rapid infusion of large volumes of blood. It is advisable in the individual with chronic anæmia to give packed red cells and at the same time give diuretic drugs. The transfusion should be given slowly, *i.e.* 1 unit over 4–6 hours and if necessary on 2 separate occasions.

(2) **'Transfusion Reactions'.**—These may be the result of:—

(i) *Incompatibility.*—This should be avoided if the correct procedures of grouping and cross-matching have been adopted but in fact it is nearly always due to human error in the collection, labelling or checking of the specimens and donor bags. The patient develops a rigor, temperature, pain in the loins and may become extremely alarmed. The transfusion should be stopped immediately and a fresh specimen of venous blood and urine from the patient be sent together with the unwashed donor bag to the laboratory for checking.

A close watch should be kept on the patient's pulse, blood pressure and urinary output. Mannitol i.v. (p. 82) may be used to provoke a diuresis. Hæmodialysis may be necessary in renal failure.

(ii) *Simple pyrexial reactions* in which the patient develops pyrexia, rigor and some increase in pulse rate. These are the result of 'pyrogens' in the donor apparatus and are largely avoided by the use of plastic disposable giving sets.

(iii) *Allergic reactions* in which the patient develops mild tachycardia and an urticarial rash. This is the result of allergic reaction to plasma products in the donor blood and is most common in those individuals receiving repeated transfusions. The reaction is treated by slowing the transfusion and giving an appropriate antihistamine drug.

(iv) *Antibody production.*—This is not uncommon in those patients who have received many transfusions in the past, *e.g.* for hæmophilia or thalassæmia. The individual develops antibodies to donated white cells, platelets or to plasma proteins which cause reactions with each transfusion. They may be minimised by giving packed red cells from which plasma and 'buffy coat layers' have been removed or by 'washing' of donor cells. Antihistamines or steroids may also be given to the recipient if necessary.

(3) **Infections.**—There are two main reasons for blood transfusion causing infection in the recipient.

(i) *Serum hepatitis virus* may be transmitted from the donor and is usually a severe hepatitis arising approximately 3 months after the transfusion. It should be avoided by adequate verbal screening of the blood donor and by testing for the presence of the hepatitis associated antigen in the blood prior to transfusion.

(ii) *Bacterial infection* as the result of faulty storage. This arises most commonly from the donor blood being left in a warm room for some hours before the transfusion is commenced. This allows proliferation of any bacteria and transfusion of such infected blood may result in severe septicæmia in the recipient and rapid death.

(4) **Thrombophlebitis** (p. 175).

(5) **Air Embolism** (p. 156).

(6) **Disseminated Intravascular Coagulation.**—This may occur in patients receiving massive transfusions and may be contributed to by the underlying pathology.[1] There is an acute fall in the level of fibrinogen, platelets and some other clotting factors resulting in further hæmorrhage. It may be treated by replacement of the deficient factors such as fibrinogen and platelets, and heparin may indeed be advisable if the coagulation process is continuing.

Hæmophilia and the Congenital Hæmorrhagic Diseases

Hæmophilia (hæmophilia A) is a hæmorrhagic diathesis caused by the congenital deficiency in the blood of Factor VIII (antihæmophilic globulin AHG). It is a sex-linked characteristic, transmitted by the asymptomatic female carriers, and manifest only in males.

The levels of Factor VIII in the blood of severe hæmophiliacs may be as low as 0–3% of the average normal level. In the case of spontaneous hæmorrhage, e.g. into joints, treatment should aim at raising the level to at least 20%. Should surgery be anticipated in the hæmophiliac, the level should be raised to 50–100%.

Factor VIII therapy is best done by intravenous administration of cryoprecipitate, 5–10 units being given by simple injection. This should be done *immediately* prior to surgery since the half-life of Factor VIII is only 18 hours and the levels should be maintained daily after operation depending on the extent of the surgery.

Alternative forms of therapy include fresh blood, if necessary for blood loss, fresh frozen plasma, or, more rarely, dried concentrates of animal A.H.G.

Christmas Disease[2] (hæmophilia B) is a congenital disease resulting from the deficiency of Factor IX (Christmas factor). Clinically the manifestations of the disease

[1] Other acute causes of disseminated intravascular coagulation include: major trauma, burns, lung and prostate operations, cardiac arrest, pulmonary embolism, amniotic fluid embolism in ruptured placenta, acute liver failure. Disseminated malignant disease, and a retained dead fœtus are chronic causes.

[2] So-called after the name of the first patient in whom the disease was discovered.

are similar to hæmophilia, but treatment is less predictable. Factor IX is replaced by the transfusion of fresh-frozen plasma, or by reconstituted dried concentrates of human Factor IX.

Hæmophilic Joints.—In both hæmophilia and Christmas disease hæmorrhage into joints is very common, and persistent and recurrent hæmarthrosis may result in permanent damage to the articular surfaces and disorganisation of the joint. The most important feature of treatment is that it should be prompt and efficient. Replacement of the clotting factor should be instituted immediately and before severe tension is allowed to build up in the joint. Should this occur, however, aspiration may be advisable under cover of clotting factor replacement therapy.

Von Willebrand's Disease, with bizarre episodic bleeding manifestations is a type of Factor VIII hæmorrhagic disease, with low plasma levels of Factor VIII, and absence of Factor VIII related antigen, and platelet abnormalities.

Sickle cell anæmia can be a serious problem in surgery, especially children. With the associated sickle cell trait in mind it is essential to screen all patients of the negroid ethnic type in order to take steps to reduce the chance of sudden hæmolysis. These include, where possible, the preoperative transfusions of packed cells until the hæmoglobin-S level is reduced to less than 30 per cent, and transfusions during and after operation. Oxygen is needed as well as the prevention of hypoxia, hypothermia, and dehydration. Spinal anæsthesia and tourniquets are contraindicated. Bilirubin gallstone formation and splenomegaly may become additional burdens on these patients.

BLOOD SUBSTITUTES—PLASMA, DEXTRAN

One of the most urgent requirements in a patient suffering from acute blood loss is the re-establishment of a normal blood volume. This may be achieved satisfactorily with a number of plasma substitutes.

Plasma protein fraction (P.P.F.) has now largely superseded the use of dried plasma and can be used without delay since cross-matching is unnecessary. There is no risk of transmitting hepatitis and excellent results may be obtained in severely shocked patients by giving two to three units (1·4 l) of P.P.F. intravenously in half an hour. It is particularly valuable in patients with burns where there has been severe loss of protein.

Dextrans are polysaccharide polymers of varying molecular weight producing an osmotic pressure similar to that of plasma.

The high molecular weight dextrans (110,000 and 70,000) (Dextran 110 and Dextran 70) are less effective in the early phase of hypovolæmia but are longer-acting since they are retained for some time within the circulation. Dextrans have the disadvantage of inducing rouleaux of the red cells which may lead to problems in blood grouping and cross-matching procedures.

Low molecular weight dextran (40,000) (Dextran 40, Rheomacrodex) has a more immediate effect in restoring plasma volume but the smaller molecules are more readily excreted by the kidney and the effect is thus less prolonged. It may be of advantage in preventing sludging or stasis of red cells in the smaller blood vessels, *e.g.* of the kidney, and thus preventing the ' renal shutdown ' sometimes associated with severe hypotension. It is less likely to induce rouleaux formation than the high molecular weight compounds.

All the dextrans may be associated with abnormal bleeding and this appears to be more marked the higher the molecular weight, presumably because of the longer period of retention in the circulation. The cause for this increase in hæmorrhagic tendency is not known, but is not uncommonly seen

E. A. Von Willebrand, 1870–1949. Finnish Physician who described the clinical features in 1926.

when the blood volume has been inadvertently overexpanded. For this reason it is recommended that the total volume of Dextran given should not exceed 1,000–1,500 ml.

Mannitol, a sugar of low molecular weight, not a substitute for blood, is used to prevent acute renal failure following renal arteriolar constriction in shock, and in the crush syndrome (p. 86). It reduces the mortality from renal failure after operations for obstructive jaundice (Dawson). It promotes an osmotic diuresis. Dose.—Up to 1 g/kg body weight i.v. in 12 hr as a 20% solution, e.g. three infusions of 100 ml during and after operation. Test doses of mannitol may be used in cases of diminished urine output to determine whether hydration therapy or the strictly-limited-fluid regimen (Chap. 55) is to be adopted. Catheterisation (indwelling) is helpful to prevent retention due to rapid overfilling of the bladder and to provide means of accurate recording of the response to therapy.

SHOCK

Shock is a combination of a threat to existence and the response of the body to this threat. The common factors are:—

1. *A loss or withdrawal of blood volume* from the main vascular channels of the body, affecting cardiac output, central blood pressure and the perfusion pressure of oxygen into the tissues, particularly the heart and brain.

2. *Vasoconstriction* (Nature's first aid for 1.), which serves to maintain the peripheral vascular resistance, the arterial pressure, and therefore the oxygen perfusion of the vital structures. This is a protective mechanism which plays for time—time for the threat to cease (e.g. arrest of hæmorrhage and blood transfusion), so that full recovery is possible (reversible shock). If the threat persists, is repeated, or replacement of blood volume is not carried out, the anoxia to heart muscle and brain stem causes failure of these organs, thus impairing oxygen perfusion of tissues still further and causing a vicious circle (irreversible shock) ending in death (p. 70). Contributory causes are exposure, fatigue and starvation, and the extremes of life.

Clinically the condition of (1) loss of blood volume and (2) vasoconstriction is manifest as pallor, empty veins, cold skin, sweating and increased pulse.

The Types of Shock

Vasovagal Shock. Neurogenic Shock. Psychogenic Shock.—Occurs immediately as a result of a sudden fright (e.g. bad news) or severe pain (e.g. a blow on the testes). The effect varies in intensity from a slight faint to sudden death. The expression ' I nearly died of fright ' is not necessarily hyperbolical. The hypoxia of the vital centres is brought about by pooling of blood in the large vascular reservoirs (limb muscles), and by dilatation of the splanchnic arteriolar bed, with consequential low cardiac output and fall of arterial pressure until restored by peripheral vasoconstriction. A similar phenomenon of pooling and fainting occurs in guardsmen or other service personnel standing to attention for long periods.

Hæmorrhagic Shock.—See hæmorrhage, p. 70.

Burns Shock (p. 126).—The loss of blood volume is due to rapid plasma loss from the damaged tissues and whole blood destruction. A delayed form

John Leonard Dawson, Contemporary. Surgeon, King's College Hospital, London.

of burns shock may be caused by a breakdown product of cell membranes as well as from infection.

Dehydration, Diarrhœa, and Vomiting.—Severe extravascular fluid loss is usually associated with infection (exotoxins and endotoxins) and loss of protein and electrolytes as a result of inflammation of the intestinal mucous membrane (e.g. cholera, staphylococcal enterocolitis (p. 21), etc.).

Pleural, Peritoneal, Mediastinal, Retroperitoneal Shock.—Sudden leakage of blood, or alimentary secretions into these compartments will cause sudden collapse. Examples are penetrating chest wounds and hæmothorax, a leaking œsophageal or intestinal anastomosis, acute pancreatitis, biliary peritonitis.

Endotoxin Shock (syn. Gram Negative Shock, E. Coli Shock).—Multiplication of bacillus coli occurs in segments of strangulated intestine (strangulated hernia, mesenteric thrombosis), and in peritonitis. The endotoxin from the bacilli causes splanchnic arteriolar dilatation (splanchnic pooling) and blood volume discrepancy. Release or removal of the strangulated segment, or the drainage of pus is the important practical factor in recovery of the patient.

Bacteræmic Shock from other Infections.—Gas gangrene is a typical example, causing decreased blood volume and also anæmia from hæmolysis.

Anaphylactic shock, histamine shock, may follow injection of serum (e.g. A.T.S.), in spite of precautions, into patients who have had serum before, or who have a history of allergy. There is wide dilatation of the splanchnic vessels (splanchnic pooling) with consequential fall of venous return and cardiac output (Chap. 41).

Diagnostic Difficulties in Shock.—Because of a thready and irregular pulse, the differential diagnosis of cardiac infarction and even pulmonary embolism is notoriously confusing. In both cases there is a shock-like state from a degree of failure of cardiac output and compensatory vasoconstriction. An E.C.G. and an X-ray of the chest do not always determine the diagnosis. The measurement of the central venous pressure (C.V.P.) (p. 73) and its response to a small rapid transfusion may be of assistance. With intra-abdominal catastrophes the surgeon cannot escape the responsibility of performing an exploratory operation for suspected peritonitis, for after adequate operative treatment, intravenous fluids hitherto ineffective have their desired effect and the patient begins to recover.

Treatment of Shock

Treatment, in principle, is the removal of the cause and the replacement of lost blood-volume. In hæmorrhage this implies the arrest of hæmorrhage and blood transfusion. In burns the loss of plasma is replaced by plasma, plasma protein fraction, dextran and blood.

Endotoxin and bacteræmic shock require equally vigorous 'aggressive' treatment. This means immediate and adequate transfusion of blood or substitutes, with central venous pressure control (p. 73), full doses of the currently favoured broad spectrum antibiotic (e.g. ampicillin, the cephalosporins and gentamycin) and the appropriate treatment of the causal lesion.

Hydrocortisone 200 mg i.v. is usually given immediately and the dose, or

Hans Christian Joachim Gram, 1853–1938 (*see p. 19*).

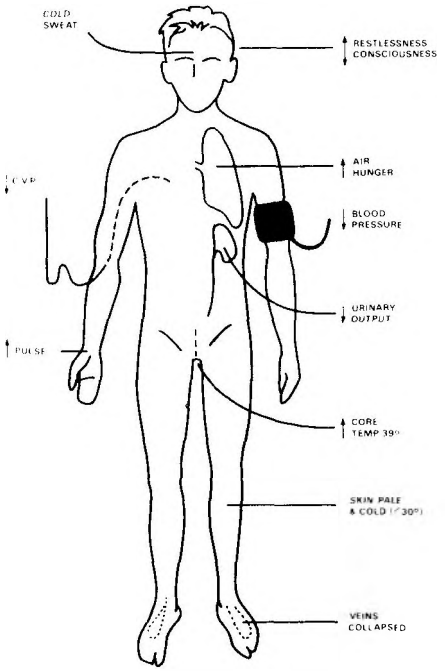


FIG. 74.—Clinical features of shock.
(*W. F. Walker, F.R.C.S., Dundee.*)

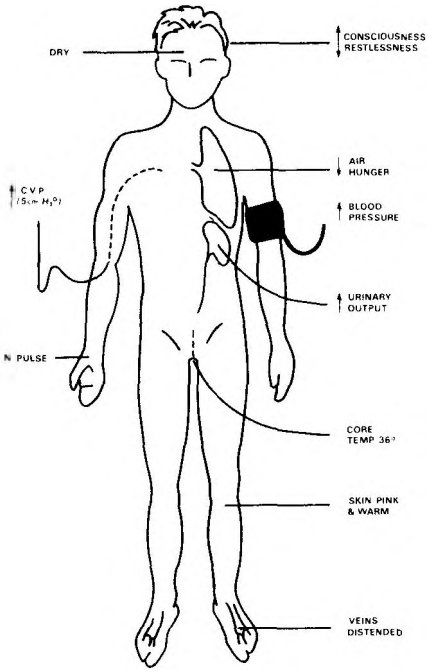


FIG. 75.—Response of patient to adequate volume replacement.
(*W. F. Walker, F.R.C.S., Dundee.*)

100 mg, repeated. A good example of this clinical situation is gas gangrene (p. 33).

Position and Limb Compression.—Venous return is improved, and pooling of blood in the large muscles of the legs is reduced by raising the foot of the bed and bandaging the limb so as to compress the peripheral blood-vessels.

Relief of pain is achieved by giving morphine 10–15 mg i.m. or i.v. It is not given subcutaneously as it is not easily absorbed, and if repeated doses are administered, an excessive amount is liable to be absorbed when the circulation improves. It is not to be given in head injuries.

Sedation for a frightened patient may be by diazepam B.P. 5–15 mg i.v.

Vasoconstrictor Drugs.—These should be given early. Given late, their action, if any, will only serve to accelerate the damage caused to kidneys and liver by intense vasoconstriction. The substances used include intravenous hydrocortisone 100 mg, and methylamphetamine (Methedrine) 15 mg. Hydrocortisone raises the blood pressure possibly by sensitising the peripheral vascular nerve endings to naturally circulating noradrenaline.

Vasodilator Drugs.—Excessive adrenergic activity with high levels of circulating catecholamines (adrenaline and noradrenaline) can sometimes be a factor producing delay in recovery. *a*-blocking agents and chlorpromazine may be used *with extreme caution.* The sudden production of vasodilation in a vasoconstricted and dehydrated patient can be followed by a catastrophic fall in arterial blood pressure, unless *vigorous intravenous infusion*, with C.V.P. monitoring (p. 73), accompanies the use of these agents, which should be given in small repeated doses only until extremities become warm and pink and the veins dilated and well filled.

It must be emphasised that the best chance of success in *clinical situations* would

therefore appear to be to use drugs *early* rather than late, i.e. as soon as it becomes evident that conventional treatment is not resulting in any clinical improvement.

OXYGEN THERAPY

Indications in Surgery.—In shock its value has been disputed, but it is of special value in chest injuries, in severe hæmorrhage, in toxic hæmolysis, e.g. gas gangrene, in coal gas poisoning and in over-morphinisation. It may also be used in pulmonary embolism, fat embolism, spontaneous pneumothorax, acute pulmonary œdema, and cardiac infarction, and in cases of acute bronchitis, pneumonia, and anoxic cor pulmonale. The usual method employs a disposable polythene mask, delivering 4–6 litres per minute. For patients with chronic bronchitis and emphysema there is a risk of a high concentration of oxygen reducing the carbon dioxide drive for respiration, so causing carbon dioxide narcosis. The safe concentration (Campbell) is about 27 per cent oxygen delivered by a Venturi or disposable Ventimask in which a jet of oxygen at 4 litres per minute strikes the side walls of the air intake aperture and sucks in about 50 litres of air per minute thus giving the necessary mixture.

Oxygen tents may also be used if a high concentration of oxygen is required. Children should be reassured, as they are likely to be frightened by confinement in an enclosed space, and, if possible, nervous patients should be accustomed to a tent before the necessity arises. Nurses are warned against the risk of explosion, and naked lights and smoking are forbidden in the proximity of the tent.

Hyperbaric Oxygen. Oxygen Drenching.—Oxygen, when it is administered at 1 or 2 atmospheres above atmospheric pressure in a compression chamber (fig. 76) will at least double the arterial plasma oxygen saturation and so the oxygen perfusion of tissues can be increased. When such a chamber is available it can supplant the use of carbogen (oxygen with 5 or 7 per cent carbon dioxide) in the treatment of carbon monoxide poisoning, and may be used for the treatment of anaerobic infections, e.g.

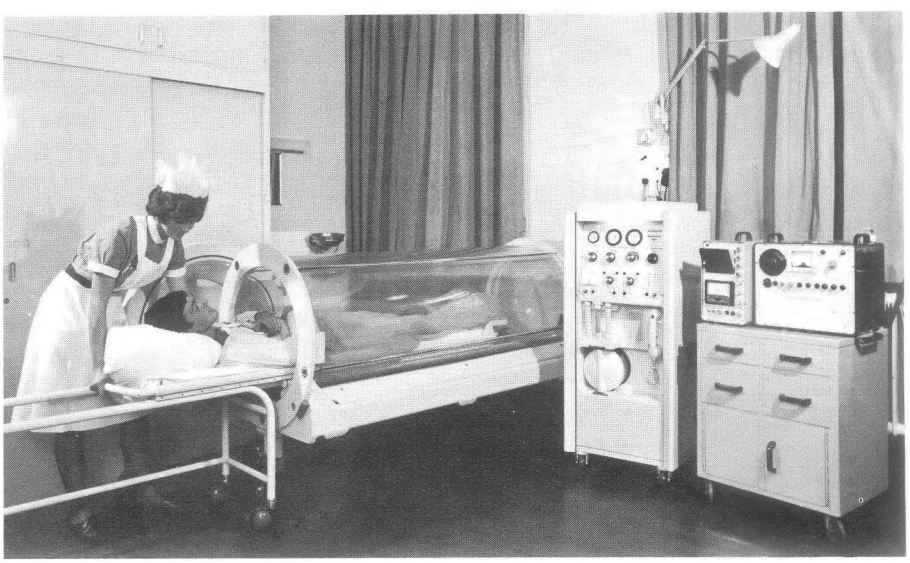


FIG. 76.—Hyperbaric oxygen chamber.
(*Vickers Ltd.*)

Edward James Moran Campbell, Contemporary. Professor of Medicine, McMaster University, Hamilton, Ontario, Canada.

tetanus and gas gangrene, bedsores and frostbite. Oxygen is also a radio-sensitiser in the treatment of cancer. Drenching has been used to treat paralytic ileus and meteorism, for since the gas in the distended bowel is nitrogen, breathing pure oxygen so reduces the partial pressure of nitrogen in the lungs and plasma that reabsorption from the bowel takes place.

Topical Oxygen Therapy.—Hydrogen peroxide 5 or 10 volumes, and 2 per cent zinc peroxide, giving off nascent oxygen are used locally on wounds infected with anaerobic or microaerophilic organisms, e.g. spreading symbiotic gangrene of the abdominal wall (Chap. 53). It is also used to free the sloughs of ulcers.

CRUSH SYNDROME

This syndrome is commonly associated with earthquakes, air-raids and with mining and other accidents. As a result of massive crushing of muscles, oligæmic shock occurs, due mainly to extravasation of blood into adjacent muscles. Myohæmoglobin enters the circulation and acute renal tubular necrosis is likely to result. The degree of shock has no relation to the development of the syndrome. A similar effect may follow the application of a tourniquet for too long a period (p. 74)!

First-aid treatment may necessitate the application of a tourniquet to the affected limb, which is gradually released so that deleterious substances are admitted to the circulation in small quantities. The crushed muscle often swells considerably, and being confined within unyielding deep fascia, tension develops, impeding the circulation and increasing the extent of ischæmic damage. The limb feels tense and pain is severe. Tension should be relieved by parallel incisions, through which the swollen muscle protrudes. The patient may appear to be comparatively well for two or three days following the accident, but excretion of urine is scanty. Apathy, restlessness, and possibly mild delirium indicate deficient renal function, and uræmia supervenes. If oliguria develops, the fluid intake must be restricted to 'x' plus 400 ml, 'x' being the urinary output for the previous twenty-four hours. The urine should be rendered alkaline by the administration of sodium citrate and sodium bicarbonate. *Rheomacrodex* (p. 81), is useful, by clearing blood sludging and myohæmoglobin, or *mannitol* (p. 82). Dialysis may be life-saving.

CHAPTER 8

FLUID, ELECTROLYTE AND ACID-BASE BALANCE NUTRITION

DAILY WATER BALANCE OF A HEALTHY ADULT (70 kg)

Intake		*Output*	
Water from beverage	1,200 ml	1,500 ml	Urine
Water from 'solid food'	1,000 ml	900 ml	Insensible loss from skin and lungs
Water from oxidation	300 ml	100 ml	Fæces

The Intake is derived from two sources (*a*) exogenous, and (*b*) endogenous.

Exogenous water is either drunk or ingested in solid food. The quantities vary within wide limits, but average 2 to 3 litres per twenty-four hours, of which nearly half is contained in solid food.

Taking into consideration their body-weight, the water requirements of infants and children are relatively greater than those of adults because of (*a*) the larger surface area per unit of body-weight; (*b*) the greater metabolic activity due to growth; (*c*) the comparatively poor concentrating ability of the immature kidneys of the neonate.

Endogenous water is released during the oxidation of ingested food and the amount is less than 500 ml per twenty-four hours. During starvation this amount is supplemented by water released from the breakdown of body tissues.

The Output.—Water is lost from the body by four routes:

1. *By the Lungs.*—About 400 ml of water is lost in expired air each twenty-four hours. In a dry atmosphere, and when the respiratory rate is increased, the loss is correspondingly greater.

2. *By the Skin.*—When the body becomes overheated there is visible perspiration, but throughout life invisible perspiration is always proceeding. The cutaneous fluid loss varies within wide limits in accordance with the atmospheric temperature and humidity, muscular activity, and body temperature. In a temperate climate the average loss is between 600 and 1,000 ml per twenty-four hours.

3. *Fæces.*—Between 60 and 150 ml of water are lost by this route daily. In diarrhœa this amount is multiplied not only by the number of stools, but according to their fluidity and size.

4. *Urine.*—The output of urine is under the control of a secretion of the posterior lobe of the pituitary gland (the anti-diuretic hormone) which con-

trols the tonicity of the body fluids, a function that it performs by stimulating the reabsorption of water from the renal tubules, thus varying the amount excreted after the requirements of the first three routes have been met. The normal urinary output is 1,500 ml per twenty-four hours, and provided the kidneys are healthy, the specific gravity of the urine bears a direct relationship to the volume. A minimum urinary output of 400 ml per 24 hours is required to excrete the end products of protein metabolism.

Water Depletion.—*Pure water depletion*, apart from the concomitant electrolyte depletion which follows diarrhœa or vomiting, is usually due to a diminished intake. The causes include: (*a*) weakness, exhaustion, mental changes, and cerebrovascular states, (*b*) restricted intake after gastro-intestinal operations, or (*c*) inability to swallow as in painful conditions of the mouth or throat or in œsophageal obstruction. The main symptom is intense thirst and the leading sign is oliguria. As the dehydration progresses, these features (and weakness) increase, but the patient still remains capable of physical and mental exertion. Pure water depletion causes intracellular dehydration and is not accompanied by peripheral circulatory failure. Pure water depletion may also follow the increased loss from the lungs after tracheostomy and may amount to as much as 500 ml in twenty-four hours in excess of the normal insensible loss. After tracheostomy humidification of the inspired air is an important preventive measure.

Treatment.—The intake of water must be increased. If swallowing is possible, much depends upon the nursing staff ensuring a regular half hourly or hourly intake. Intravenous 5 per cent dextrose or dextrose saline (p. 94) is given if swallowing is impossible (water is not isotonic and would hæmolyse blood). Careful charting of intake and output is essential. A diuresis signals that enough has been given, and thereafter care must be taken not to overload and cause water intoxication.

Thirst.—The seat of the osmoreceptors (drinking centre) is in the supra-optic nucleus of the anterior hypothalamus (E. B. Verney). Thirst is experienced in acute hypovolæmic shock and after major surgical operations. This is due to intracellular dehydration and corresponds to the period of rapid loss of potassium from the cells of the body. If necessary, the urge to satisfy thirst can be temporarily assuaged by sham drinking—contact of water with the pharynx—by washing out the mouth with ice-cold water.

Water Intoxication, which can occur without sodium loss, especially if the excessive intake of water is rapid, is more likely to supervene when impaired renal function leads to slowing of diuresis. There is always a danger of this happening when there is (1) failure to keep accurate intake and output fluid-balance charts, (2) failure to add up the twelve- or twenty-four-hourly totals on the charts (fig. 83), as well as (3) failure to be aware that water intoxication can actually occur. It is particularly likely (*a*) when isotonic (5 per cent) dextrose solution is being administered intravenously, (*b*) when fluid therapy is being undertaken by the administration of tap water per rectum and (*c*) following recto-colonic washouts of plain water or sodium-free solutions, particularly in children with Hirschsprung's disease (Chap. 49).

Ernest Basil Verney, 1894–1967. Shield Professor of Pharmacology, Cambridge, England.

Other causes of water intoxication may need to be considered. These include lung conditions such as lobar pneumonia and empyema, ADH-secreting tumours such as oat-cell carcinoma of the bronchus, and hypopituitary states.

Clinical Features.—These include drowsiness, weakness, sometimes convulsions and coma. The patient appears to be in shock—which is incorrect, because the blood-pressure is not low and the pulse is not unduly rapid until the patient is moribund. Nausea and vomiting of clear fluid are common, and usually the patient passes a considerable amount of dilute urine.

Treatment.—The intake of water having been stopped, the best course is masterly inactivity. If after several days the water-logged patient is still stuporous (due, no doubt, to renal inactivity), it is permissible to infuse *very slowly* not more than 200 ml of hypertonic (5·85 per cent) saline solution intravenously. If this brings about improvement and a diuresis begins the infusion should be stopped forthwith, otherwise circulatory failure from overloading (due to the sudden shift of fluid from cells and tissue spaces) or cardiac arrest is liable to ensue.

ELECTROLYTE BALANCE

When inorganic salts are in solution, as in the extracellular or intracellular fluids of the body, they dissociate into ions. Ions are of two kinds (*a*) *Cations*, which are electropositive and (*b*) *Anions*, which are electronegative: collectively these are the electrolytes. The most accurate way of describing the chemical concentration, reactivity, and osmotic power of these ions is in terms of their milli-equivalents per litre (mEq/l) or in S.I. units as milli-moles per litre (mmol/l), see p. 93. The cations include sodium, potassium, calcium, and magnesium ; the anions chloride, phosphate, bicarbonate, and sulphate. The distribution of the salts within the fluid compartments of the body controls the passage of water through the cell walls and maintains acid-base equilibrium.

In S.I. units, mEq/l is replaced by mmol/l. Thus comparing the total of anions with the total of cations is no longer possible. However for monovalent ions (sodium, potassium, chloride and bicarbonate) 1 mEq = 1 mmol, so numerically at least the additions are possible.

Sodium Balance

Sodium is the principal cation content of the extracellular fluid. The total body sodium amounts to approximately 5000 mEq (5000 mmol), of which 44 per cent is in the extracellular fluid, 9 per cent in the intracellular fluid, the remaining 47 per cent being in bone. The sodium housed in bone merits special notice: a little more than half of it is osmotically inactive, and requires acid for its solution; the remainder is water-soluble and exchangeable. Thus there is a large storehouse of sodium ready to compensate abnormal loss from the body. The daily intake of sodium is inconstant. On an average it is 80 to 100 mEq (80–100 mmol) (5 to 6 g) sodium chloride or 570 ml of isotonic 0·9 per cent saline solution. An equivalent amount is excreted daily, mainly in the urine, also a little in the fæces. The loss in perspiration normally is negligible; however, in persons not acclimatised to tropical heat prolonged profuse sweating results in a considerable loss of sodium—as much as 85 mEq (85 mmol) per hour (J. Nash). If water alone is given to counterbalance the fluid loss, serious sodium depletion can occur from excessive sweating. (See also mucoviscidosis, Chap. 47.)

Michael Faraday, 1791–1867. Pioneer of electro-magnetism, introduced the words 'electrolyte', 'cation', 'anion'.
Joseph Nash, 1902–1945. Assistant Professor of Surgery, New York College of Medicine, New York, U.S.A. Died in Germany on active service while Surgical Chief of the Ninetieth General Hospital, U.S. Army.

Control by Adrenal Corticoids.—The output of sodium, governed by the variation in the avidity with which the renal tubules reabsorb sodium from the glomerular filtrate and the amount of sodium excreted by the sweat glands, is under the control of the adrenal corticoids, the most powerful conservator of sodium being *aldosterone*. When the adrenal glands have been destroyed by disease or extirpated, there is an unbridled loss of sodium in the urine.

The Sodium Excretion Shut-down of Trauma.—Following trauma of any kind (including operation trauma) there is a spell, the length of which varies directly with the degree of tissue damage, of almost non-excretion of sodium. During this period the output of sodium, reduced to not more than 10 mEq (10 mmol) *per diem*, cannot be increased by a supplemented intake of sodium. For this reason it is most inadvisable to administer large quantities of isotonic (0·9 per cent) saline solution soon after an operation. The period of sodium shut-down lasts for at least forty-eight hours and is due to increased adrenocortical activity.

Sodium Depletion (*syn.* **Hyponatræmia**).—The most frequent cause of sodium depletion seen in surgical practice is obstruction of the small intestine, with its rapid loss of gastric, biliary, pancreatic, and intestinal secretions by antiperistalsis and ejection, whether by vomiting or aspiration. Duodenal, total biliary, pancreatic, and high intestinal external fistulæ also are all notorious for bringing about early and profound hyponatræmia. Hyponatræmia is also seen in the syndrome of inappropriate secretion of antidiuretic hormone as in bronchial carcinoma.

There is one other less obvious, nay surreptitious, means whereby the patient is robbed of sodium, and that is by gastric aspiration combined with allowing the patient to drink as he pleases and promptly aspirating the fluid swallowed. The act of drinking excites the flow of gastric juice, and this is also aspirated. During this form of therapy, should the patient be receiving intravenous dextrose solution, as opposed to dextrose-saline solution, to maintain fluid balance, he will soon become a victim of hyponatræmia.

Clinical features of hyponatræmia with salt and water depletion are due to extracellular dehydration. In established cases the eyes are sunken and the face is drawn. In infants the anterior fontanelle is depressed. The tongue is coated and dry; in advanced cases it is brown in colour. Unlike the dehydration produced by loss of water only, in water + salt depletion thirst is not particularly in evidence. The skin is dry and often wrinkled, making the patient look older than his years. The subcutaneous tissue feels lax. Peripheral veins are contracted and contain dark blood. The arterial blood-pressure is likely to be below normal. The urine is scanty, dark in colour, of a high specific gravity, and except in cases of salt-losing nephritis contains little or no chloride.

Presuming that the hæmoglobin level before the dehydration commenced was normal, the hæmatocrit reading (P.C.V.) provides an index of the degree of hæmoconcentration. On the other hand, hæmoconcentration can be masked by pre-existing anæmia (p. 72).

Treatment.—The sodium deficit is replaced by an intravenous infusion of 0·5 to 2 litres of isotonic saline or Ringer's solution (p. 95), depending on the severity of the hyponatræmia. It is important to watch the plasma protein levels in order to avoid overdosage, thus causing hypoproteinæmia and œdema. In severe cases the first step is to restore the circulating blood volume by a rapid infusion of plasma or plasma substitute (p. 81).

Post-operative Hyponatræmia.—Hyponatræmia with a normal or increased extracellular fluid volume arises as a result of too prolonged administration of a sodium-free solution (5 per cent dextrose) intravenously. Symptoms seldom present until such treatment has been in progress for more than forty-eight hours (cf. Water Intoxication, p. 88). Early symptoms are headache and giddiness. Hyponatræmia causes pylorospasm and the resulting vomiting adds to the sodium deficit. Should renal function be impaired there will be an associated low level of urinary chlorides.
Treatment.—See water intoxication, p. 88.

Sodium excess (**syn. Hypernatræmia**) is likely to arise if a patient is given an excessive amount of 0·9 per cent saline solution intravenously during the early post-operative period when, as has been described, some degree of sodium retention is to be expected. The result is an overloading of the circulation with salt and its accompanying water. Even a sub-clinical degree of sodium excess is harmful, for it results in œdema of intestinal and other suture lines. In addition there is danger of pulmonary œdema.

Clinical Features.—Slight puffiness of the face is the only early sign. The patient himself makes no complaint. Pitting œdema should be sought, especially in the sacral region, but for pitting œdema to be present at least 4·5 litres excess fluid must have accumulated in the tissue spaces (Marriott). The patient's weight increases *pari passu* with the water-logging, and the aid of bed-scales should be sought. Signs of overhydration in infancy (infants are very susceptible) are increased tension in the anterior fontanelle, increased weight, increase in the number of urinations, and œdema.

Treatment.—The infusion must be stopped. In established cases with distended jugular veins and pulmonary œdema, treatment should follow the lines suggested in Chap. 40.

Potassium Balance

Potassium is almost entirely intracellular. No less than 98 per cent is intracellular, and only 2 per cent is present in the extracellular fluid. Three-quarters of the total body potassium (approximately 3 500 mEq (3 500 mmol)) is found in skeletal muscles. When the body needs endogenous protein as a source of energy, potassium, as well as nitrogen, is mobilised. The mobilised potassium passes to the extracellular fluid, but the surplus over and above the normal content is so rapidly excreted by healthy kidneys that the concentration of potassium in the plasma remains unaltered. Each day a normal adult ingests 2 to 3 g (52 to 78 mEq (52–78 mmol)) of potassium in food; fruit, and milk and honey, are rich in this cation. Except for a very small quantity in formed fæces and a still smaller quantity in sweat, an amount corresponding to the intake is excreted in the urine.

Hugh Leslie Marriott, Contemporary. Emeritus Physician, Middlesex Hospital, London.

Potassium Depletion

The Augmented Potassium Excretion of Trauma.—Following trauma, including operation trauma, there is a spell, varying directly with the degree of tissue damage, of increased excretion of potassium by the kidneys. This loss is greatest during the first twenty-four hours and lasts, for example in the case of partial gastrectomy, about three or four days. It will be noticed that the behaviour of the body's potassium in the post-trauma phase is the exact opposite to that of sodium—the potassium flees; the sodium stays. So great are the body's reserves of potassium that, unless the patient was severely depleted at the time of the operation, the therapeutic administration of potassium (always a responsible undertaking) does not arise until the fourth day of intravenous alimentation.

Hypokalæmia can occur suddenly or gradually.

Sudden hypokalæmia is unlikely to be encountered in surgical practice. It occurs most frequently in diabetic coma treated by insulin and prolonged infusion of saline solution.

Gradual hypokalæmia is the type met with in surgical practice. The diarrhœa from ulcerative colitis, villous tumours of the rectum (Chap. 52) and the loss from external fistulæ of the alimentary tract are common causes (e.g. duodenal fistula, ileostomy); the potassium content of the discharge from some of these fistulæ is twice that of the plasma potassium concentration. Another frequent cause of hypokalæmia is prolonged gastroduodenal aspiration with fluid replacement by intravenous isotonic saline solution. It is also prone to occur in the post-operative period following extensive resections for carcinoma of the alimentary tract, because often the operation has to be undertaken after months of weight-loss and potassium depletion.

Clinical Features.—The patient lies listlessly in bed. Speech is slurred and slow; often the patient fails to complete a sentence. Intense drowsiness soon follows and all the patient desires is to be left in tranquillity to sleep. Muscular hypotonia is the outstanding physical sign. Reflexes are lost and incontinence of urine is common. Abdominal distension amounting to paralytic ileus is a constant accompaniment, and in all cases of paralytic ileus the plasma-potassium value should be investigated.

Weakness of the respiratory muscles frequently occurs and results in rapid, shallow, gasping respirations; these are conducive to post-operative pulmonary complications. The diastolic blood-pressure is low, but there is usually a bounding pulse and a presystolic murmur. The diagnosis should be confirmed by electrocardiography, which may show a prolonged QT interval, depression of the ST segment, and a lowering or inversion of the T wave (fig. 77).

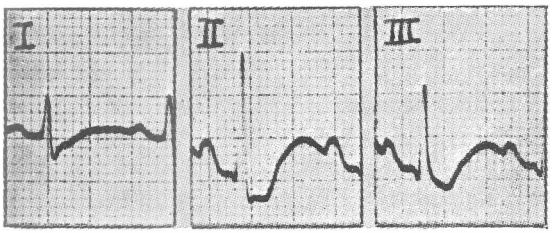


FIG. 77.—Electrocardiographic changes in severe hypokalæmia. The plasma potassium estimation was 1·6 mEq/l (1·6 mmol/l). (*W. F. Walker, F.R.C.S., Dundee.*)

Treatment

Oral Potassium.—Potassium is usually given in the form of milk, meat

extracts, fruit juices and honey. Potassium chloride, 2 g effervescent tablets, is given by mouth six-hourly.

Intravenous Potassium.—Should ingestion be inadequate or contraindicated, this route must be used. It is not without the danger of giving too much too quickly (especially when renal function is impaired), thus raising the plasma concentration to a dangerous level and causing cardiac arrest. Administration should be properly controlled; the plasma level of potassium should be checked daily; the urine output must be adequate (500 ml per twenty-four hours—20 ml per hour). *When there is no associated alkalosis*, the potassium deficit can be restored by adding 20 mEq or mmol potassium chloride to a litre of 5 per cent dextrose or 0·9 per cent normal saline solution, which is given over eight hours.

Hypokalæmic alkalosis, see p. 101.

Estimation of Electrolyte Balance.—For full electrolyte determinations it is best to take sufficient blood from a vein to fill to the brim a small tube (6–7 ml) into which a small quantity of standard lithium heparin solution has been introduced and dried. Replace the stopper, mix by repeated inversion and dispatch to the laboratory immediately. Every effort must be made to avoid hæmolysis. The plasma separated from hæmolysed blood is useless for potassium estimations.

The Plasma-Sodium Level.—Sodium, with its equivalent anions, accounts for about 90 per cent of the osmotic pressure of the plasma. Changes in the sodium content coincide with changes in the osmolality of the plasma, and so of all the body fluids. The best index of the total electrolyte concentration of the plasma is the sodium value, which normally is between 137 and 147 mEq (137 and 147 mmol) per litre. Whenever possible the *plasma chloride* and *bicarbonate* should be estimated simultaneously, since variations in the one may be accompanied by opposite changes in the other. The normal level of chloride is 95 to 105 mEq/l (mmol/l), and of bicarbonate 25 to 30 mEq/l (mmol/l); the sum of the two remains roughly constant at 120 to 135 mEq per litre.

Potassium deficiency is present if the plasma-potassium value is less than 3·5 mEq/l (mmol/l). The normal range is 3·5 to 5·0 mEq/l (mmol/l). It must be remembered that intracellular potassium deficiency may be present although the plasma concentration is normal, and that deficiency is to be expected if oral feeding has been withheld for more than four days. Estimation of potassium in the urine or aspirated gastro-intestinal contents serve as a guide to the rate of depletion and the replacement necessary (W. J. Griffiths). Electrocardiography may also be used (fig. 77).

Calcium

Calcium is an extracellular cation with a plasma concentration of 4·5–5·5 mEq/l (2·2–2·5 mmol/l). It exists in three forms: bound to protein, free non-ionised and free ionised, the last form being the component necessary for blood coagulation and affecting neuromuscular excitability. The ionised proportion falls with increasing pH; thus in respiratory alkalosis due to hyperventilation there may be tetany—with an apparently normal total plasma calcium level. In the urine the ionisation and the solubility of calcium is similarly depressed if the pH is elevated, thus promoting stone formation. The plasma level of calcium is likely to be modified by any factor promoting or inhibiting its absorption from the bowel, its storage in bone or its elimination by the kidneys: such factors include vitamin D and phytic acid, parathormone and calcitonin (Chap. 36), and the state of renal and small bowel function.

The management of abnormal calcium blood levels depends, where possible, on removal of the cause, e.g. removal of a parathyroid tumour (Chap. 36). In hypocalcæmia with tetany (Chap. 36) or in the coagulation disorder due to massive transfusion of blood containing acid citrate dextrose (ACD, p. 76), 10 ml of 10% calcium gluconate may be injected slowly intravenously. If oral administration is possible, calcium aspirin is useful (Chap. 36). On a long-term basis the diet should be adjusted to provide a high calcium and a low phosphate intake.

William James Griffiths, Contemporary. Formerly Chemical Pathologist, St. Thomas's Hospital, London.

Magnesium

Magnesium is an intracellular cation which shares some of the properties of potassium and some of calcium. The normal magnesium concentration is 1·8 mEq to 2·2 mEq (0·7–0·9 mmol) per litre. The average daily intake is approximately 20 mEq (10 mmol). Magnesium deficiency may occur when there is prolonged loss of gastrointestinal secretions due to fistulæ or ulcerative colitis, very prolonged administration of intravenous fluids without magnesium supplements, following massive small bowel resections, and in some cases of cirrhosis of the liver or disease of the parathyroids. The clinical picture of magnesium deficiency is marked by central nervous system irritability, E.C.G. changes, lowered blood pressure and lowered protein synthesis. The best method of diagnosing magnesium deficiency is the measurement of urinary magnesium after an intravenous loading dose. Normally 90% of the loading dose is promptly excreted in the urine.

Treatment.—In severe depletion 80 mEq (40 mmol) as magnesium sulphate can be given in 5% dextrose or normal saline over a 24-hour period. Magnesium supplements are essential in hyperalimentation.

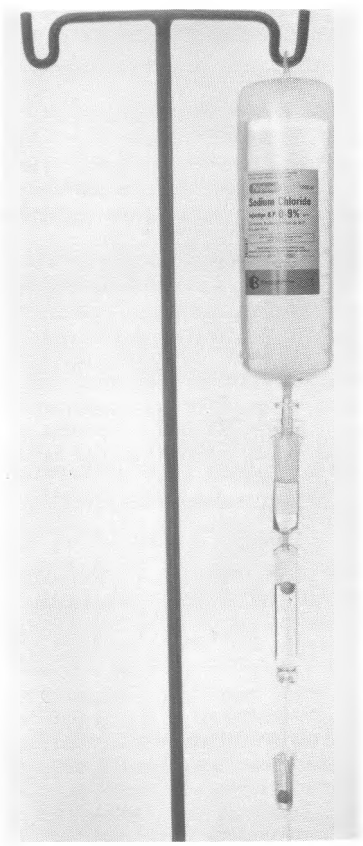


FIG. 78.—Plastic bottles are useful for intravenous therapy. The quantity administered may be read from the calibrations on the bottle. Note the double drip chamber with plastic base which prevents air embolus by dropping down and occluding the tubing should the bottle empty unnoticed. The drip rate can be controlled by the clip seen at the bottom of the picture. The infusion can be pressurised by squeezing the container.

PARENTERAL FLUID THERAPY

To overload the circulation is a grievous fault, and grievously does the patient pay for it.

The administration of fluid by any route other than the alimentary canal, i.e. intravenous, intramuscular, subcutaneous, or into the bone marrow, is known as parenteral administration (fig. 78).

The sterility of a solution for parenteral use must be assured. Furthermore, the solution must be free from dead bacteria and other particulate matter, as also must the delivery tube.

Solutions mainly in use:—1. **Plasma** (p. 81).

2. **Dextrose 5 per cent** is an isotonic solution that supplies calories without electrolytes. It is useful mainly in the very early post-operative period when sodium excretion is reduced (p. 90). It is also valuable when the salt requirements of a patient needing much fluid have been satisfied on a particular day. Prolonged administration of 5 per cent dextrose solution is liable to result in hyponatræmia, and may cause thrombosis of the vein used.

3. **Isotonic (0·9 per cent) saline solution** is needed in the following conditions: when a large amount of sodium has been lost by vomiting, or by gastric, duodenal, or intestinal aspiration or through

an alimentary fistula. Possibly, on occasions, excessive sweating may justify its use.

These two solutions meet all the requirements in 95 per cent of cases.

4. **Dextrose 4·3 per cent with Saline 0·18 per cent** (one fifth normal saline).—This solution is isotonic. Usually it is referred to as dextrose-saline. It must not be confused with 5 per cent Dextrose in saline which is hypertonic.

5. **Ringer's Lactate solution** contains sodium, potassium and chloride in almost the same concentrations as they are in the plasma. It also contains some calcium and some lactate. This solution can be used in hypovolæmic shock while awaiting blood. It is also suitable for replacing lost intestinal secretions.

6. **Darrow's solution** contains sufficient potassium to combat hypokalæmia and in surgical practice it is a safe and convenient method of supplying this cation provided alkalosis is not present (p. 100). The rate of infusion should not exceed 60 drops a minute.

COMPOSITION OF PLASMA AND INTRAVENOUS FLUIDS (mEq/l = mmol/l)

	Na	K	Cl	HCO_3	Lactate
Plasma	137–147	4–5·5	95–105	22–25	—
Isotonic[1] Saline	153	—	153	—	—
1/5 Isotonic Saline 4·3% Dextrose	30·6	—	30·6	—	—
Ringer Lactate	130	4	110	—	28
Darrow's	124	36	104	—	56

A Suggested Routine Post-operative Fluid Regime.—From the above information we know the average patient requires (1) 2 000/3 000 ml of water daily; (2) At least 100 mEq (mmol) of Na daily; (3) about 60 mEq (mmol) of K daily. These requirements are altered by the metabolic response to trauma. In the first 24 hours after surgery there is an increased secretion of anti-diuretic hormone and aldosterone. The patient will require no salt and less water than normal in this 24-hour period. Two litres of 5% dextrose is sufficient intravenous fluid when all abnormal operative loss has been replaced. During the second and subsequent 24-hour periods the metabolic response to trauma diminishes and the patient needs 2 litres of 5% dextrose and 1 litre of isotonic saline per 24 hours. On the 4th post-operative day and thereafter 20 mEq (mmol) of potassium is added to each litre to give a total of 60 mEq (mmol) per 24 hours.

An alternative regime is to give 3 litres of solution 4·3% dextrose 0·18% isotonic saline daily and add 20 mEq (mmol) of potassium chloride to each litre on the 4th post-operative day.

Continuous Intravenous Infusion (Venoclysis).—Because of the certainty with which the fluid enters the circulation without the necessity of being absorbed, this is by far the most usual method of parenteral administration. If a suitable vein can be rendered prominent by the application of a light tourniquet, it can be entered by a hollow needle (venepuncture) (fig. 79). The

[1] It is inadvisable to use the term 'normal' saline because chemically normal saline contains 5·85 g/100 ml.

Sydney Ringer, 1835–1910. Physician, University College Hospital, London.
Daniel Cady Darrow, 1895–1965. Professor of Pediatrics, Duke University School of Medicine, Durham, North Carolina, U.S.A.

FIG. 79.—Intravenous cannula (ABBOCATH)[1]. The cannula is made of teflon and is radiopaque.

limb is then immobilised by placing it on a light splint or, if a vein of the forearm is used, with a few strips of adhesive plaster.

Site.—As a routine measure, a vein on the forearm, or on the back of the hand is chosen (fig. 80). In women, young children, and even some men, especially when the venous pressure is low, a visible or palpable vein may not be found in the arm; consequently another site must be used. The external jugular vein, internal jugular vein, subclavian vein, or cephalic vein in the deltopectoral groove, can be selected. Because of the risk of thrombo-phlebitis and pulmonary embolism, a vein of the leg (e.g. the long saphenous vein in front of the medial malleolus) should not be selected if it can reasonably be avoided. This injunction does not hold good for infants and small children, who are singularly immune to thrombo-phlebitis.

FIG. 80.—Hand or forearm veins are used to give the patient maximum mobility.

Contraindications to Intravenous Therapy.

1. *The Failing Heart.*—A history of dyspnœa on exertion, uncompensated valvular disease, or any possibility of myocardial insufficiency should call for hesitation in increasing the bulk of circulating fluid.

2. *Pulmonary Congestion.*—Where there are signs of œdema of the bases of the lungs the method should be absolutely forbidden.

Clinical Guide to Fluid Requirements.—When a patient is admitted in a dehydrated condition it is of course impossible to measure the loss of fluid and electrolytes he has sustained, but a detailed history of the nature and quantity of the fluid lost (usually, in surgical practice, by vomiting), and particularly the appearance of the patient (p. 99), are the means by which the amount of electrolytic solution he should receive is estimated.

The hæmatocrit reading (p. 72) may be a help in this direction. If large quantities of fluid are necessary, the monitoring of central venous pressure and urinary output may help in determining the optimum rate and volume of

[1] Abbocath, Abbott. A manufacturer's instructions concerning the insertion and fixation of any plastic catheter must be followed carefully, as fracture of the tube and disappearance of part of it up a vein has occurred. If difficulty is encountered, both needle and catheter should be withdrawn at the same time.

the infusion (p. 73). In most instances the replacement is commenced by giving isotonic (0·9%) saline solution intravenously. When the bulk of the loss has been made up in this way, the *maintenance* of fluid balance is effected with dextrose-saline solution. In cases of extreme dehydration, where peripheral circulatory impairment (shock) is in evidence, it is necessary to commence by supplementing the volume of circulating fluid by dextran or plasma infusion (p. 81), and to follow this by substituting isotonic saline solution. When the fluid loss is considered to be rectified and fluid by mouth is contraindicated, it is again advisable to supply the maintenance fluid requirements in the form of dextrose-saline solution.

In infants with dehydration, the amount of fluid required will be approximately the same as the weight loss, i.e. the difference between the estimated and the actual weight (1 g equals 1 ml).

Rate of Flow.—Using a plastic transfusion set, 15 drops of fluid are approximately equal to 1 ml. At a rate of 30 drops per minute 1 litre will be given in eight hours, at 45 drops per minute it will take six hours, and at 60 drops some four hours. In severely dehydrated patients the rate of the first hour can be accelerated to well over 100 drops per minute, after which it should be cut to 50 or 60 drops. The maintenance dose rate is about 30 drops (3 litres in twenty-four hours).

The programme of rates to be used should be written down on the treatment card or fluid balance chart, and without further instructions the nursing staff should not exceed this rate.

Injection of Drugs and other Additives.—These may be given either via diaphragm of the connector close to the cannula (fig. 80), or by using a Y connection to another infusion bottle. In like manner an infusion pump can be used to administer small volumes of a drug accurately and continuously, e.g. heparin (fig. 151).

I.V. Filtration/Air Elimination.—Fig. 81 depicts a filter which will exclude microbial contaminants and reduce the incidence of thrombophlebitis, as well as venting entrained air.

Continuous rectal infusion (proctoclysis) was introduced by the celebrated J. B. Murphy, of Chicago, yet to-day it is seldom employed. The administration of fluid via the rectum has the advantage of simplicity. It requires but little apparatus, and neither asepsis nor isotonicity.

Avery Jones advises *dilute* saline solution for proctoclysis, *viz.* to 1 pint (568 ml) of isotonic saline solution is added to 4 pints (2·3 litres) of *tap water*. Dilute saline given per rectum avoids the absorption of an excessive quantity of water that may well result in hydræmia. Via a catheter in the rectum, the fluid is given by the drip method according to the needs of the patient. The rate of administration should not be greater than 50 drops a minute. It should be noted that there is no point in administering dextrose per rectum for its calorie value, because too little of it is absorbed.

Charting Fluid Intake and Output (figs. 82 and 83).—It is essential that the amount of fluid the patient receives is recorded. It is equally important that all urine passed in each twenty-four hours is measured and recorded. To this figure is added fluid recovered by gastro-intestinal

John Benjamin Murphy, 1857–1916. Professor of Surgery, Northwestern University, Chicago, Illinois, U.S.A. Introduced Continuous Rectal Infusion in 1908.
Sir Francis Avery Jones, Contemporary. Consulting Physician, Central Middlesex Hospital, London.

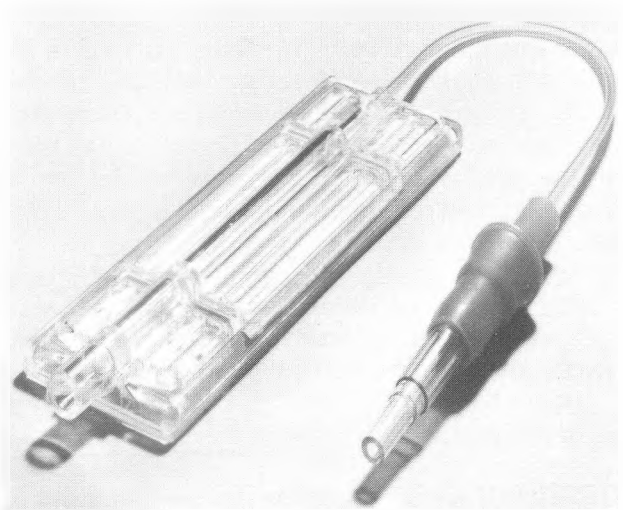


FIG. 81.—The Pall® i.v. filter/air eliminator[1].

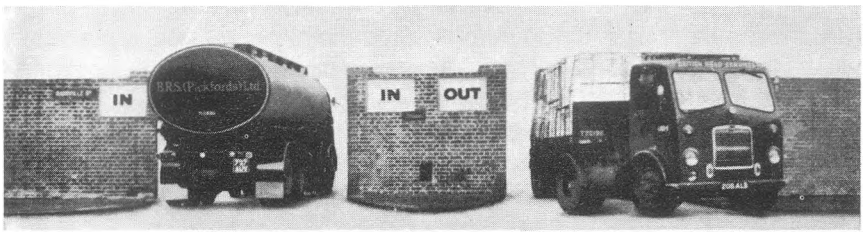


FIG. 82.—What goes in should balance with what comes out.

FLUID BALANCE CHART

HOUR ENDING	ORAL		INTRAVENOUS		URINE	ASPIRATION OR VOMIT	OTHER
	AMOUNT	TYPE	PUT UP	GIVEN			
24 hour TOTALS							

FIG. 83.—Fluid balance chart. There is no column for insensible loss because the nurse is unable to measure this. Insensible loss is taken into consideration by the Medical Officer when he writes up orders for the ensuing twenty-four hours.

[1] Pall Biomedical Ltd., Portsmouth, PO6 1TD, England.

aspiration, and that lost from an intestinal, urinary, biliary, or pancreatic fistula plus 1000 ml for insensible loss from the lungs and skin. If the patient has been sweating, the last figure is increased.

Other Aids to Maintaining Correct Fluid Balance.—In addition to the fluid balance chart, the following also are important:

1. **Clinical.**—Much information can be gleaned at the bedside.

If the fluid balance is correct, a warm, dry, pink, elastic skin denotes a good circulation and a satisfactory blood volume.

Circulatory overloading requires constant watchfulness, for the initial symptoms are nil and the signs non-proclamatory. A raised jugular venous pressure (fig. 84) indicates that the patient has received too much fluid, or too much salt.

2. **Blood Indices.**—Plasma electrolyte estimations, as well as those of the hæmoglobin, hæmatocrit and the plasma-proteins, are all helpful in ascertaining that the amount of fluid the patient is receiving is adequate, yet not excessive.

3. **Weight.**—Daily weighing is extremely useful in assessing that the amount of fluid given is correct. In patients subsisting entirely on parenteral fluid there should be a daily *loss* of weight of 150 g.

4. **Blood Volume Estimations** (p. 72).

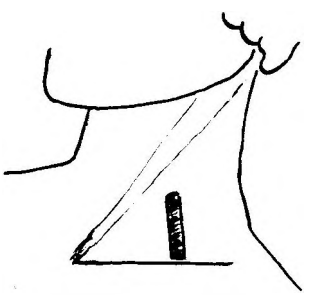


FIG. 84.—Fullness of the external jugular vein in a patient undergoing fluid therapy indicates that too much fluid has been given (see Central Venous Pressure, p. 73).

Vitamin Requirements (see also p. 105).—Antibiotics produce vitamin deficiency, notably of complex B. Therefore it is best to err on the side of safety and assume that a liberal amount of vitamins B will be required. Vitamin C should be given in large doses, to ensure saturation.

Twin ampoules of Parenterovite[1] are recommended once or twice a week. They are mixed before being injected intramuscularly or via the intravenous drip.

ACID-BASE BALANCE

In health the reaction of the blood lies within the range pH 7·36 to 7·44. The terms acidosis and alkalosis in clinical practice indicate a change or a tendency to a change in the pH of the blood in a particular direction. In acidosis there is an accumulation of acid or a loss of base causing a fall or a tendency to a fall in the pH. The converse occurs in alkalosis. The pH of the blood is regulated and controlled by various buffering systems essentially consisting of weak acids and bases, of which the most important is the bicarbonate-carbonic acid ratio $\left[\frac{HCO_3}{H_2CO_3}\right]$. It is also regulated by the removal of CO_2 by the lungs and by the excretion of both acids and bases by the kidneys.

The ratio of bicarbonate to carbonic acid is normally 20:1. Alteration in this ratio alters the pH regardless of the absolute values of the bicarbonate

[1] Contains large doses of vitamin B complex and ascorbic acid (Vitamins Ltd.).

and carbonic acid. A decrease in the ratio leads to increased acidity, and vice versa.

The bicarbonate level can be altered by metabolic factors, while the carbonic acid level is subject to alteration by respiratory factors. Alteration of one is followed automatically by a compensatory alteration in the other so that the ratio (HCO_3:H_2CO_3) and therefore the pH of the blood remains constant.

Measurement of Acid-base Disturbances.—The Astrup apparatus, and similar equipment has facilitated the measurement of acid-base disturbances in routine clinical practice. This equipment with the aid of nomograms allows rapid calculations of the blood pH, PCO_2, PO_2, standard bicarbonate and base excess or deficit. These measurements are ' normally made on arterial or arterialised capillary ' blood. PCO_2 is a measurement of the tension or partial pressure of carbon dioxide in the blood. The normal arterial PCO_2 is 31–42 mmHg (4·1–5·6 kPa). PO_2 is a measurement of the tension or partial pressure of oxygen in the blood. The normal arterial PO_2 is 80–110 mmHg (10·5–14·5 kPa).

Standard bicarbonate is the concentration of the plasma bicarbonate after *fully oxygenated* blood has been equilibrated with CO_2 at 40 mmHg (5·3 kPa) at 38°C. This eliminates respiratory causes and respiratory compensation for altered bicarbonate levels. Normal 22–25 mEq/l (mmol/l).

Base excess or deficit expresses in mEq (mmol) the total of buffer anions present in the blood in excess or deficit of normal. (Normal base excess or deficit ± 2·5). Base excess or deficit multiplied by 0·3 times the body weight in kg gives the total extracellular excess or deficit of base in mEq (mmol) (Mellemgaard and Astrup). Metabolic causes of acid base disturbances are indicated by changes in the standard bicarbonate level and base excess or deficit. Respiratory causes of acid base disturbances are indicated by changes in the PCO_2 and PO_2.

ALKALOSIS

Metabolic alkalosis (a condition of base excess or a deficit of any acid other than H_2CO_3) can be caused by:

(*a*) Excessive ingestion of absorbable alkali. Such is not uncommon in patients who take proprietary indigestion remedies without medical supervision.

(*b*) Loss of acid from the stomach by repeated vomiting or aspiration.

(*c*) Cortisone excess, usually the result of over-administration of adrenal corticoids, but occasionally due to Cushing's syndrome (Chap. 36).

Compensation is effected by (i) retention of carbon dioxide by the lungs and (ii) excretion of bicarbonate base by the kidneys (alkaline urine).

Alkalosis due to loss of acid from the stomach is by far the most common and most important. In its most typical form it is seen in patients with pyloric stenosis in whom the loss of acid by repeated vomiting is often accentuated by the taking of medicines containing sodium bicarbonate. The most striking feature of severe alkalosis is Cheyne-Stokes' respiration with periods of apnœa lasting from five to thirty seconds. Tetany sometimes occurs. Latent tetany (in which the serum calcium remains just above the low value necessary for tetanic muscular contractions) is more common, and can be unveiled by Trousseau's sign (Chap. 36). Regarding other signs, the dual phenomenon of severe alkalosis and hypokalæmia (p. 92) are so interwoven that their clinical separation is well-nigh impossible. Subclinical degrees of alkalosis are recognisable only by a raised standard bicarbonate

K. Mellemgaard, Contemporary. Physician, Rigshospitalet, Copenhagen.
Poul Astrup, Contemporary. Clinical Chemist, Rigshospitalet, Copenhagen.
John Cheyne, 1777–1836. Physician, Meath Hospital, Dublin (1811–1831). First Professor of Medicine, Royal College of Surgeons in Ireland (1813–1828), and Physician General to the Forces in Ireland (1820–1831). Described this type of Periodic Respiration in 1818.
William Stokes, 1804–1878. Physician, Meath Hospital, Dublin (1826–1875), and Regius Professor of Medicine, Dublin (1845–1878). In an account of Periodic Breathing in 1846, he acknowledged Cheyne's observation.

concentration and a positive base excess. Severe alkalosis may result in renal epithelial damage, and consequent renal insufficiency.

Treatment.—Metabolic alkalosis without hypokalæmia seldom requires direct treatment. The cause of the alkalosis should be removed where possible and a high urinary output encouraged.

Hypokalæmic Alkalosis is seen in patients who have lost potassium and acid due to repeated vomiting from pyloric stenosis. The low serum potassium causes potassium to leave the cell and be replaced by Na^+ and H^+ ion. The shift of H^+ ion into the cell causes intracellular acidosis and increases the extracellular alkalosis. The kidneys continue to excrete K^+ and an acid urine due to intracellular acidosis of the kidney cells themselves.

Treatment.—An ampoule of 20 mEq (mmol) of potassium chloride is added to 500 ml 5% dextrose solution and given intravenously.

Respiratory alkalosis a condition where the CO_2 tension in the arterial blood (PCO_2) is below the normal range of 31–42 mmHg (4·1–5·6 kPa), is caused most commonly in surgical practice by excessive pulmonary ventilation carried out upon an anæsthetised patient who has been given a muscle relaxant. Other causes are hyperventilation occasioned by high altitudes hyperpyrexia, a lesion of the hypothalamus, and hysteria. Compensation, which depends on increased renal excretion of bicarbonate, usually is inadequate. During anæsthesia alkalosis is accompanied by pallor and a fall of blood-pressure. In severe cases respiratory arrest follows.

Treatment.—Respiratory arrest due to alkalosis is rectified by insufflation of carbon dioxide.

ACIDOSIS

Metabolic acidosis (a condition where there is a deficit of base or an excess of any acid other than H_2CO_3) occurs as a result of:

(*a*) *Loss of bases* such as occurs in sustained diarrhœa, ulcerative colitis, gastrocolic fistula, a high intestinal fistula, or prolonged *intestinal* aspiration.

(*b*) *Increase in fixed acids* due to the formation of ketone bodies as in diabetes or starvation, the retention of metabolites in renal insufficiency, and the rapid increase of lactic and pyruvic acids by anærobic tissue metabolism due to shock, following cardiac arrest (Chap. 41), or the release of the clamped aorta in the surgery of abdominal aneurysm (p. 150). Acute acidosis with pH levels of 7·1 are frequently encountered in such cases.

Clinical Features.—In severe acidosis the leading sign is rapid, deep, noisy respirations which are unremittent save, perhaps, momentarily while the patient endeavours unsuccessfully to moisten his dry lips with his parched tongue. The hyperpnœa is due to over-stimulation of the respiratory centre by the reduction of pH of the blood, and the physiological purpose of overbreathing is to eliminate as much as possible of the acid substance H_2CO_3. Except in renal acidosis, the pulse-rate and the blood-pressure are raised. The urine is strongly acid. The standard bicarbonate level is lowered and there is a base deficit (p. 99 acid-base measurements).

Treatment.—Cases belonging to class (*a*) are readily rectified by the administration of Ringer's lactate solution or slow infusion of dilute sodium

bicarbonate solution. When dehydration is in evidence the prelude to treatment with sodium bicarbonate solution should be rapid infusion of sufficient isotonic saline solution to restore the extracellular fluid volume. The treatment of acidosis due to renal failure is discussed in Chap. 55.

The acute acidosis after releasing a cross-clamped aorta or in cardiac arrest requires the infusion of 50–150 mEq (mmol) of 8·4 per cent sodium bicarbonate solution (see base excess, p. 100).

Acidosis due to transplantation of the ureters into the colon is discussed in Chap. 56.

Respiratory Acidosis (a condition where the PCO_2 is above the normal range) is caused by impaired alveolar ventilation.

In practice this problem most commonly occurs when there is inadequate ventilation of the anæsthetised patient, or when the effects of muscle relaxants have not worn off or been fully reversed at the end of the anæsthetic. There is also a risk of respiratory acidosis when the patient undergoing surgery already has pre-existing pulmonary disease, (e.g. chronic bronchitis or emphysema), and this is accentuated by thoracic and upper abdominal incisions.

MEASUREMENT OF ACID-BASE DISTURBANCES

In addition to the older and better known methods of assessing the metabolic balance of the patient (such as the standard bicarbonate), it is important to know the respiratory balance of a patient. Direct estimations of the pH, PCO_2 and PO_2 of arterial blood are made, though in most situations the pH and PCO_2 can be measured using capillary blood.

Examples of acid-base disturbances

1. pH 7·30, Pco_2 60 mmHg (8 kPa), Base excess + 1. This is uncompensated respiratory acidosis.
2. pH 7·21, Pco_2 40 mmHg (5·3 kPa), Base excess − 10. This is a severe metabolic acidosis. A 70 kg man with this acid-base picture would require 70 × 0·3 × 10 mEq (mmol) of bicarbonate (see base excess, p. 100).
3. pH 7·29, Pco_2 60 mmHg (8 kPa), Base excess + 5. This is a respiratory acidosis with a compensatory metabolic alkalosis.
4. pH 7·20, Pco_2 64 mmHg (8·5kPa), Base excess − 8. This is a mixed respiratory and metabolic acidosis.

NUTRITION

Malnutrition.—In surgical practice, the nutritional state of the patient always demands careful consideration, for it is to be remembered that malnutrition is common, being present before or occurring after operation in about 50 per cent of patients, possibly more in some parts of the world.

Preoperative Malnutrition may be due to starvation or to a failure of digestion. Starvation is caused by (i) difficulty in obtaining food (poverty), (ii) difficulty in swallowing food (dysphagia), and (iii) difficulty in retaining swallowed food (vomiting). Failure of proper digestion may, for example, be due to pancreatic or biliary disease (carcinoma, jaundice due to stones), and duodenal and jejunal conditions (fistula, blind loop syndrome).

Postoperative (*Post-traumatic*) *Malnutrition* is hopefully in most cases of a transient nature consequent upon a short period of starvation and the stress reaction to trauma. Recovery from any nitrogen deficit due to protein catabolism will follow on the return of normal feeding. But any delay in return to a normal diet such as may be imposed by the dictates of the operation (œsophagectomy), or a complication (paralytic ileus from peritonitis) means that severe malnourishment is likely to occur.

Hypercatabolic State.—Severe sepsis (subphrenic abscess), severe trauma (burns) and other severe disturbances of major viscera (pancreatitis, lobar pneumonia) are accompanied by an accelerated and profound breakdown of tissue proteins.

TYPICAL DAILY POSTOPERATIVE NITROGEN LOSS

	Nitrogen Loss g	*Muscle Loss g*
Herniotomy	3	80
Appendicectomy	6	200
Cholecystectomy	12	320
Fractured Femur	15	400
Partial Gastrectomy	15	400
Œsophagectomy	90	2,500
Peritonitis	18	570
Sepsis	23	730

In starvation the metabolic changes are directed to minimising tissue loss and in some circumstances a man can survive for about 120 days. Glucose reserves are available only for 24 hours and thereafter are derived principally from muscle, so that catabolism begins almost immediately after food deprivation. In the first 72 hours there is a rapid weight loss due to loss of sodium and water, then the resting metabolic expenditure falls and daily nitrogen losses over two weeks fall from about 10 g to 3–4 g. Progressively fat provides most of the energy requirements yielding 9 cal/g, while carbohydrate derived by gluconeogenesis in the liver from amino acids is utilised by brain, adrenal glands and red cells—all obligatory glucose users. After about 21 days the central nervous system adapts to using ketones derived from fat. The gluconeogenesis and ketosis of starvation may be easily inhibited by glucose intake.

After injury or surgical operation there is an increased oxygen and calorie consumption and a negative nitrogen balance (Cuthbertson). The increase in resting metabolic expenditure ranges from minimal after uncomplicated surgery to 30% with multiple fractures, 45% in peritonitis and up to 100% in burns. An increase in metabolic rate and protein catabolism of more than 25% is regarded as a hypercatabolic state.

The endocrine profile, activated by fear, apprehension and nervous stimuli from damaged tissues is altered after stress with an increase in secretion of most hormones in turn resulting in changes in substrate handling by the body.

Glycogen breakdown in muscle and liver is accelerated principally by adrenaline and glucagon leading to increased blood glucose levels, while increased cortisol and glucagon induces gluconeogenesis from amino acids. Lipolysis—fat breakdown—is increased by growth hormone glucagon and noradrenaline. Thus control of glucose levels is impaired, and, together with depressed peripheral clearance, results in ' the diabetes of injury '.

The stimulated protein breakdown in the postoperative period is associated with a change in synthesis rate in the body cell mass and this results in a negative nitrogen balance indicating loss of protein derived from muscle and viscera. Larger losses are

Sir David Cuthbertson, C.B.E., Contemporary. Honorary Consultant in Clinical Biochemistry, Glasgow Royal Infirmary, Scotland.

seen in muscular athletic men and the smallest losses in wasted patients. Epidural anæsthesia inhibits the normal postoperative increase in cortisol adrenaline aldosterone and growth hormone, and so may reduce the negative nitrogen balance.

The Effects of Malnutrition include poor wound healing manifest as wound dehiscence (p. 3) and leaking anastomoses of bowel, delayed callus formation, disordered coagulation, reduced enzyme synthesis, immunological depression with increasing susceptibility to infection, decreased tolerance to radiotherapy and cytotoxic chemotherapy, all with the severe mental apathy and physical exhaustion of the patient which adds to the problems of nursing care.

SOME CLINICAL INDICATIONS FOR NUTRITIONAL SUPPORT

1. Preoperative Nutritional Depletion
2. Postoperative Complications
   a. Ileus more than 4 days
   b. Sepsis
   c. Fistula formation
3. Intestinal Fistula
4. Massive Bowel Resection
5. Management of
   a. Pancreatitis
   b. Malabsorption Syndromes
   c. Ulcerative Colitis
   d. Radiation Enteritis
   e. Pyloric Stenosis
6. Anorexia Nervosa
7. Intractible Vomiting
8. Maxillo-facial Trauma
9. Traumatic Coma
10. Multiple Trauma
11. Burns
12. Malignant Disease
13. Renal Failure
14. Liver Disease
15. Cardiac Valve Disease

Assessment and Management

Thus it is essential for the clinician to be aware of the need (a) to assess the state of nutrition of a patient, and if malnutrition is present or threatens (b) to consider the nutritional requirements and then (c) to use methods of sustaining normality or rectifying any deficiency.

(*a*) **Assessment.**—A malnourished patient has a characteristic appearance, lean and hungry in most cases of starvation, lean and apathetic in post-traumatic depletion, with a superimposed hectic flush around sunken cheeks and pinched nose in a hypercatabolic state. The clinician as he places his comforting hand on the patient's shoulder discerns the bony scapula bereft of almost all its muscle. However, these clinical observations only detect gross malnutrition and therefore measurement of the nutritional status is essential (Goode). The parameters include:—

(i) *Body weight.*—Careful weighing on a bed weighing machine is the obvious way of detecting the progress or otherwise of the patient. The desirable weight of the patient can be checked by reference to the appropriate tables, or by applying the body mass index (B.M.I.). $= \frac{\text{Wt (kg)}}{\text{Height}^2\text{ (m)}}$. A woman should have an index of 20, 21, or 23, and a man 20·5, 22, 23·5 according to size of frame (small, medium, large).

(ii) *Upper arm circumference.*—Feeding is indicated if less than 23 cm in females, and 25 cm in males.

Anthony William Goode, Contemporary. Senior Lecturer in Surgery, Charing Cross Hospital Medical School, London.

(iii) *Triceps skin fold thickness.*—Using a skin fold caliper, the minimum is 13 mm in females and 10 mm in males.

(iv) *Serum albumin* should not be less than 35 g/l.

(v) *Lymphocyte count.*—Less than 1 500/mm^3 indicates an impaired cellular defence mechanism.

(vi) *Candida skin test.*—A negative reaction also means defective cell mediated immunity.

(vii) *Nitrogen balance studies.*—The total nitrogen intake is compared with the loss from all sources, such as urine, fistula drainage and nasogastric aspirate (1 litre = 1 g nitrogen). A greater loss than intake indicates a negative balance and tissue breakdown. A positive balance means anabolism-tissue synthesis.

DAILY CALORIE AND NITROGEN REQUIREMENTS
(*after Ivan D. A. Johnston*)

	Postoperative	*Hypercatabolic States*
Water ml/kg	35	40–45
Kcal/kg	35	45–55
Kcal/g Nitrogen	180	200–280
Nitrogen g/kg	0·2	0·25–0·30

Other measurements include those determining the rate of muscle breakdown, such as urinary creatinine excretion, or 3-methyl histidine excretion. Total body potassium and nitrogen are used to assess the absolute size of the body cell mass. C_{14} leucine incorporation is a measure of the synthesis rate, while serum transferrin is used a measure of visceral protein synthesis (needs to be more than 1·5 g/l).

(*b*) **Nutritional requirements** include carbohydrate, fat, protein, vitamins, minerals and trace elements.

Calories are provided by carbohydrate and fat. A healthy adult at rest requires 1 500–2 000 non-protein calories per day for energy. Carbohydrate provides 4·1 kcal/g (16·8 MJ/g) and fat 9·1 kcal/g (37·8 MJ/g). The number of non-protein calories given should bear definite relationship to the nitrogen intake. A calorie nitrogen ratio of about 200:1 is usually adequate but a higher ratio is sometimes necessary. When fat is used as a source of calories, 30% of the total calories should be provided by carbohydrate. 'Carbohydrate provides the fire in which fat is burnt.' It is useful to remember the classical formula for a nonketogenic diet—$F < 2C + P/2$.

Nitrogen requirements.—The minimum for dynamic tissue turnover and so to keep a healthy adult in positive nitrogen balance is about 35–40 g of protein or 5·5–6·5 g of nitrogen per day. The hypercatabolic patient requiring hyperalimentation may need three or four times this amount of protein. A daily negative nitrogen balance of 10 g N is not unusual and is equivalent to a loss of 62·5 g of protein or 300 g of muscle tissue.

Vitamins.—Whatever the method of feeding, vitamins are necessary as

Ivan David Alexander Johnston, Contemporary. Professor of Surgery, University of Newcastle-upon-Tyne.

supplements, as they are essential for the maintenance of normal metabolic function and cannot be synthesised by the body.

The water-soluble vitamins B and C act as coenzymes in collagen formation and wound healing. Postoperatively the vitamin C requirement increases to 60–80 mg/day. Preoperative depletion is exacerbated by anorexia, smoking, aspirin and barbiturate therapy. Vitamin B_{12} is given 500 μg i.m. weekly particularly to those with initial low levels (cœliac disease, Crohn's disease, ileal resection or bypass, blind loop syndrome, tapeworm infestation, reduced pancreatic secretion, tropical sprue, excess alcohol intake, anticonvulsant therapy, and after gastric surgery). As the serum folate falls, especially in those on parenteral nutrition, folinic acid is required daily in doses of 3–6 mg i.m.

The fat-soluble vitamins A, D, E, and K are reduced in steatorrhœa and the absence of bile. Vitamin A, 5000 units per week, is required after surgery, and when appropriate it enhances the antitumour effect of cyclophosphamide. Vitamin K 5–10 mg i.m. weekly reduces any bleeding tendency. If commercially available vitamin additives are put into an infusion, the container should be protected from the light.

Minerals and trace elements.—In addition to the sodium and potassium balance, iron deficiency must be sought and corrected. Calcium and magnesium (pp. 93, 94). Zinc deficiency is manifest as a rash on the face and perineum which does not respond to antifungal therapy, stomatitis which causes disturbance of taste (dysgeusia), and alopecia. Copper deficiency results in leucopænia and anæmia, while lack of chromium may give rise to glucose intolerance. As well as deficiency due to gross malnutrition and diarrhœa, long-term parenteral nutrition can result in depletion. Of the 14 trace elements that are considered essential for normal enzyme activities within the body are also included manganese, cobalt, molybdenum and vanadium.

(*c*) **Methods of Feeding.**—These are predominantly enteral and less commonly parenteral.

Enteral Nutrition

(1) **By mouth.**—Obviously, as this is the natural way it should always be attempted. Only when it is known that this route cannot be used or is ineffective are other methods considered.

Feeding by mouth demands *common sense, cleanliness and compassion* on the part of the medical attendants. It is *common sense* to ensure that an adequate, palatable and varied diet, including all the nutritional requirements is provided at regular intervals, more frequently than regular meal times if necessary. It is common sense to begin with a liquid diet as soon as bowel sounds return after an abdominal operation, and not, for example, to allow a plate of fish and chips to be put in front of a patient the day after his gangrenous appendix has been removed. Promotion to semi-solid (light) and then to more solid food (full diet) follows in steps of 3–7 days according to progress. *Cleanliness* in the preparation and serving of food, and of the utensils used is

of paramount importance in avoiding gastrointestinal infection causing vomiting and diarrhœa. A salmonella infection amongst elderly patients and children may be a mortal blow. *Compassion* is needed to ensure that the patient actually receives and ingests the proffered food. Food must be placed within reach of an enfeebled patient. Assistance is often required and should be freely given.

(2) **By tube.**—A nasogastric tube which has been passed to allow regular gastric aspiration to be performed may also be used for feeding liquidised diets. Fine bore tubing can be used instead, being favourably received by most patients as not so irritating as the larger tube. It is invaluable when passed with the aid of an endoscope through an œsophageal stricture into the stomach, enabling thereby the effects of starvation to be reversed.

ENTERAL DIETS[1] (Constituents per Litre)

	Nitrogen (g)	*Kcal/g N*	*Fat (g)*	*CHO (g)*	*Osmolality mOsm*	*Relative cost*
London Hospital Diet	8	186	60	80	369	1
Complan + Caloreen or Calonutrin supplement	4·8	196	24	178	444	1
Clinifeed 400	4·8	142	27	110	306	3
Vanilla	4·8	142	27	110	310	3
Chocolate	9·6	79	22	140	567	3
500 Vanilla						
Isocal	5·4	172	44	133	290	5
Flexical	3·7	252	34	153	450	7
Vivonex HN (High Nitrogen)	6·0 as amino acids	127	0·8	208	800–850	10

Technique of Fine Bore Tube Insertion and Usage.—The patient may be sitting or lying, preferably the former. The introducer wire is lubricated with water and inserted into the fine bore tube. The tube is passed through the nose via the nasopharynx and œsophagus into the stomach. Withdraw the wire and tape the tube to the patient. Check the position of the tube by x-ray as passage into a bronchus can occur quite easily. Alternatively a quick check can be made by injecting 5 ml of air down the tube and listening for its bubbling entry into the stomach through a stethoscope.

Administration of the feeds is either by gravity or by means of an infusion pump.

Notes on Problems of Tube Feeding.—Gastric emptying should be normal. In ill patients ensure that the stomach empties by injecting 60 ml water down a nasogastric tube and aspirate 4 hourly. If after 24 hours fluid is passing through the stomach, commence feeding for the first day and aspirate intermittently to ensure that the stomach empties. Then remove the tube and introduce the fine-bore tube.

Blockage of a 1 mm bore tube is cleared by flushing through with 2 ml water—do not add effervescent potassium to the feed as curdling will follow.

Most fine-bore tubes incorporate a male ' luer ' lock connector making the nasogastric drip system incompatible with intravenous lines.

Check the drip rate hourly.

[1] Complan (Farley-Health Products, Plymouth, England).
Clinifeed, Caloreen (Roussel Laboratories, Wembley, Middlesex, England).
Calonutrin (Geistlich Sons, Newton Bank, Long Lane, Chester, England).
Isocal, Flexical (Mead Johnson Laboratories, Bristol, England).
Vivonex (Eaton Laboratories, Woking, Surrey, England).

All feeds should be stored at 4°C until use, not exposed to room temperature for more than eight hours, and discarded if not used after twelve hours. As diet kitchens may be a source of klebsiella infection the bacteriological monitoring of feeds is desirable.

Unwanted effects.—Nausea, vomiting and pulmonary aspiration are avoided by regulation of the infusion rate and ensuring initial gastric emptying. Diabetes and hyperosmolar states are related to high carbohydrate intake with particular hazard for the established diabetic. *Diarrhœa* is common and the pathogenesis is not fully understood, but fluid and electrolytes are secreted into the bowel in response to a high osmotic load. Also the use of broad spectrum antibiotics is associated with diarrhœa. Commence with half strength feed and increase slowly to standard concentration over days given at a slow constant rate, avoiding milk because of possible lactose intolerance, and a high fat content giving steatorrhœa. Mild disturbances of liver function can be associated with enteral feeding.

(3) **Gastrostomy. Jejunostomy.**—These time honoured methods are especially useful when carried out at the end of any operation which is likely to be followed by a period of malnutrition (e.g. peritonitis with bowel decompression and resection). On such an occasion it is used for gastric drainage and avoids the use of a nasogastric tube, and as soon as bowel sounds return it is used for feeding. A simple rubber or plastic tube (8–10 mm diam) with an additional hole made near the tip is passed through a separate ' stab ' incision through the upper left rectus muscle. On other elective occasions a short muscle-splitting incision is made (even under local anæsthesia). The tube is inserted through a ' stab ' incision in the anterior wall of the stomach for about 10 cm and fixed to the wall by catgut. Using two rows of purse-string sutures tube and stomach are pushed in to form an ink-well (Kader–Senn). The stomach is tacked to the anterior parietes. The tube should be directed towards the fundus of the stomach as paradoxically it seems to function better in this direction. The tube is secured to the skin by tunnelling within an adhesive plaster dressing and connected to a bag (or the feed) using wide bore connections to avoid blockage by curds, mucus etc. The use of catgut enables the tube to be removed when desired after about ten days.

A jejunostomy is not often used. The tube is inserted, pointing distally this time, and then is laid on the jejunum and enclosed in a guttered tunnel using catgut (Witzel).

Parenteral Nutrition

Parenteral nutrition by intravenous feeding is used in less than 4–5 per cent of all hospital admissions, either when enteral feeding is not possible, or to supplement imperfect enteral feeding. Contraindications include cardiac failure, severe liver disease, disorders of fat metabolism, uncontrolled diabetes, shock and severe blood dyscrasias. It must be remembered that total parenteral nutrition is not to be undertaken lightly. It is potentially hazard-

Monitoring Feeding Regimens

Daily	Body weight Fluid balance Full blood count, urea and electrolytes Blood glucose Urine and plasma osmolality Electrolyte and nitrogen analysis of urine and gastrointestinal losses Acid base status
Thrice Weekly	Serum calcium magnesium and phosphate Plasma proteins Liver function tests Clotting studies
Ten Days	Serum B12, folate, iron lactate and triglycerides Trace elements

Bronislaw Kader, 1863–1937. Surgeon, Poland.
Nicholas Senn, 1844–1908. Surgeon, U.S.A.
Friedrich Oskar Witzel, 1856–1925. Surgeon, Germany.

ous and can be dangerous in inexperienced hands. The recent trend towards the formation of multidiscipline nutritional care teams is a major advance and their advice should be sought.

When planning an intravenous feeding regimen, first weigh the patient and calculate the fluid needs for the next 24 hours. Calorie and nitrogen intake should be calculated on a body weight basis remembering that daily needs may change. Fat may provide up to 30% of total daily calories. Daily biochemical patient monitoring is essential.

SOME AVAILABLE SOLUTIONS FOR I.V. FEEDING

	Non-protein Calories per litre	*N/g/l*	*Na*	*K*	*Mg*	PO_4	*Ca*
			(mmol/litre)				
Vamin Glucose	400	9·4	50	20	1·5	—	2·5
Synthamin 14	—	14·3	73	60	5	30	—
Aminoplex 14	—	13·4	35	30	—	—	—
Intralipid 20%	2,000	—	—	—	—	15	—
Dextrose A (20%)	760	—	—	—	28	—	26
Dextrose B (20%)	760	—	—	60	—	60	—

As most glucose solutions are hypertonic—50% dextrose solution, 3800 mosmol/litre—and irritant, they can only be given through central veins, the cannulation of which requires technical finesse. Attempts to avoid this have centred around the peripheral administration of isotonic amino acid solutions alone or with fat (280–320 mosmol/litre), however the efficacy remains sub judice.

Technique for Central Venous Catheter Insertion with a Skin Tunnel (fig. 85).—The procedure is carried out in a clean room using strict aseptic technique. The patient is sedated and placed in a head down position. A silicone rubber catheter of 1 mm diam (Vygon[1]) is inserted by infraclavicular approach, the introducer inserted under local anæsthetic through a 1 cm skin incision (A) 2 cm below the midclavicular point. The position of the catheter is checked radiologically ensuring the tip is in the superior vena cava or right atrium. The catheter hub is removed and the introducer withdrawn. The introducer is now inserted through the skin puncture at (B) about 7 cm below and medial to (A); it is passed through the subcutaneous tissue to emerge at (A) and the catheter threaded through the introducer at (A) until it is seen within the transparent introducer at (B). The introducer is partially withdrawn and cut at X—X so that 2 cm remains in the tunnel and 2 cm protrudes, and the catheter pushed

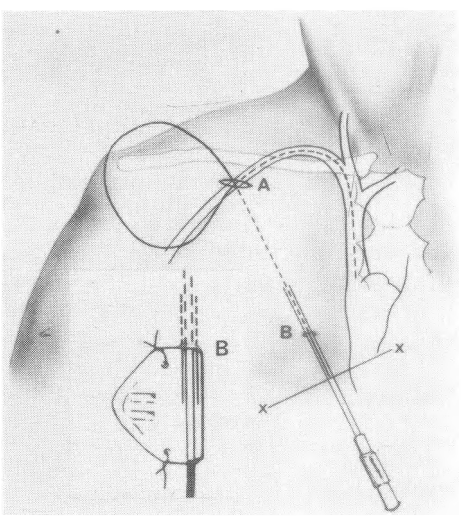


FIG. 85.—Subclavian catheterisation with a skin tunnel (*after J. Powell-Tuck*).

[1] 'Vygon' U.K. Ltd., Uxbridge, England.

Jeremy Powell-Tuck, Contemporary. Senior Registrar, Charing Cross Hospital, London.

fully through the 4 cm sleeve avoiding kinks at (A). A plastic clip grips the sleeve and the emerging catheter and is anchored. (A) and (B) are cleaned with chlorhexidine in alcohol and sprayed with povidine iodine powder. Incision (A) is closed with a stitch and covered with a sterile dressing while at (B) a clip is placed over the catheter and sleeve of the introducer and fixed to the skin with sutures.

Commence using half strength solutions and increase to the desired daily intake over days. Additives to solutions should be avoided and given through a separate line, although central mixing in pharmacy allows addition under sterile conditions into a 3 litre bag delivery system.

Notes on Management.—Fructose sorbital or alcohol solutions are potentially disadvantageous due to an associated lactic acidosis or hepatocellular damage. Fat, isotonic preparations of vegetable oils in water with an emulsification agent, or purified egg phospholipid in intralipid (to stabilise the mixture) should not be used 12 hours before blood sampling as they interfere with autoanalysis. The rate of clearance of intravenous fat is increased after surgery or when energy demands are high (Feggetter).

The nitrogen sources are either casein hydrolysates, or lævorotatory isomers of amino acids. In such solutions all essential amino acids should be present with a broad spectrum of the non-essential amino acids. No single amino acid should predominate since if its utilisation is inefficient this will interfere with the utilisation of the others (Tweedle). In sepsis, renal failure and hepatic failure the use of solutions containing only essential amino acids, isoleucine, leucine, valine, phenylalanine, lysine and tyrosine is under investigation.

Complications.—The initial complications of parenteral feeding arise from *malposition of the catheter tip*, and x-ray confirmation of the site of the catheter tip is

Biochemical Complications During Parenteral Nutrition and Their Correction

Complication	*Clinical Presentation*	*Cause*	*Correction*
Hyponatræmia	Confusion Apathy	Water intoxication G.I. or renal loss	Reduce water intake Increase Na^+ intake
Hypokalæmia	Apathy Cardiac irregularity	Inadequate K^+ intake G.I. or renal loss	Increase K^+ intake
Hypomagnesemia	Weakness Vertigo Convulsions	Inadequate Mg^{++} intake G.I. or renal loss	Increase Mg^{++} intake
Hypophosphatæmia	Apathy, confusion, Panesthesis, RBC oxygen dissociation	Inadequate inorganic phosphate intake	Increase PO4 intake
Azotæmia	Apathy	Dehydration Calorie: Nitrogen imbalance	Increase water intake Increase Calorie: Nitrogen ratio to 185:1
Hyperammonæmia	Apathy, seizures, coma	Hepatic dysfunction Amino acid deficiency	Reduce infusion rate or stop
Hyperglycæmia	Increased blood sugar	Unsuspected diabetes Underlying sepsis Excess infusion of carbohydrate	Treat underlying cause Treat underlying cause Reduce infusion rate
Hypoglycæmia	Lethargy Vasoconstriction	Interrupted infusion	Give dextrose
Hyperosmolar dehydration	Hyperglycæmia Dehydration Increased serum sodium and osmolality Apathy Coma	Hyperglycæmia	Correct hyperglycæmia and give half strength saline

Jeremy George Weightman Feggetter, Contemporary. Surgeon, Ashington and Freeman Hospitals, Newcastle-upon-Tyne.
David Ernest Frederick Tweedle, Contemporary. Senior Lecturer in Surgery, University of Manchester.

mandatory before infusion. *Infection*, particularly septicæmia, arises from continuous direct vascular access and immune depression in malnourished patients, together with the solutions being ideal bacterial and fungal culture media. Catheter insertion with strict asepsis, daily care of the entry site cleaned with 1–2% tincture of iodine and alcohol solutions and the strict use of the line only for nutrient solutions all contribute to prevention. However, if a patient develops an unexplained fever, hypotension, vomiting, diarrhœa, confusion or seizures, a full clinical examination with appropriate radiology and swabs for bacteriological culture of blood, sputum, urine and catheter site are indicated. If a source of infection is discovered it is treated, but if no source is found and the fever persists for 24 hours remove the catheter and send the tip for bacteriological and fungal culture, candida albicans, staphylococci or klebsiella frequently being isolated. Recommence the parenteral regimen using a new catheter at a fresh site.

Prolonged use of amino acids and glucose alone will result in essential fatty acid deficiency, with *dermatitis*, *anæmia and increased capillary permeability*, a complication avoided by the use of intravenous fat solutions or essential fatty acids rubbed into the skin weekly. Similarly *hypophosphatæmia* is seen with such regimens and accentuated by the use of insulin, resulting in enzyme defects, ATP deficiency and a shift to the left in the oxygen dissociation curve, is prevented by a daily intake of 13 mmol of potassium dihydrogen phosphate. *Jaundice* occuring during parenteral nutrition is cholestatic perhaps the result of sepsis, malnutrition and hypoxia and feeding should be temporarily discontinued. *Metabolic acidosis*, much less common if fructose and alcohol are avoided, may arise from infusion of available hydrogen ions in amino acid solutions and is simply corrected with sodium bicarbonate solution.

Steroids. Insulin.—Anabolic steroids (e.g., Durabolin 25 mg i.m. weekly) may be used to improve nitrogen balance, but the effect may not be observed for up to 6 days. In hypercatabolism an intravenous insulin-potassium regimen has been advocated, but it must be used with great caution.

CHAPTER 9

SKIN. BURNS. SKIN GRAFTING

CALLOSITIES, CORNS, AND WARTS

A **callosity**[1] (*French: callosité*) is a localised thickened or hardened part of skin caused by friction. It is commonly occupational, e.g. on a gardener's hand or the finger of a violinist.

A **corn** (*Old French: corn = grain*) is a horny induration of the cuticle with a hard centre, caused by undue pressure, chiefly affecting toes and feet. Most corns yield to the chiropodist, provided that footwear is suitable. Skilled treatment is important in patients with diabetes or a poor peripheral circulation, as secondary infection may precipitate gangrene Salicylic acid in collodion (20 per cent) applied for a few nights, followed by soaking in hot water, is often effective in removing a corn.

A **wart** (*Old English: wearte*) is a dry rough excrescence on the skin (fig. 86). *The common wart* usually occurs on the hands, particularly in children. It is due to a virus and is infectious. As warts often disappear spontaneously, any treatment which is in use at the time will gain an undeserved reputation as a wart 'cure'. Curetting and cauterisation of the centre is the radical treatment, but further crops may appear.

FIG. 86.—Warts on the hand.

Plantar warts occur in the sole, and are usually multiple. They may be so tender as to render standing or walking exceedingly uncomfortable.

Treatment.—The nightly application of 3 per cent formaldehyde lotion for eight weeks is effective in 60 per cent of cases. Curettage is more certain. Under a local or general anæsthetic, a scalpel, held vertically to the skin, incises the hard skin at the boundary of the lesion. A sharp curette can be inserted beneath the wart to remove it.

The footwear must be treated if re-infection by the virus is to be avoided. Cotton socks should be worn, as these can be boiled. Shoes can be disinfected by formalin vapour.

Seborrhœic (*syn.* '*Senile*') warts, see p. 115.

Venereal warts and moist warts (papillomata acuminata), see Chapter 59.

INFECTIONS

Impetigo is an intradermal infection. When caused by the streptococcus the primary lesion is bullous, which soon ruptures to form an erosion and then a crust. In the staphylococcal type, the primary lesion is also bullous, but the bullæ are more durable (e.g. in neonatal pemphigus). The infection is contagious, and in rugby football one player so infected can spread the disease amongst his team mates and the opposing side (the condition being known as 'scrum-pox'). *Treatment* includes the careful washing of the face to remove crusts, using hexachlorophene soap and lotion (1 per cent). Systemic antibiotics, according to the sensitivity tests, are only used in those cases which are resistant to local treatment.

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[1] Other definitions of names of skin lesions in common usage include:
Nævus (*Latin: Nævus*) = A birth mark, but often used to mean simply a mark or blemish on the skin (see Hæmangioma and Melanoma).
Mole (*Anglo-Saxon: Mâl = a lump*) = A spot or blemish on the skin, sometimes hairy.
Mollusc (*Latin: Molluscus = soft*) = A soft protuberance on the skin.

Boil (*syn.* Furuncle).—A boil is an acute staphylococcal infection of a hair follicle, with perifolliculitis, which usually proceeds to suppuration and central necrosis. A painful and indurated swelling appears which gradually extends. After two or three days, the centre softens and a small slough is discharged with a bead of pus, and in the large majority of cases the condition then subsides. A 'blind boil' is one which subsides without suppuration. Boils are common on the back of the neck.

FIG. 87.—Hydradenitis-infection of axillary sweat glands and hair follicles (see also hyperhidrosis Chap. 28).

Infection of the perianal hair follicle (perianal abscess), with suppuration, is likely to result in a sinus. Furunculosis of the external auditory meatus is extremely painful, as the skin is attached to the underlying cartilage, and swelling is accompanied by considerable tension.

Complications of Boils.—(i) Cellulitis, especially in debilitated subjects. (ii) Infection of the lymph nodes draining the affected part. (iii) Secondary boils due to infection of neighbouring hair follicles (e.g. in hydradenitis fig. 87).

Stye (*syn.* hordeolum).—A *stye* is due to infection of an eyelash follicle.

Treatment consists of improving the general health of the patient, since boils are frequently associated with overwork, worry, debility, examinations, or other undermining influences. Incision is unnecessary, as a touch of iodine or liquid phenol on a skin pustule will hasten necrosis of the overlying skin so that pus can then escape. Should softening occur around a hair follicle, particularly an eyelash (stye), removal of the appropriate hair allows the ready escape of pus. The sensitivity of the organisms is determined and the appropriate antibiotic may be given. Washing the surrounding skin twice a day with suitable disinfectant, e.g. hexachlorophene, discourages the development of secondary boils. A paste, composed of anhydrous magnesium sulphate (24 parts) and glycerin (11 parts) exercises a valuable osmotic effect.

FIG. 88.—Carbuncle of the neck.
(*Herbert Bourns, F.R.C.S., Bristol.*)

Cellulitis and Erysipelas (pp. 27 and 35).

Carbuncle.—This is an infective gangrene of the subcutaneous tissues, due to staphylococcal infection. It is uncommon before the age of forty, and males are the usual sufferers. *Diabetes* may be present.

A carbuncle often occurs in the nape of the neck. The subcutaneous tissues become painful and indurated, and the overlying skin is red (fig. 88). Unless the con-

Carbunculus in Latin, Anthrax in Greek, is the word for charcoal. The ancients saw in these conditions burning sores upon the skin—hence they likened them to glowing coal.

dition is aborted by prompt treatment, extension will occur, and after a few days areas of softening appear, the skin gives way and thick pus and sloughs discharge. Usually there is one central large slough, surrounded by a ' rosette ' of smaller areas of necrosis. Infection may extend widely and fresh openings appear on the surface and coalesce with those previously formed.

Treatment.—The general treatment and organism identification is similar to that described for boils. Many carbuncles are aborted if penicillin is used adequately in the early stages. Local treatment by osmotic pastes is often supplemented by infra-red or short-wave diathermy.

Lupus vulgaris (tuberculosis of the skin) usually occurs between the ages of ten and twenty-five, the face being the site of election. One or more cutaneous nodules appear, with congestion of the surrounding skin. On applying pressure with a glass slide the nodules are seen to be the colour of apple jelly. Extension occurs very slowly, but ulceration is likely to follow sooner or later. The resulting ulcer tends to heal in one situation as it extends to another. The mucous membranes of the mouth and nose are sometimes attacked, either primarily or by extension from the face. Œdema occurs if the fibrosis caused by the lupus obstructs the normal lymphatic drainage. Infection in the nasal cavity may be followed by necrosis of underlying cartilage. Epithelioma is prone to occur in a lupus scar (fig. 89).

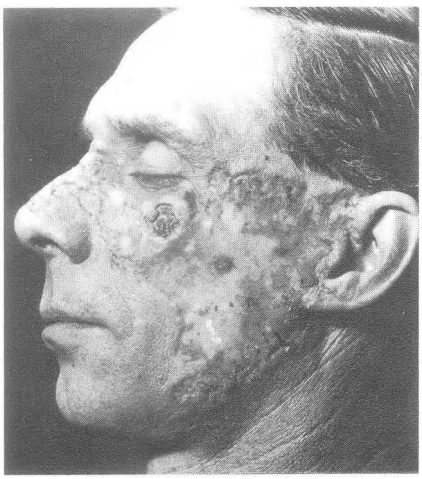


FIG. 89.—Advanced lupus vulgaris. Carcinoma has developed on the cheek.

Treatment is by chemotherapy (p. 17). If healing is slow, the lesion should be excised.

CYSTS

A sebaceous cyst (*syn.* a wen) follows obstruction to the mouth of a sebaceous duct, and is therefore a retention cyst. Pathologically, it is classed as an epidermoid cyst (p. 51). It commonly occurs on the face or scalp (Chap. 26), but can occur anywhere except on the palms and soles, which are devoid of sebaceous glands.

A typical cyst appears as a hemispherical swelling, firm or elastic in consistency, and with no definite edge. It is more or less adherent to the skin, especially if it has been previously inflamed or is subjected to pressure. The punctum of the obstructed duct can sometimes be seen on the summit of the cyst, and sebaceous material may be expressed from the duct. An uncomplicated cyst contains yellowish-white material composed of fat and epithelial cells, of a putty-like consistency, so it can often be indented by a finger-tip. Rarely, a minute worm, *Demodex folliculorum*, which is harboured in sebaceous glands, may be seen on microscopy of the cyst wall.

Complications.—(i) *Infection.*—The cyst becomes enlarged and painful, and the overlying skin is red. Recurrent attacks cause the cyst wall to become adherent to surrounding subcutaneous tissue, and consequently more difficult to remove. The contents of an infected cyst become semi-liquid and usually very fœtid.

(ii) *Ulceration.* — An infected cyst may discharge its contents. An ulcerated surface remains, which resembles an epithelioma, and to which

Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London (1800–1825). In 1821 he received a Baronetcy and one thousand guineas for successfully removing an infected sebaceous cyst from the head of King George IV.

the term Cock's 'peculiar tumour' is applied (fig. 90).

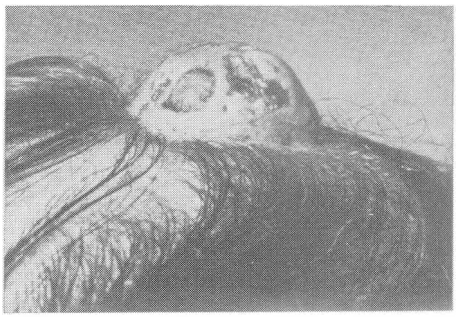


FIG. 90.—Cock's 'peculiar tumour' of the scalp.

(iii) *Sebaceous Horn.*—The contents of a cyst sometimes escape slowly from the duct orifice and dry in successive layers on the skin, forming a sebaceous horn (fig. 91 and see frontispiece).

Treatment.—(*a*) Incision–Avulsion.—Under local anæsthesia an incision is made through the skin, into the cyst. The contents are squeezed out and the cyst wall is seized with artery forceps and carefully avulsed.

(*b*) Dissection is necessary for cysts which have been previously inflamed. An incision is made over the cyst, the wall is defined, and the cyst is dissected from adjacent tissue and removed intact. Unless the wall is completely removed recurrence is probable.

FIG. 91.—A recent example of sebaceous horn of the scalp.

Sequestration and **Implantation Dermoid Cysts** are described on p. 63.

PAPILLOMAS AND MISCELLANEOUS SKIN LESIONS

Papilloma

A papilloma of the skin may be derived from either the squamous or the basal cell layers. The *squamous cell papillomas* include the following types:

(1) *Congenital* (*syn. Nævus Verrucosus*), which may be single or multiple, and it appears either at birth or in early life. It is a warty growth of brownish colour, but large horny excrescences may be present. (2) *Infective Wart* (*verruca vulgaris*)—the common wart—described on p. 112. (3) *Soft Papilloma*, which often occurs on the eyelids of elderly people. (4) *Keratin Horn* (fig. 92).—This is also seen in old people and is a papilloma with excess keratin formation.

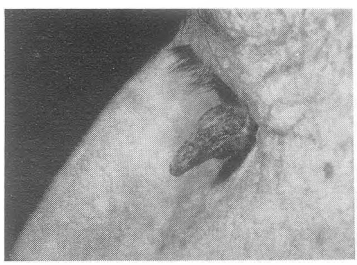


FIG. 92.—Keratin horn beside the eye.

Basal Cell (*Seborrhœic, or Senile, Wart*).—Often develops in large numbers on the trunk, face, and arms of persons in or past middle life. Circular, slightly raised, warty, often brownish, they look as if they are stuck on the skin (fig. 93).

Edward Cock, 1805–1892. Surgeon, Guy's Hospital, London.

Molluscum Fibrosum.—Soft fleshy skin tags which may be polypoid or filiform and which may occur in large numbers on the neck, trunk, and face in persons past middle age.

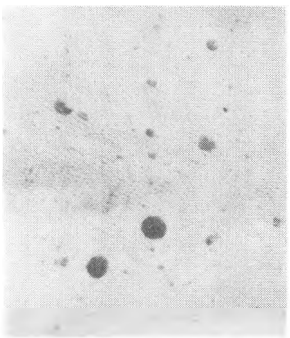


FIG. 93.—Basal cell seborrhœic warts.

Molluscum Sebaceum (*syn.* Keratoacanthoma).—Commonly occurs on the face or hand during the fifth and sixth decades. It presents as a firm hemispherical nodule, which may reach a size of up to 2 cm in diameter in six weeks (fig. 94). The summit of the tumour ulcerates and forms a crust beneath which is a crater. After a time, the crust is shed and spontaneous healing occurs. This may leave a scar. The tumour may be curetted and a caustic (e.g. carbolic or silver nitrate) applied to the crater. A quicker cure is obtained by excision, which permits adequate microscopy. Undoubtedly in the past many cases were regarded as epitheliomas and helped to swell the number of ' cures ' for this condition.

Sclerosing Angioma (*syn.* Dermatofibroma; Subepidermal Nodular Fibrosis).—This occurs in skin as firm, indolent, single, or multiple nodules. The nodules, in adults, are situated most commonly on the extremities. As a rule they are only a few millimetres in diameter. Most are reddish, others are yellowish-brown (due to lipid) or blue-black (due to hæmosiderin). The latter may resemble melanoma. *Dermatofibroma protuberans* grows persistently outwards to attain considerable size (fig. 95). It may be a low-grade fibrosarcoma.

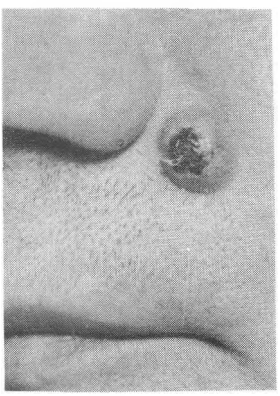


FIG. 94.—Molluscum sebaceum.

FIG. 95.—Dermatofibroma protuberans.
(*R. Naik M.S. Durg. India.*)

Granuloma Pyogenicum (fig. 96).—This is a traditional but misleading name. The lesion looks like a hæmangioma but has a typical natural history. Usually single, it consists of a dull red, soft or moderately firm, more or less pedunculated nodule which grows rapidly to a size varying from 0·5 to 2 cm in diameter. The surface shows atrophic epidermis, but often has crusts. It may occur anywhere on the skin, but most commonly it is seen on the face, the fingers and toes. Treatment is excision.

Benign Calcifying Epithelioma (Malherbe's epithelioma). — These are solitary, hard, tumours that appear to be growing from the deep surface of the skin. They occur mostly on the face, neck and arms. The size usually varies between 0·5 to 3 cm in diameter. The tumour may arise at any age but is common in childhood. Clinically it resembles a rather hard sebaceous cyst.

FIG. 96.—Granuloma pyogenicum.

Tumours of Accessory Skin Structures. —These are rare. (1) *Sebaceous nævus:* A congenital condition presenting as a pinkish-yellow plaque in the scalp. (2) *Sebaceous adenoma:* Isolated lesions are very rare. Multiple sebaceous adenomata occur in association with epilepsy and mental deterioration. (3) *Tumours of sweat glands:* Are closely related to the basal cell carcinomata but present deep to the skin and are often cystic (Papilliferous cystadenoma).

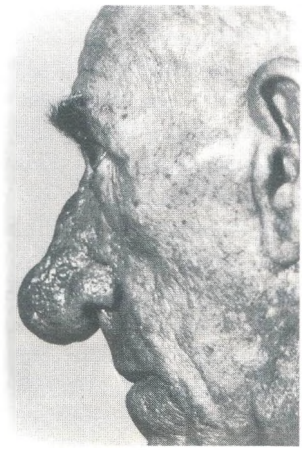


FIG. 97.—Rhinophyma.

Sarcoid (Boecks) (p. 196).—This is a generalised disease which may affect skin. In the skin it occurs as reddish-brown nodules which are soft, and rarely ulcerate. Giant cells are found, but tubercle bacilli can never be isolated.

Rhinophyma (*syn.* Potato nose) (fig. 97).—This is a glandular form of Acne Rosacea. The skin of the nose, particularly the distal part, becomes immensely thickened and the openings of the sebaceous follicles are easily seen. The capillaries become dilated and the nose assumes a bluish-red colour. Surgical treatment, by paring away the excess tissue, gives a great improvement.

Cutaneous Horns.[1] — May be (i) Sebaceous horns, (ii) wart or corn horns, (iii) cicatrix horns, and (iv) nail horns (frontispiece, figs. 91, 92, 98, 99).

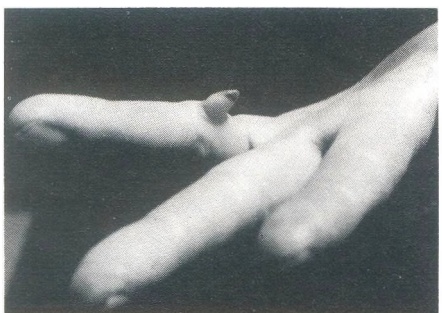


FIG. 98.—Horn of a corn. (*Dr. Sheetal Singh, Srinagar, Kashmir, India.*)

FIG. 99.—A cicatrix horn in a burn scar. (*R. R. Deskmukh, M.S., Dhautoli, Nagpur, India.*)

[1] Sir John Bland-Sutton, 1855–1936. Surgeon, The Middlesex Hospital, London, and President of the Royal College of Surgeons, classified horns in 1911.

Albert Malherbe, 1845–1915. Professor of Pathological Anatomy and Histology, Nantes, France.

HÆMANGIOMA

A hæmangioma is a developmental malformation of blood-vessels rather than a true tumour, and is therefore an example of a hamartoma (p. 58). It may occur in any tissue of the body, but is most common in the skin and subcutaneous tissues. A hæmangioma is either capillary, venous (cavernous), or arterial in type.

Capillary Hæmangioma

(1) **Salmon Patch.**[1]—This is present at birth over the forehead in the midline, and over the occiput. It disappears by the age of one year.

(2) **Port Wine Stain** (Nævus flammeus) (fig. 100).—Present at birth, it changes very little throughout life, although the colour may alter a little and it may become nodular in some areas. *Treatment* is for reason of appearance. The texture of the skin is quite normal and in a girl the blemish can be disguised by the skilful use of cosmetics. In a boy, treatment by excision and grafting may be considered. Radiation and other destructive treatment is disappointing.

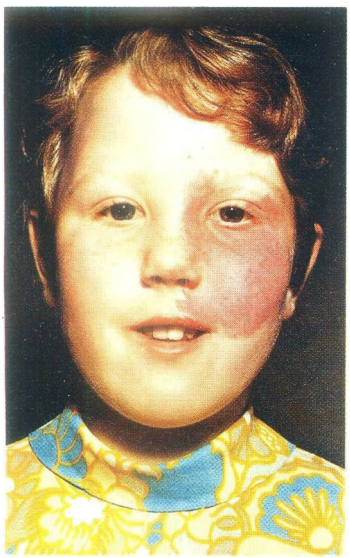


FIG 100.—Port wine stain.

(3) **Strawberry Angioma** (fig. 101).—This common condition has a typical history. The baby is normal at birth and at the age of one to three weeks is noted to have a red mark. This increases rapidly for some weeks or even up to three months, until the typical strawberry or raspberry-like swelling is present. Clinically, the sign of emptying may be demonstrable. The lesion is composed of immature vaso-formative tissue. The subcutaneous tissue as well as the skin is often involved, and in severe cases the muscles may be affected. Sub-mucous nævi are prone to hæmorrhage, which is sometimes alarming.

From the age of three months to one year the nævus grows with the child. Then it ceases to grow. Eventually the colour fades and flattening occurs so that at the age of seven or eight years involution is complete. *Treatment.*—The final result (fig. 102) is better if natural involution is allowed to occur rather than if regression is hastened (as it can be) by any operative or physical methods (e.g. carbon dioxide snow, injection of hot water or sclerosant fluid or hypertonic saline, etc.). X-ray therapy carries a high risk of disturbance of growth and is dangerous.

Venous Angioma (**cavernous**) is relatively uncommon. It is present at birth and consists of a multiplicity of venous channels of varying calibre. Usually it shows no tendency to involution and may become larger and

[1] Also called 'stork bites'! Not to be confused with the patch in syphilis.

more troublesome later. Sometimes the whole of one limb and the adjacent part of the trunk is affected. Occasionally the nævus is associated with a lipoma (nævo-lipoma). In some cases arteriovenous communications are

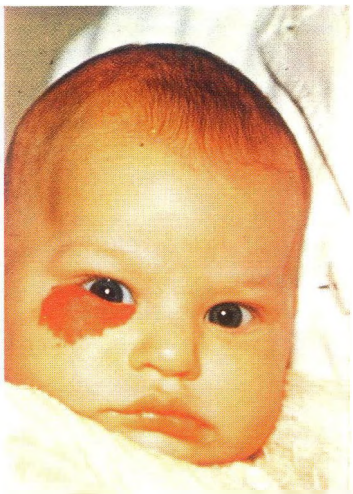


FIG. 101.—Strawberry angioma.

FIG. 102.—Strawberry angioma. Same case as fig. 101 with conservative treatment, about 7 years.

present (p. 152). The skin overlying the nævus may be atrophic, and besides being in danger of severe hæmorrhage from trauma, the patient may suffer from septicæmia if organisms gain entry, in which case the energetic use of antibiotics is an urgent matter. *Treatment.*—In general this is conservative. The repeated injection of hot water and other sclerosing agents may help, but the method is uncertain. If arteriography reveals a feeding artery, therapeutic embolisation (p. 157) may be possible.

Arterial (plexiform) Angioma is a type of congenital arteriovenous fistula (p. 152). The pulsating swelling of arteries and arterialised veins is often called a *cirsoid aneurysm* (Chap. 26).

Spider Nævus.—This may be associated with liver disease (Chap. 45) if present in the skin over the manubrium sterni, but it may occur quite innocently. It shows the characteristic sign of emptying.

Nævus tardes are small angiomas occurring *in adults*, often around the mouth. They may be associated with vasospastic conditions and scleroderma (p. 164).

Lymphangiomas are described on p. 189. Sometimes they are associated with a hæmangioma (hæmo-lymphangioma).

PREMALIGNANT CONDITIONS OF THE SKIN[1]

(1) **Bowen's disease** is an intradermal precancerous condition. A brownish induration with a well-defined edge appears in the skin (fig. 103). Microscopically

[1] *Acanthosis Nigricans*, a pigmented hypertrophic condition of the skin folds usually in the axillæ *is not* in itself premalignant, but is associated with cancer of the gastro-intestinal tract and the breast.

John Templeton Bowen, 1857–1940. Professor of Dermatology, Harvard Medical School, Boston, Massachusetts, U.S.A. Described Precancerous Dermatosis in 1912.

large clear cells similar to those found in Paget's disease of the nipple (Chap. 37) are in evidence. Sooner or later carcinoma develops, and wide excision is then necessary.

(2) **Paget's Disease of the Nipple** (Chap. 37).

(3) **Leukoplakia** (Chap. 32).

(4) **Senile (or Solar) Keratosis.**—Occurs occasionally as multiple lesions on the face and backs of hands in persons past middle life. Exposure to sun is the important predisposing factor. Usually the lesions are less than 1 cm and have a dry, hard scale with little or no infiltration. Squamous cell carcinoma may develop.

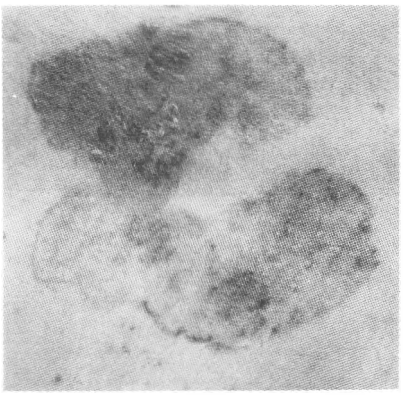


FIG. 103.—Bowen's disease of the abdominal wall.

(5) **Radiodermatitis.**—(*a*) *Early:* Erythema occurs which goes on to desquamation and pigmentation. If the dose is very great, ulceration may occur. (*b*) *Late:* Atrophy, irregular hyper-pigmentation and telangiectasis and hair loss occur. Eventually, squamous cell carcinoma may develop.

(6) **Chronic Scars** (Marjolin's Ulcer).—A carcinoma which develops in a scar presents the following characteristics:

(i) It grows slowly, as the scar is relatively avascular.

(ii) It is painless, as scar tissue contains no nerves.

(iii) Secondary deposits do not occur in the regional lymph nodes, as lymphatic vessels have been destroyed. If the ulcer invades normal tissue surrounding the scar, it extends at a normal rate, and lymph nodes are then liable to be involved.

MALIGNANT DISEASE OF THE SKIN

Basal Cell Carcinoma (Rodent Ulcer).—A common tumour of low-grade malignancy which occurs in white-skinned people. Exposure to sunlight is a predisposing factor and it is therefore common in Australia. It occurs in middle or late age and ninety per cent of lesions are found on the face, usually above a line from the lobe of the ear to the corner of the mouth, the commonest site being around the inner canthus of the eye. Although often called a rodent 'ulcer' many of the lesions are non-ulcerated, and have a nodular appearance with a pearly or darkly translucent colour as if containing water, and with a network of fiery red blood-vessels on the surface. Thus the common types are: nodular, cystic, and ulcerated (figs. 104 and 105). An unusual type is the 'field fire' rodent.

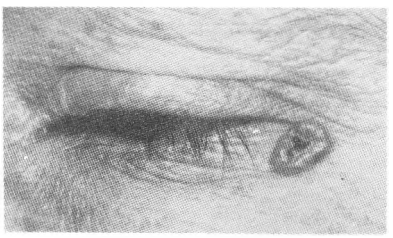


FIG. 104.—Rodent ulcer.

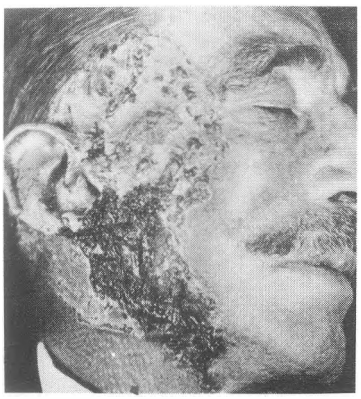


FIG. 105.—Disfigurement caused by rodent ulcer.

The ulcerated type has a typical appear-

Jean Nicolas Marjolin, 1780–1850. Surgeon, Paris. Described the development of Carcinomatous Ulcers in Scars in 1828.

ance—a raised rolled edge, like a motor car tyre, with central ulceration (fig. 104). Temporary healing often takes place, to be followed by further ulceration with serous discharge and bleeding. The patient gives a history of a 'spot' which never really heals: 'it keeps scabbing over and breaking down'.

Though the tumours are slow-growing, if ulcerated they will, as the name implies, gradually erode deeper tissues, such as muscles, cartilage, and bone, producing severe disfigurement (fig. 105). Dissemination by lymphatics or the blood-stream does not occur. Rarely the ulcer takes on a squamous-cell carcinomatous change.

Microscopic examination shows masses of darkly staining cells with a characteristic arrangement—an outer palisade layer of columnar cells surrounding a central mass of polyhedral cells. Cystic spaces may be seen.

Treatment. — Surgery or radiotherapy is necessary. Excision allows the whole lesion to be examined. With small lesions the operation can be described as an excision-biopsy. It is essential to excise a margin of healthy tissue around and beneath the tumour. A skin graft (p. 131) may be needed to cover the defect. In large lesions some form of plastic repair by a pedicle-flap graft may be required—cosmetic results should be good (fig. 120).

FIG. 106.—Squamous-celled carcinoma. The patient was a chemical worker.

Cylindroma (*syn.* 'turban' tumour) is so called from the arrangement of the stroma in peculiar transparent cylinders. It is considered by some to be a basal-celled carcinoma, while others classify the tumour as endothelioma (p. 60). The tumour gradually forms an extensive turban-like swelling extending over the scalp. Ulceration is uncommon, and the tumour is relatively benign.

Squamous Cell Carcinoma (Squamous Cell Epithelioma, Epithelioma) (fig. 106).—This is less common, but it is more malignant and more rapidly growing than rodent ulcer. A squamous cell epithelioma can occur *de novo* in the skin of the face of elderly people, but more often it occurs in a pre-existing skin lesion, or as a result of past irradiation. The pre-malignant conditions are listed above, and other conditions include long-standing venous ulcers, chronic lupus vulgaris lesions, and the prolonged irritation of the skin by various chemicals, e.g. dyes, tar or soot (p. 50).

A typical carcinomatous ulcer is irregular in outline, and the edges are raised and everted. The base is indurated and sooner or later becomes attached to the deeper structures. A blood-stained discharge occurs, which is increased in amount with the advent of secondary infection. The regional lymph nodes become involved, and the deposits are liable to undergo mucoid degeneration, to which secondary infection is sometimes added.

Treatment.—Treatment must be adequate. Immediately after biopsy

confirmation of the diagnosis, the surgical treatment is wide excision, which may require some form of skin graft to the defect. Whether radiotherapy should be employed instead of surgery depends upon the condition of the patient, the size and attachments of the tumour, and the radiotherapy facilities.

The presence, or subsequent appearance, of enlarged mobile lymph nodes is an indication for block-dissection, unless it is considered that secondary infection is responsible, in which case the decision to operate will await the effect of the appropriate antibiotic. Fixed enlarged lymph nodes are not removable; some regression may be obtained by radiotherapy.

MELANOCYTIC TUMOURS AND MALIGNANT MELANOMA

Simple Melanocytic Tumours

The melanocyte is generally believed to be derived from the neural crest. In normal skin, melanocytes appear as clear cells in the basal layer of the epidermis. They may become increased in number in the layers of the skin to form benign pigmented nævi (moles) as follows: 1. within the basal layer of the epidermis (*Lentigo*), 2. as localised aggregations projecting into the dermis (*Junctional nævus*), 3. entirely within the dermis (*Dermal nævus*) and 4. when 2 and 3 are combined (*Compound nævus*). These simple melanocytic tumours may arise anywhere in the skin including the nail bed (fig. 110). They are seen occasionally in the conjunctiva of the eye but are rare in other mucous membranes.

Clinical Features.—Pigmented nævi may occur at any age but commonly appear in childhood and adolescence as small, brown, flat or slightly raised lesions in the skin. These are usually of junctional or compound type but with increasing age they either atrophy or mature into dermal nævi.

Another clinical type is seen in middle-aged patients, particularly in the face. These appear as small, rounded swellings, often non-pigmented and sometimes bear bristly hairs. They are dermal nævi.

Congenital nævi are rare (fig. 107). They are often darkly pigmented and may be hairy and papillary in appearance. Occasionally they cover extensive areas of skin, up to 25% or more of the body surface. They may undergo malignant change even during childhood. With this exception malignant melanoma is very rare before puberty.

FIG. 107.—Congenital melanoma.

Treatment may be indicated (i) for cosmetic reasons, (ii) if by reason of its position the lesion is subject to the nuisance of repeated trauma, e.g. cut when shaving or rubbed by the brassiere strap, though there is no evidence that trauma causes malignant change, (iii) prophylactically in sites where there is a predisposition to malignant change, e.g. the palms, soles or external genitalia, or (iv) if the history suggests that malignant change has occurred (see below).

Surgical excision is the only acceptable treatment, and all lesions should be sent for histological examination.

MALIGNANT MELANOMA

It is now generally agreed that malignant melanoma of the skin should be regarded as a carcinoma—melanocarcinoma. A malignant melanoma may arise (i) in a pre-existing pigmented nævus or (ii) *de novo* in apparently normal skin. Pathological studies suggest that malignant change in a benign nævus occurs only in the junctional or compound varieties; the pure dermal nævus is safe. Little is known of the causation but it is commonest in fair skinned individuals who live in hot climates and prolonged exposure to ultra violet light has been incriminated.

FIG. 108.—Malignant melanoma of the leg, showing pigmented and amelanotic areas.

Clinical Recognition.—Malignant melanoma is almost unknown before puberty. It should be suspected after puberty when 1. a previously existing mole begins to enlarge, itches, weeps, scabs or bleeds, becomes ulcerated or more deeply pigmented, or produces a halo of pigment in the surrounding skin. 2. A pigmented lesion appears in an adult and grows progressively. 3. A rapidly growing, fleshy, ulcerated skin tumour appears which looks as though it may be malignant; some malignant melanomas are amelanotic, so that this may be an amelanotic melanoma. The commonest site in females is the lower leg (fig. 108), and in males the front or back of the trunk. In the African Negro the sole of the foot is the most frequent site; malignant melanoma does not arise from the blacker skin of the remainder of the body for reasons which are not understood.

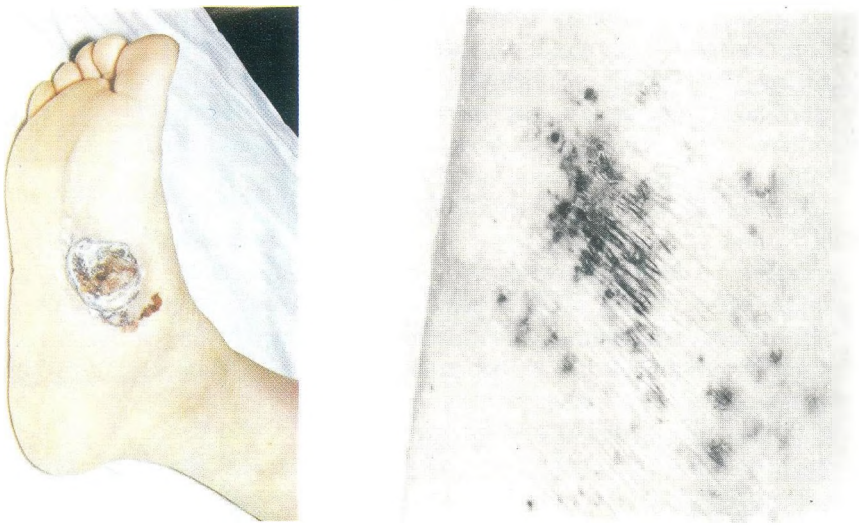
Differential diagnosis is from (i) histiocytoma (sclerosing angioma), (ii) pigmented basal cell carcinoma, (iii) basal cell papilloma (seborrhœic wart), (iv) cavernous hæmangioma.

Spread.—Malignant melanoma may spread by local extension, by the lymphatics or by the blood-stream. Tumour cells may reach the regional lymph nodes by embolism but spread by lymphatic permeation is also seen producing local satellite nodules (fig. 109(*a*)) and/or 'in transit' deposits between the primary growth and the regional nodes (fig. 109(*b*)). Secondary lymphœdema may occur (fig. 183). Blood borne metastases are seen in the lungs, liver, brain, bones and skin. They may also involve unusual sites, e.g. the breasts, small intestine and heart. Secondary deposits are typically black (fig. 111) but sometimes contain little or no melanin, even when the primary tumour is heavily pigmented. Extensive visceral involvement may cause melanuria.

The Choroid.—Malignant melanoma may arise in the choroid of the eye where it presents with blurring of vision. It spreads by the blood-stream, frequently giving rise to visceral deposits which can be enormous, especially in the liver. These metastases may not appear clinically for many years after the removal of the eye.

Treatment.—Surgical excision is the only certain method of cure. Malignant melanoma of the skin is only rarely radio-sensitive. Excision of

skin, subcutaneous fat and sometimes deep fascia must be wide with at least 5 cm clearance all round the tumour where anatomically possible. The large resulting defect almost always requires skin grafting.



(*a*) (*b*)

FIG. 109.—(*a*) Melanoma malignum of the foot, with local satellite deposits. (*b*) 'In transit' deposits between the primary growth and the regional nodes.

When the clinical diagnosis is in doubt local excision biopsy with a narrow margin of clearance is done and either frozen or rapid paraffin sections examined. This is safe provided the wide clearance described is performed within 48 hours of biopsy.

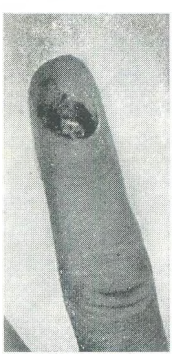


FIG. 110.—Subungual malignant melanoma

Moles must never be cauterised or curetted because the vital histological evidence is destroyed and if the lesion is malignant the disease may be disseminated.

A proven subungual malignant melanoma (fig. 110) requires amputation of the affected digit.

Malignant melanoma of the choroid is treated by enucleation of the eye.

Malignant melanoma may develop in the conjunctiva but unlike that of skin can sometimes be successfully treated by careful radiotherapy.

Malignant melanomas arising in other mucous membranes, e.g. mouth (fig. 601) or anal canal are by contrast highly dangerous and seldom curable.

Management of Lymph Nodes.—If the primary tumour is adjacent to the lymphatic field, i.e. on the face or neck, or near the axilla or groin, it is widely excised in continuity with the regional nodes so as to include all the intervening lymphatic pathways. Excision of lymph nodes must be radical, e.g. the axilla is cleared in the same meticulous manner as for breast carcinoma,

and a groin dissection includes not only the inguinal nodes but also those in relation to the three iliac vessels.

If the primary tumour is remote from the lymphatic field, e.g. ankle region, it is dealt with as already described. If the regional nodes are not clinically involved, prophylactic block dissection is not carried out but the patient is carefully observed. Only if or when the nodes are enlarged are they dissected as a separate operation (fig. 111).

FIG. 111.—A malignant melanoma. Block dissection of lymph nodes in the groin.

Other Methods of Treatment.—Cytotoxic agents are only of limited value when the disease is disseminated. They do however help in control of locally advanced disease in the limbs if administered by the specialised technique of isolated perfusion, where high local concentrations of the drug can be produced without generalised side effects. In this way major amputations are often avoided. Immunotherapy has been tried but its value is not yet established.

Prognosis.—The prognosis of cutaneous malignant melanoma depends mainly on the stage at which it is detected and to a lesser degree on the site of the tumour. Small, flat lesions are commonly curable by adequate primary treatment. Larger lesions which are raised and ulcerated, with accompanying tumour penetration into the deeper layers of the skin, carry a much higher mortality. Satellite and ' in transit ' nodules or involvement of the regional lymph nodes are also bad prognostic signs. The importance of early recognition of this disease is therefore crucial. Melanomas of the trunk are less favourable than those arising on the limbs or face. The out-look is better in the female than the male though the disease is not influenced by hormone therapy. Pregnancy makes pigmented moles darker and sometimes larger but does not affect the course of malignant melanoma.

Hutchinson's melanotic freckle is an irregularly shaped flat pigmented lesion found most commonly in sun damaged skin of the cheek or temple in elderly people. Very occasionally it can appear in other exposed areas e.g. the dorsum of the hand. It enlarges slowly over the years and may show spontaneous regression in some areas while extending in others. Malignant change can occur after periods of the order of ten years or more, and is recognised by the formation of a nodule or nodules. This form of malignant melanoma (lentigo-maligna melanoma) is relatively less aggressive than the others and less frequently metastasises to the regional lymph nodes or systemically. It is treated by excision which does not require such a wide margin of healthy tissue as in the more usual types of the disease.

Kaposi's Sarcoma may rarely be confused with malignant melanoma. It occurs mainly in Italians and in Eastern European Jews. It is also prevalent in equatorial Africa. It principally involves males of middle age or above. Multiple bluish nodules occur especially on the limbs. In due course the nodules ulcerate and late on the disease may disseminate widely. These tumours are sensitive to radiotherapy and chemotherapy.

Sir Jonathan Hutchinson, 1828–1913. Surgeon, The London Hospital. He described the melanotic freckle in 1892.
Moritz Kaposi, 1837–1902. Professor of Dermatology, Vienna. Described Pigmented Sarcoma of the skin in 1872.

BURNS AND SCALDS

The majority of burns are caused by heat. A scald is a burn caused by moist heat. Other causes include actinic rays (sunburn), irradiation, chemicals, electricity and friction. A burn is a wound in which there is coagulative necrosis of the tissues. In this respect it will be appreciated that cold (e.g., frostbite) also causes coagulative necrosis and that the word ' coldburn ' is not really a contradiction in terms.

The management of a patient with a heat burn depends upon the consideration of:

1. **Shock.**—The area of the body involved is important in relation to the development of shock (p. 82). The surface area is estimated by Wallace's ' Rule of Nine ' (fig. 112). It should be remembered that this rule does not apply strictly to infants and children. Children, compared to adults, incur greater fluid losses as they have a higher ratio of surface to body area.

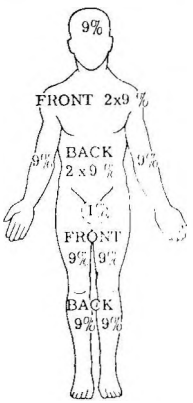


FIG. 112.— ' Rule of nine ' for estimating the extent of a burn. By adding the affected areas together the percentage of the total body surface burnt can be calculated quickly. Remember that infants have a greater percentage of head and neck surface area (18 per cent) and a smaller leg surface area (9 per cent) than adults.

Burn shock is contributed to by 1. the overwhelming sudden intensity of pain, but is mostly caused by 2. the loss of circulating blood volume (= hypovolæmia) in the first 24 hours through water, electrolyte and plasma protein loss in blister fluid, exudate, œdema and by evaporation. This is due to vasodilatation and increased capillary permeability of the wound, probably mediated by the liberation of histamine, kinins, prostaglandins and fibrin degradation products. Heat loss from a burned area may be considerable.

2. **Healing.**—The depth of a burn determines the manner of healing. Two main grades are considered: (*a*) *partial skin thickness burns* (fig. 113) and (*b*) *a full thickness burn.*

(*a*) *A partial thickness burn* will heal from the epithelial elements in the deeper part of the skin (hair follicles, sebaceous and sweat glands).

(*b*) *In full thickness burns* all the epithelial elements have been destroyed and the dead skin (slough) will separate in two or three weeks revealing a red granulating surface. This can only heal from the edge very slowly and with the risk of contracture (fig. 20). This type of burn requires skin grafting either after the slough has separated or, better still, by excision and skin grafting 4–6 days post-burn.

Assessing the depth of burn.—In a partial thickness burn the nerve endings are still viable, so the burn wound is sensitive. A deep burn is insensitive. The simplest method of assessment is the use of a sterile needle with which the clinician can distinguish between the sensitive and the insensitive areas.

The Deep Dermal Burn is a borderline burn, between a partial and a full

Alexander Burns Wallace, 1906–1974. Formerly Reader in Plastic Surgery, University of Edinburgh.

thickness burn, in which a few epithelial elements remain. This responds better to tangential excision (below) than to exposure treatment.

3. **Scarring.**—Partial thickness burns usually heal without much scarring. Alteration in pigmentation is quite common. Full thickness burns tend to cause unsightly incapacitating scars and contractures. To some extent these can be minimised by properly timed skin grafting and the reduction of sepsis. In late contractures it may be necessary to excise the scar completely and to replace it with a full thickness skin graft (p. 132).

Compression.—It has been discovered that the disfigurement caused by hypertrophic post-burn scars can be minimised by the wearing of elasticated compression garments (p. 24).

A Treatment Programme for Burns

The sections that follow include first aid, a treatment programme for burns shock, the local treatment of the wound and the general treatment. The section on skin grafting follows thereafter.

First Aid should include:—Immersion in cold water, preferably running water for three minutes, is especially suitable for burns of hands, arms and legs. Pain, exudation and shock are thereby reduced. Charred clothing that is close to, or adherent to, the skin is not removed, it is likely to have been sterilised by the heat. The area is covered by a piece of clean material, including any handy polythene film and aluminium foil. Clean plastic bags are suitable for hands and feet. Pain relief includes the use of pethidine or morphine, but not for infants and young children. Large doses are avoided in order not to stimulate the vomiting centre, and for the same reason oral fluids are given in limited quantities. Should there be any delay in getting the patient to hospital, steps should be taken where possible to prevent the second accident (footnote p. 6) by making an estimate of the degree of burn and procuring the means of instituting an intravenous infusion. Prevent undue cooling of the whole body. Reassure the patient at all times.

Chemical burns require thorough douching and irrigation with water. Vinegar or acetic acid is applied to alkali burns, and sodium bicarbonate solution (teaspoonful to a pint of water) to acid burns. Constant wetting of phosphorous burns is necessary until the arrival of a 2 per cent solution of copper sulphate and then sodium bicarbonate.

Treatment of Burns Shock (A treatment programme)

(1) *Estimate percentage of surface burns.*—This is done using the 'Rule of Nine' (fig. 112).

(2) *Estimate weight* (in kg) from (*a*) known weight or (*b*) measured weight or (*c*) age and height tables.

(3) *Transfusion with Dextran* 110 (*in saline*), *or plasma* (preferably plasma protein fraction P.P.F.) (p. 81).—(*a*) All adults with burns over 15 per cent. (*b*) All children with burns over 10 per cent. (Some children with 5 to 10 per cent surface burns need careful watching.)

(4) *Method of intravenous administration.*—In a shocked patient with collapsed veins, do not waste time trying to put up a drip with needles—do a cut-down immediately. Avoid the use of veins in the legs if at all possible (use forearm, arm, or external jugular veins) (p. 96).

(5) *Volume to be transfused.*—The plan is to give six equal 'rations' of Dextran or plasma in six periods. The volume is calculated by the following formula (after Muir and Barclay).

$$\text{One 'ration' (in ml)} = \frac{\text{Total per cent of burn} \times \text{weight (kg)}}{2}$$

Three rations are given in the first twelve hours, two rations are given in the second twelve hours, leaving the last ration for the third twelve hours.

Dextrose i.v. solutions (p. 109) are used for nutritional requirements.

Examples.—An 80 kg patient with a 30 per cent burn needs a ration of 1,200 ml and therefore 3,600 ml in the first 12 hours, 2,400 ml in the second, and 1,200 ml in the third period. If 70 kg with a 40 per cent burn the volumes will be 4,200, 2,800, and 1,400 ml. If 60 kg with a 50 per cent burn, 4,500, 3,000, and then 1,500 ml will be required.

Treatment in the first 24 hours by electrolyte infusion only.—Adequate early infusion with Ringer lactate solution without any added colloid, but with added dextrose i.v. solution for nutritional requirements, is being claimed as a suitable alternative to the above. Larger volumes are required to a total of about 3 ml/kg/per cent burn, and for children nearer 4 ml/kg/per cent. Colloid in smaller amounts is given in the second 24 hours, and intravascular overload must be watched for.

Examples (compared with above).—The 80 kg patient with a 30 per cent burn requires 8,200 ml (6,000 ml) in the first 24 hours. If 70 kg with a 40 per cent burn, 8,400 ml (7,000 ml), and if 60 kg with a 50 per cent burn, 9,000 ml (7,500 ml).

(6) *Progress and control.*—The fluid requirements of the patient are adjusted mainly by clinical and to a lesser extent by laboratory observations. These include pulse rate and blood pressure, urine volume, colour of mucous membranes and finger tips, nausea and vomiting or gastric aspirate, hæmatocrit (p. 72) and CVP. If the urine output is poor (see below) a CVP catheter is inserted if not already in position, and the rate of infusion is increased until the CVP is 10–15 cm saline depending upon the zero reference point (p. 73).

(7) *Morphia.*—10 to 20 mg for adults is given intravenously or into the drip as indicated, bearing in mind that morphia stimulates the vomiting centre.

(8) *Urine.*—(*a*) Urine is collected hourly, measured and tested. An output of 30–50 ml/hour is necessary in an adult. A child should be infused to attain 1–1·5 ml/kg/hour.

(9) *Oral fluids.*—Water is required for metabolic fluid requirements. Start with 60 ml/hour and proceed to 100 ml/hour if there is no nausea. A nasogastric tube is usually passed in burns of 35 per cent or over, and the stomach aspirated hourly before giving the oral fluid.

(10) *Blood transfusion.*—In deep burns, as opposed to superficial burns, red cells are destroyed. Blood is given instead of the second ration (above) where the deep burn is 10–25 per cent, and in place of the second and the

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Thomas Laird Barclay, Contemporary. Plastic Surgeon, Bradford Royal Infirmary, Bradford.

sixth rations if the deep burn is 25–50 per cent of the skin area. Blood may also be required if escharotomy (below) or burn excision are performed. It is also given for post-burn anæmia.

Local Treatment

The aim of local treatment is to achieve skin cover as soon as possible (page 1), and to prevent infection meantime. Quick healing minimises scarring, deformity and loss of function.

In minor burns local treatment can be started immediately. If the patient has to be transfused, local treatment should be delayed until it is certain that the circulation is stable (the burn is covered with a sterile towel).

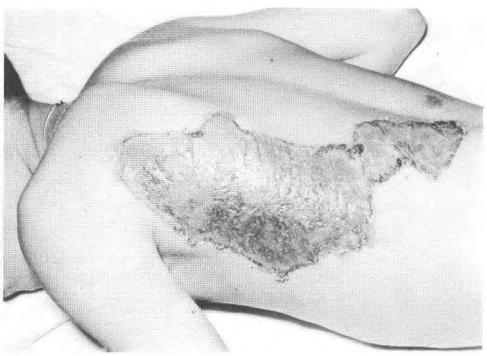


FIG. 113.—Partial thickness burn. Exposure treatment. *(I. F. K. Muir, F.R.C.S., Aberdeen.)*

(*a*) *Exposure treatment:* The burn is cleaned with cetramide-chlorhexidine solution, loose skin is removed, and blisters are pricked. The area is then exposed to the air preferably a laminar airflow in a room temperature of 75°F–80°F and low humidity, and a crust is allowed to form which protects the burn and permits healing to take place (fig. 113). This method is suitable for burns of the head and neck, and burns of a single surface of trunk or limbs. As the burn heals the crust lifts up at the edge and can then be snipped away.

(*b*) *Dressings:* The burn is cleaned as above and then dressed. The dressings are applied in three layers:

(i) First layer. The best available antiseptic cream is used (silver sulphadiazine 1 per cent). But, in order to reduce bacterial resistance to this type of sulphonamide, alternation with 0·5 per cent silver nitrate as a compress, or as a cream with 0·2 per cent chlorhexidene, is helpful. Mafenide, a methylated sulphonamide (10 per cent in a water miscible base), is another effective and non-toxic preparation, albeit painful to apply.

(ii) The second layer of cotton gauze.

(iii) An outer layer of absorbent wool is applied and held in place with gauze or crepe. This should be sufficiently thick to absorb all exudate without penetration to the surface and yet be porous. Should the wool become saturated, a further layer must then be applied without removing the original dressing. This dressing should be changed on the third and tenth days.

Plastic film and bags in the treatment of burns. In addition to the use of these in first aid, ' Opsite ' (see footnote p. 22) is a useful addition to the armamentarium of dressings if applied early in superficial burns (fig. 114). It excludes bacteria and, with moisture vapour permeability characteristics, provides the right environment for the regrowth of epithelium.

Plastic bags are particularly useful in the treatment of burnt hands and feet with antiseptic solutions, creams and powders—excluding bacteria and allowing free movement and maintenance of function.

Specific Local Treatment.—(1) *Partial thickness burns:* The treatment should be continued as above until the burn is healed.

(2) *Full thickness burn:* If a full thickness burn is present, the area of dead skin will be obvious as a black slough in two to three weeks. Treatment should therefore be changed to a method which will help separation of the slough. This can be achieved by baths and frequent dressings with hypochlorite solutions e.g. Eusol[1]. As soon as the slough has separated and clean granulations are present, thin split skin grafts cut from the undamaged areas should be applied to expedite healing.

FIG. 114.—A superficial burn of the back of the hand covered by ' Opsite '.

Excision and Skin Grafting of Burns.—In the knowledge that a burn is a wound which may become a serious entry of infection into the body if skin cover is not achieved quickly (p. 1), it is obvious that for deep burns, excision of the wound and skin grafting is the treatment of choice. Small deep burns can be excised immediately, but because of the overriding need for the treatment of shock the larger deep burns are usually excised in 20 per cent portions beginning about 4–6 days post-burn. In addition to the autografting of the patient's own split skin, allografts of lyophilised cadaver skin or xenografts of porcine skin are most useful in providing skin cover. Amnion (p. 65) is another type of skin cover, which stimulates granulation tissue to become a good base for autografting. These allografts and xenografts need to be inspected daily and they probably need changing every 2–3 days.

Tangential excision of a deep dermal burn.—Using a skin grafting knife, the burn is shaved in layers until it is judged that a viable surface has been reached immediately to be covered by a split thickness skin graft. The technique requires patience and practice. It is better to slice away more than is first thought necessary.

(3) **Special types of burn:**—(i) *Circumferential burns.* These cause such shrinkage of the skin as to create severe problems of general and local anoxia. If involving the neck or thorax there is serious embarrassment to respiration, and if the arms or legs be affected venous congestion or ischæmia and even gangrene can occur. Immediate release is necessary by making longitudinal incisions (escharotomy) down to healthy and yielding subcutaneous and subfascial layers.

(ii) *Pulmonary burns* must always be considered. Burns around the

[1] Eusol = *E*dinburgh *U*niversity *SOL*ution. It must be freshly prepared.

nostrils and lips do not necessarily presage burns of the trachea and bronchi, neither does an x-ray of chest help in diagnosis. Degrees of severity encountered include œdema of the glottis, bronchial spasm becoming so intense in some cases to be tantamount to ' status asthmaticus ', and pulmonary œdema. A definitive diagnosis is by fibre optic bronchoscopy and ^{133}Xenon lung scan, if available. Management depends upon good physiotherapy, breathing humidified air and oxygen, bronchodilator drugs and the culture of the sputum so that the appropriate antibiotics can be used as required. When necessary nasotracheal intubation is preferred to tracheotomy. Steroids are contraindicated unless used as a single bolus in a ' status asthmaticus ' situation.

(iii) *Electrical burns* (fig. 115) are localised and often very deep, and if the patient survives the electric shock, may need a complicated repair.

(iv) *Eye burns* (p. 524).

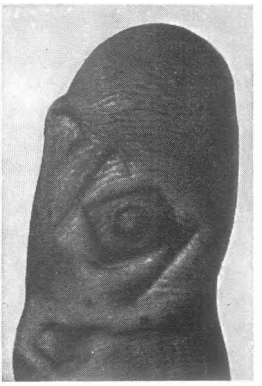


FIG. 115.—A post-mortem photograph of an electric burn. The deceased touched a live terminal.

General Treatment

The medical and nursing staff should take every precaution to prevent infection of a burn. It is desirable that the patient should be isolated (in a cubicle if possible), and barrier nursing instituted to prevent cross-infection. Special beds and slings are used for the exposure treatment.

(*a*) Respiratory damage (see above).

(*b*) Sedation to reduce pain and restlessness—morphia 10 to 20 mg may be given intravenously, usually via the drip. In children—morphia is best avoided but 0·5 mg for each year of age may be prescribed.

(*c*) Diet: Large amounts of protein are necessary to stimulate healing and to compensate for protein losses due to catabolism (Chap. 8).

(*d*) Antibiotics: Systemic antibiotics should be given as the clinical condition demands and in accordance with bacterial cultures.

(*e*) Late blood transfusion: Anæmia commonly develops and blood transfusion must be given if the Hb falls below 11·7 g/100 ml (g/dl).

(*f*) Immunisation: A reduction of immune status is believed to be a factor in the morbidity and mortality from burns. Therefore convalescent serum (passive immunity) or *Pseudomonas* vaccine (active immunity) injections may be tried.

SKIN GRAFTING

Skin grafting, by free grafts or pedicled flap, is necessary to obtain healing and limit deformity and disability when a large area has been lost by injury (e.g. burn), by disease (e.g. varicose ulcer), or by surgical excision of a neoplasm (e.g. malignant melanoma).

Free Skin Grafts.—These grafts provide the simplest method of restoring skin cover. They can be used when the raw surface is composed of healthy

vascularised tissue (e.g. subcutaneous tissue, deep fascia, paratenon), or clean granulations. Ischæmia and infection prevent grafts from surviving.

1. *Split thickness grafts* are the most generally useful grafts. Though often suitable for permanent repair they have a tendency to shrink. The grafts originally popularised by Thiersch were very thin, but the grafts now commonly used consist of one-third to two-thirds of the thickness of the skin. They are usually taken from the thigh by means of a special knife (Blair or Humby pattern, figs. 116, 117 and 118). The graft may be affixed by sutures. If a large area has to be covered (e.g. an extensive burn), the electric dermatome is useful. The Padgett dermatome, which necessitates the use of an adhesive, can be used to cut grafts from the abdomen or back. Alternatively, a large area can be covered by making a mesh of the graft in a special machine, the epithelium grows out from the strands of the mesh to cover the intervening bare areas.

FIG. 116.—A split skin graft, cut with a Blair knife. *(I. F. K. Muir, F.R.C.S., Aberdeen.)*

FIG. 117.—Humby's knife.

Or pieces of postage stamp size are applied.

The graft may be spread on paraffin tulle gras and laid or sutured on the area to be grafted. This will not be necessary if 'Opsite' (p. 22) is used for cutting the graft (fig. 118). Light pressure is then exerted by either wool soaked in flavine emulsion or a piece of sterilised sponge covered by a crepe bandage. The donor site of the split skin graft is covered with a single layer of tulle gras and heals in two weeks by epithelialisation from the remaining deeper parts of the skin.

FIG. 118.—Cutting a graft through 'Opsite'. This makes the graft easy to handle and sew into position with the film still attached.

2. *Full Thickness Skin Graft* (Wolfe).—This graft consists of the full thick-

Karl Thiersch, 1822–1895. Professor of Surgery, Erlangen (1854–1867), Leipzig, Germany (1867–1895). Described his method of Skin Grafting in 1874.
Vilray Papin Blair, 1871–1955. Professor of Clinical Surgery, Washington School of Medicine, St. Louis, Missouri, U.S.A.
Thomas Graham Humby, Contemporary. Formerly Plastic Surgeon, Barbados General Hospital, West Indies.
Earl Calvin Padgett, 1893–1946. Professor of Clinical Surgery, University of Kansas, Kansas, U.S.A.
John Reissberg Wolfe, 1824–1904. Ophthalmic Surgeon, Glasgow, Scotland. Described Full Thickness Skin Graft in 1885.

ness of the skin to which *no* fat should be adherent. The criteria for the graft to take are more strict than in the case of the split skin graft but the Wolfe graft shows no tendency to contract, and is of better colour and texture than the split skin graft. It is therefore useful for defects of the face (example:

(*A*) (*B*) (*C*)

FIG. 119.—A large rodent ulcer involving the upper lip, the side of the nose, and the antrum has been excised leaving the facial deficiency (A), in which post-operative biopsies show no residual growth. The upper lip and the side of the face are reconstructed by a flap graft (B), and the residual deficiency made good by a prosthesis, (C), which is held in place by a pair of spectacles. (*I. F. K. Muir, F.R.C.S., Aberdeen.*)

when excising a rodent ulcer from a lower eyelid), when the graft may be taken from the posterior surface of the ear.

Pedicle Flaps.—Free grafts will not ' take ' (i) on cartilage, open joints, bare cortical bone, or bare tendon, (ii) if the vitality of the recipient area has been depressed by scarring or irradiation, (iii) if the defect involves a cavity (example : full thickness excision of lip or cheek (fig. 119)). In any of these circumstances the repair must be by means of a flap of skin with some subcutaneous tissue, which remains attached to the body by one of its margins through which it retains a blood supply (i.e. by a pedicle or stalk) until it has united in its new position. Once it has attained its own blood supply the original attachment can be severed and the flap adjusted.

(*a*) *Local flaps:* These are turned on their own blood supply (fig. 119).

(*b*) *Direct flaps:* If the donor site can be brought into approximation with the recipient area, the flap can be transferred at one stage and the division completed at a second stage. Examples are from one finger to another (cross finger flap, fig. 220), from the calf of one leg to cover a defect on the opposite shin (cross leg flap, fig. 120), or from abdomen to hand.

FIG. 120.— A cross leg flap. The legs are held together by a plaster cast.

(*c*) *Indirect flap:* In some circumstances it may be impossible to bring the donor area and the recipient area into proximity and the flap must first be attached to an ' intermediate carrier site ', thus tissue can be moved from the abdomen to the face or to the lower leg, using the wrist as the ' carrier '. In order to eliminate raw areas it is convenient to stitch the flap into the form of a tube, i.e. a tube pedicle flap (fig. 121).

FIG. 121.—A tube pedicle flap. The skin tube was raised on the lateral abdominal wall. In the course of months the wrist was used as the intermediate source of its blood supply before final separation from the abdomen and application to the scarred and ulcerated leg.

(*d*) *Free Flaps.*—Using the microsurgical techniques for anastomosing small arteries and veins it is now possible to cover with a full thickness graft those areas (e.g. the chin and neck) where unsightly scars and contractures follow burns. Carried out in one stage this saves months of treatment with tube pedicle flap procedures. The most popular islands of skin and subcutaneous tissue with related vessels are the groin, scalp, dorsum of the foot, and the deltopectoral area.

CHAPTER 10

ARTERIES. GANGRENE

ARTERIAL SURGERY—BASIC CONSIDERATIONS AND PRINCIPLES

The Effects of Arterial Occlusion

When the arterial pathway to any part of the body is occluded, necrosis or gangrene will ensue unless there is an alternative pathway to bypass the circulation around the obstructed vessel. An alternative pathway is present for most organs and is known as the ' collateral circulation '. Provided the collateral circulation is adequate necrosis and gangrene will be avoided.

The effects of arterial occlusion depend principally on: (1) The anatomical arrangement of arteries; and (2) The speed of the occlusion. The various possibilities are shown in fig. 122. Anatomically the best collateral pathways are through muscles

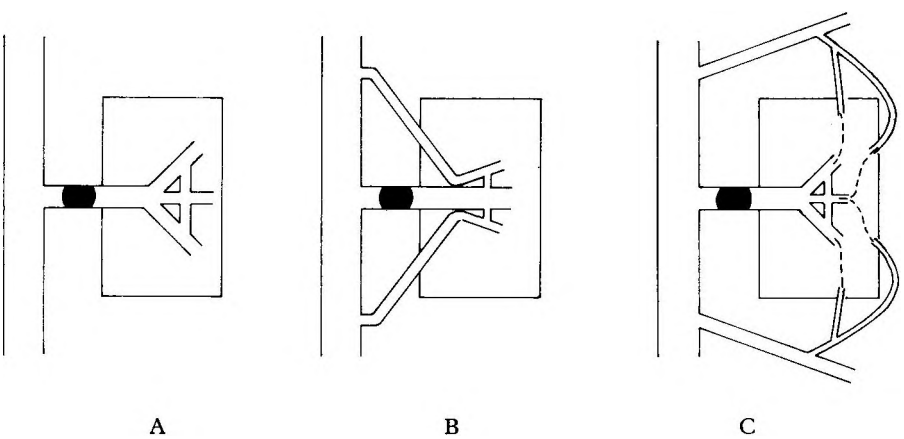


FIG. 122.—The effect of an arterial occlusion without and with a collateral circulation. A. Organ necrosis occurs with sudden or slow occlusion. B. Temporary ischæmia with sudden occlusion, no ischaemia with slow occlusion. C. Probable organ necrosis with sudden occlusion; but with slow occlusion the organ survives with greatly reduced reserve.

(fig. 123). Therefore collateral circulation around an iliac artery obstruction is good, whereas the absence of muscle around the knee joint results in poor collateral circulation around a popliteal artery occlusion.

Other factors which modify the effect of arterial occlusion include pump failure (coronary artery disease, etc.), and occlusion of collateral channels (e.g. by extension thrombosis, p. 154, or progressive atheroma).

Ischæmia

The word ischæmia means a lacking in blood flow. Commonly affected are, the heart (coronary thrombosis, angina pectoris), the lower extremities

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London. His collection of anatomical specimens forms the nucleus of the Hunterian Museum at the Royal College of Surgeons of England. Hunter was the first to demonstrate the formation of collateral circulation. He tied the external carotid artery of a stag from Richmond Park. The stag's antler fell off, but later another one grew in its place. On re-opening the original wound, Hunter found that while his ligature was intact, a collateral circulation had been established.

(intermittent claudication, gangrene), the brain (stroke), the kidneys (reno-vascular hypertension), and the intestine (mesenteric vascular accidents).

The following description applies to arterial disease of the lower limbs but the principles of management, may, suitably modified, be applied to similar arterial lesions affecting other organs.

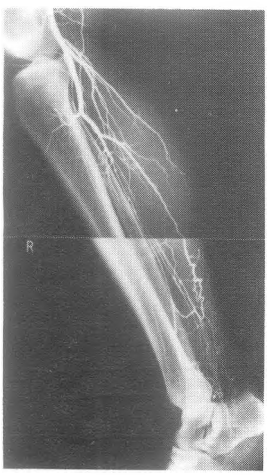


FIG. 123.—Arteriogram showing obstruction of the main arteries to the foot (anterior tibial, posterior tibial, peroneal), and the collateral circulation which has developed through the calf muscles in a patient with peripheral arteritis (p. 162).

Features of Ischæmia of the Legs

These include intermittent claudication, rest pain, coldness, numbness and paræsthesiæ, colour changes, ulceration and gangrene.

Intermittent Claudication.—The blood supply to the muscles, which, except in the most advanced cases, is adequate at rest, becomes inadequate during exercise. On walking a certain distance (claudication distance), the patient experiences a cramp-like pain, which causes him to stop. After a minute or two, perhaps while he looks into a shop window, the pain passes off, allowing him to go on, only to return once more after he has walked a similar or slightly shorter distance. The claudication distance depends on the available blood supply, which in turn depends on the site and extent of the arterial obstruction and the efficiency of the collateral circulation. The general health of the patient (heart failure, anæmia), the speed of walking, hills and wind and ambient temperature all affect claudication distance.

Intermittent claudication is commonest in the calf (femoro-popliteal obstruction) but may occur in the buttock and thigh (aorto-iliac obstruction).

Other causes of pain in the leg on walking must always be considered. To do this it is helpful to review all the anatomical components of the leg (bones and joints, veins, muscles and nerves). Thus pain may be due to arthritis, deep vein thrombosis, torn muscles, or that due to a prolapsed intervertebral disc (e.g. sciatica).

The pain of spinal nerve root origin, due to a prolapsed disc, may appear to come on with exercise, but it rarely disappears on rest. By the end of the day it becomes particularly tiresome. Answers to the questions ' Do you get the pain while watching television in the evening ? ' or ' Do you get the pain in bed, especially when turning over ? ' are usually in the affirmative. Neurospinal lesions and arterial occlusions sometimes coexist and make diagnosis difficult.

Rest Pain.—As arterial insufficiency in a leg becomes more severe, a time comes when the limb blood supply becomes inadequate at rest. This may occur gradually or may be precipitated by a local infection (paronychia, cellulitis).

Rest pain is felt in the foot and is severe, relentless and continuous. It

Claudius (Tiberius Claudius Drusus Nero Germanicus), 10 B.C.–A.D. 54. Emperor of Rome (A.D. 41–54). Was deformed by a disease contracted in childhood (possibly Poliomyelitis), and walked with a limp. Claudicare = to limp. Claudicatio = limping (Latin).

prevents sleep and dominates the patient's waking hours. A typical sufferer sits in bed, hip and knee flexed, clutching the painful foot with both hands, distracted from any normal activity. Some relief may be obtained by hanging the foot and leg down, and some patients sleep in a chair, or hang the foot over the side of the bed, or they get up many times at night and pace the bedroom. Analgesics usually are of no avail. Untreated, the patient becomes withdrawn and anorexic.

Rest pain should not be confused with acute night muscle cramps, probably venous in origin, and of doubtful relevance to arterial insufficiency.

Coldness, numbness and paræsthesiæ are common features of moderate as well as severe ischæmia.

(a) (b)

FIG. 124.—Colour changes with (a) elevation and (b) dependency (see text).

Colour changes (fig. 124).—Moderate or severely ischæmic limbs become blanched on elevation and develop a bluish-purple discolouration on dependency. Any bright red speckling is due to the extravasation of red cells through capillary walls. The angle of elevation at which blanching first occurs gives a good rough guide to the degree of ischæmia.

Ulceration and gangrene.—Ulceration occurs with severe arterial insufficiency and often presents as a painful superficial erosion between toes. Alternatively small, shallow, indolent non-healing ulcers may occur on the dorsum of the feet, on the shins, and especially around the malleoli. The blackened mummified skin and tissues of frank gangrene are unmistakeable to the observer (fig. 125), but it must be remembered that in acute ischæmia a dead white limb which becomes mottled means impending gangrene and not an improvement in the circulation. Fig. 126 shows a typical case. An account of gangrene is found on p. 166.

Pregangrene.—The combination of rest pain, colour changes, œdema, hyperæsthesia, with or without ischæmic ulceration, is frequently referred to as ' pregangrene '.

Appearance of the Ischæmic Limb

MILD	May be normal
MODERATE	Postural colour change pallor on elevation dusky cyanosis on dependency Loss of hair[1] Wasting Indolent ulcers
SEVERE	ACUTE ISCHÆMIA (fig. 126) Dead white pallor White with greyish purple blotching Gangrene
	CHRONIC ISCHÆMIA (fig. 125) Fixed discolouration—red or red purple Œdema Gangrene

FIG. 125.—Severe chronic ischæmia.

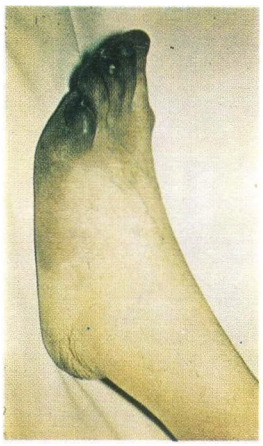


FIG. 126.—Severe acute ischæmia.

Temperature.—Severely ischæmic feet are usually cold but sometimes paradoxically may feel warmer than the non-involved foot.[2]

Sensation and movement.—Acutely ischæmic limbs are frequently paralytic and without sensation. These are ominous physical signs and such a limb has a poor prognosis in the absence of active treatment. Severe chronic ischæmia does not produce paralysis, but hyperæsthesia is common, especially in those areas of skin on the borderline of gangrene. Gentle handling of such limbs is essential.

[1] Loss of hair, a time honoured sign, is due to friction of socks and trousers far more often than to ischæmia.

[2] The acutely ischæmic limb tends to take on the temperature of the surroundings and may feel quite warm under the bedclothes.

Arterial pulsations.—Arterial pulsations below an occlusion in a main artery are usually absent or, in the presence of good collaterals, diminished.

In patients with vascular symptoms it is standard practice in each case to feel the pulsations in the radial arteries, the carotid arteries, the abdominal aorta, the femoral arteries in the groin, the popliteal arteries behind the knee, the posterior tibial arteries behind the medial malleoli and the dorsalis pedis artery on the dorsum of the foot. Diminution of a pulse can best be appreciated by comparing it with the pulse in the other limb, provided that this pulse is normal.

Expansile arterial pulsation with a mass may indicate an aneurysm (p. 148).

In arterial occlusion with a highly developed collateral circulation, or main artery stenosis, the distal pulses may be normal to palpation. The following physical sign is then useful:

The 'disappearing pulse'.—Where peripheral pulses are apparently normal, exercising the patient to the point of claudication may unmask the effect of an arterial obstruction, by causing the previously palpable pulse to disappear. After a minute or two following exercise the disappearing pulse reappears. Under these circumstances exercise produces vasodilation below the obstructing lesion. The arterial inflow, reduced by the lesion, cannot keep pace with the increasing vascular space, arterial pressure falls, and the pulse disappears.

Arterial bruits.—A vascular examination should include auscultation of the subclavian arteries in the supraclavicular fossæ, the carotid arteries behind the angle of the mandible, the abdominal aorta and the femoral arteries in the groin and over the adductor (subsartorial) canal. A systolic bruit over an artery is due to turbulence, and indicates a stenosis of the artery.

Systolic bruits are conducted distally. Thus a bruit at the angle of the mandible without any supraclavicular bruit frequently means a carotid artery stenosis. Where, however, a bruit is heard at both sites, its origin may be more proximal, e.g. aortic valve, aortic arch, brachiocephalic or subclavian arteries. Under these circumstances the carotid artery may prove quite normal. The patient with renal artery stenosis shown in fig. 127 had a bruit over the renal artery.

A Continuous Machinery Murmur over an artery usually indicates the presence of an arteriovenous fistula (p. 152).

Venous Refilling.—The limb should be elevated for thirty seconds and then laid flat on the bed. Normal refilling occurs within seconds. Reduced venous filling is often present in the severer forms of arterial insufficiency but is also common in vasospastic disease (p. 163) and in cold weather.

Harvey's Sign[1].—If the two index fingers are placed firmly side by side on a vein, and the finger nearer the heart is moved so as to empty a short length of vein, the release of the distal finger will allow the speed of venous refilling to be observed.

Increased venous return and even varicosities of the veins may be seen in arteriovenous fistulæ (p. 152).

Impotence from failure to achieve an erection is often a feature in male patients with an occlusion in the region of the bifurcation of the aorta and the internal iliac arteries (Leriche's syndrome).[2]

[1] William Harvey (1578–1657), Physician, St. Bartholomew's Hospital, London. First described circulation of the blood in his course of lectures at the Royal College of Physicians of London in 1616. Harvey's book *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus* was not however published until 1628. The sign of venous refilling should be eponymous, as it was this observation on the direction of blood flow in the veins that confirmed Harvey's views that the blood moved in a circle.

[2] Leriche's syndrome. René Leriche (1879–1955) described Leriche Syndrome in 1940.

The Relationship of Clinical Findings to the Site of Disease

By associating the symptoms and signs found in a case of arterial disease it is usually easy to determine the site of the major arterial obstruction.

Relationship of Clinical Findings to Site of Disease

AORTO ILIAC OBSTRUCTION	Pulses all absent in both limbs
	Claudication in both buttocks, thighs and calves
	Impotence common (Leriche)
ILIAC OBSTRUCTION	Unilateral absence of all pulses
	Unilateral claudication in thigh and calf and sometimes buttock
FEMORO-POPLITEAL OBSTRUCTION	Unilateral absence of pulses below femoral
	Claudication in calf and sometimes foot
DISTAL OBSTRUCTION	Ankle pulses absent
	Claudication in calf and foot

Double Blocks.—The presence of secondary obstructions can usually be inferred. Thus a patient with signs of iliac artery obstruction, but with rest pain and pre-gangrene of the foot, must have a secondary obstruction since collateral circulation around an isolated iliac artery obstruction is usually excellent. The severe symptoms would indicate a secondary obstruction probably in the femoral or popliteal arteries.

The Investigation of Arterial Disease

General Investigations.—The general examination and investigation of a case of arterial disease needs to be comprehensive. It should be appreciated that atherosclerosis is a generalised disease and the patient may suffer from ischæmic heart disease, renal impairment, cerebro-vascular disease, hypertension, etc., as well as peripheral arterial disease. Atherosclerosis is also a common accompaniment of diabetes, which always needs to be excluded. A previous history of peptic ulceration is also common.

Patients with arterial disease also frequently suffer from age related diseases, e.g. prostatic obstruction, and habit related diseases, e.g. chronic bronchitis or carcinoma of bronchus. Urine examination, chest X-ray, E.C.G., urea and electrolytes, blood count, etc., will therefore be among the appropriate investigations.

Special Investigations.—It is important to realise that it is unnecessary to submit every patient suffering from arterial disease to a series of special investigations. The decision to do so depends largely upon the degree of disability.

Plain X-ray will show the presence of arterial calcification. Flecks of calcium may outline an aneurysm.

Arteriography (figs. 127, 128).—This procedure furnishes information concerning the state of the arteries—constrictions, obstructions, or dilatations, and to some extent the state of a collateral circulation. The vascular pattern of tumours may also be studied (e.g. Chap. 56). Arteriography should be restricted to those patients in whom surgical treatment is being considered, or

in whom a diagnosis cannot otherwise be made. The occasional hazards of arteriography include thrombosis, and arterial dissection, and very occasionally arterial rupture. Paraplegia, or even a fatality, may result from aortography (see trial injection, below).

The following methods are employed:

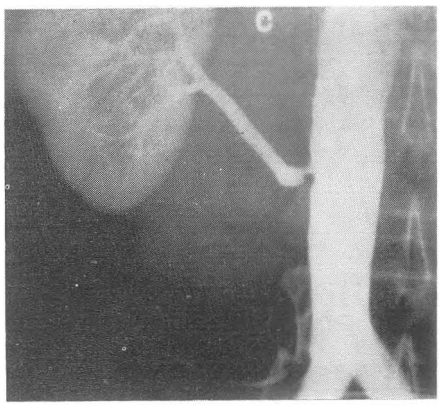


FIG. 127.—Renal artery stenosis. Arteriogram by retrograde femoral catheterisation. Note the post-stenotic dilatation.

(i) *Retrograde Percutaneous Catheterisation.*—Under a local or general anæsthetic, and with operating theatre aseptic ritual, a special needle and cannula (fig. 128) are inserted into the artery. A suitable length of flexible guide wire is threaded through the cannula, which is withdrawn so that a polythene catheter can be passed over the guide wire and into the vessel. The common femoral artery is the usual route in most instances including aortic arch, renal, mesenteric, iliac and femoro-popliteal arteriograms. The right brachial artery may be used for coronary arteriograms, and arch aortograms to demonstrate disease in the subclavian and carotid arteries.

(ii) *Direct Percutaneous Arterial Puncture.*—Now less popular, this may be used for carotid arteriograms, and for demonstrating the abdominal aorta (translumbar route) when the femoral arteries are occluded, or if the retrograde method cannot produce the necessary information (fig. 129).

A trial or test injection of 5 to 10 ml. of 45 per cent. Hypaque must be made in order (*a*) to exclude iodine sensitivity (Chap. 46) (cf. intravenous cholangiography) and (*b*) to

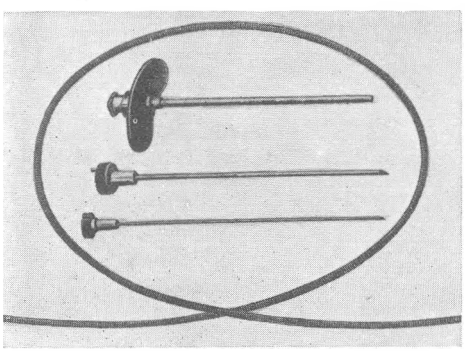


FIG. 128.—Seldinger needle and guide wire for introducing an arterial catheter.

FIG. 129.—Translumbar aortography. Atherosclerotic stenosis of both common iliac arteries prevented proper visualisation by retrograde catheterisation.

ascertain that the tip of the catheter or the point of the needle is in the correct place. A dissecting aneurysm can occur if the bevel of the needle is partly in the wall of the artery. To prevent this hazard a special needle without a bevel but with a side hole may be employed.

If the result of the test injection is satisfactory, the whole volume (30 ml.) of the contrast medium is injected rapidly as a 'bolus', and serial X-ray exposures are made to obtain radiographs of the length of the arterial tree under examination. In the

case of the leg, the examination must extend from the lower part of the abdominal aorta to the ankle, as a second obstruction may be present.

(iii) *Use of Doppler Ultrasonic Blood Velocity Detector* (figs. 130 and 131). This apparatus works on the Doppler principle. An ultrasonic signal is beamed at an artery and the reflected beam picked up in a receiver and converted to an audible signal. The pitch of the signal received varies with the velocity of blood flow. The audible signal reflects the character of the pulse wave and is modified by stenosis or a proximal arterial obstruction and will be lost entirely over the site of an obstruction. With practice it is possible to map out obstructions and stenoses from the character of the signal over arteries at various sites.

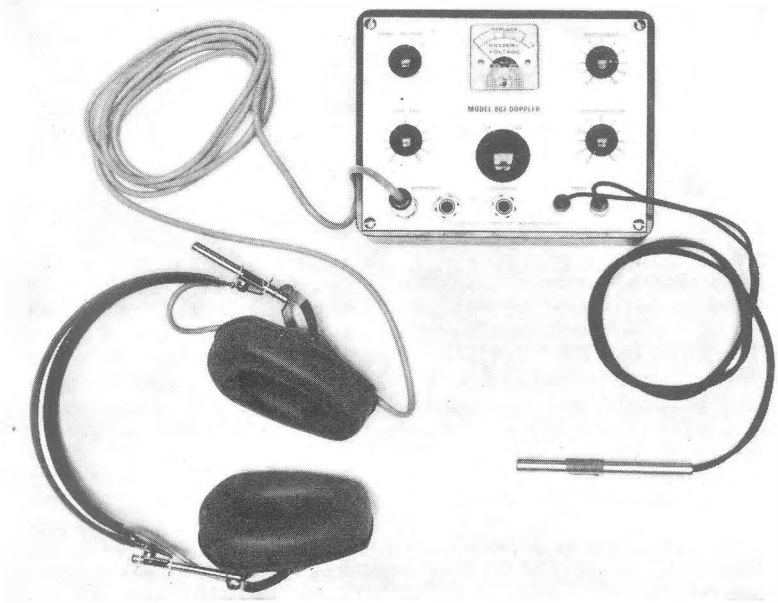


FIG. 130.—Doppler apparatus—probe, receiver and headphones.

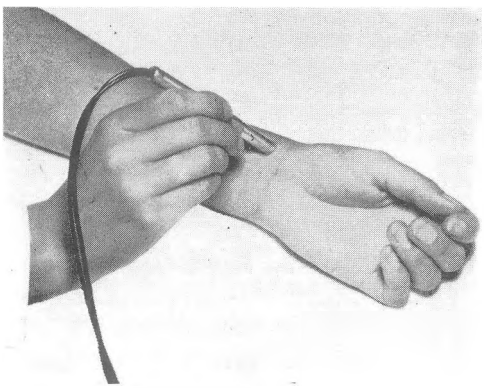


FIG. 131.—Doppler probe.

Use as a Stethoscope. The detector may also be used as a stethoscope, when, in conjunction with a standard inflatable cuff, it is possible to measure the blood pressure at sites where the arterial pulse cannot be palpated. The detector is placed over the posterior tibial artery and the arterial signal found. The cuff is placed around the ankle and inflated until the signal disappears. This level is the ankle systolic blood

Christian Johann Doppler, 1803–1853. Austrian Physicist.

pressure. With the detector still in the same place, the blood pressure at other points in the limb may be measured by altering the cuff site.

The ankle systolic blood pressure is greater than the brachial (arm) systolic blood pressure in normal individuals by 5–15 mmHg (0·67–2 kPa). Therefore the 'pressure index' $\left(\frac{\text{ankle blood pressure}}{\text{arm blood pressure}}\right)$ is normally greater than one. A 'pressure index' of less than one indicates some degree of arterial obstruction between the left ventricle and the ankle. The lower the pressure index the greater the degree of obstruction. When the ankle blood pressure falls below 30 mmHg (4·0 kPa), the measurements become difficult and gangrene of the extremity is imminent.

(iv) *Other methods of special investigation.*—Measurement of walking distances using a treadmill, and measurements of blood flow or ankle systolic blood pressure after exercise are useful for assessing treatment methods. Oculoplethysmography (OPG) is fast becoming a method of assessing blood flow and disease in the carotid arteries. ^{133}Xenon clearance is also being used to build up computerised images of flow and disease in the lower limb.

(v) *Blood lipids and glucose tolerance.*—Abnormal levels of blood cholesterol, triglycerides and lipoproteins are found in about 60% of all patients with atherosclerotic peripheral arterial disease. A proportion of these patients, while not actually diabetic, also have impairment of glucose tolerance. The significance of these biochemical abnormalities in aetiology, management and prognosis is at present uncertain.

PRINCIPLES OF MANAGEMENT

A. Conservative Methods

Conservative methods of treatment are directed towards two aims: (1) Relief of symptoms; (2) Reduction in the rate of progression of atherosclerosis.

1. **Relief of Symptoms.**—Conservative methods are rarely capable of producing marked improvement.

Vasodilator methods have no significant effect on muscle vessels, probably including most collateral vessels, but are effective in dilating skin and subcutaneous vessels. For this reason, vasodilator methods do not improve intermittent claudication but usually have a beneficial effect on ischæmic ulcers and trophic changes. They may also help the mildest forms of rest pain.

(i) *Oral Vasodilator drugs.*—These are included in the first line treatment immediately after an acute arterial obstruction. At this time the claudication is at its most severe and spontaneous improvement may be expected as collateral circulation improves. This improvement is attributed both by physician and patient to the vasodilator drug, which thereby acquires a completely undeserved reputation. Some patients are helped by alcohol by mouth (sherry, brandy, whisky, etc.) but probably not always because of the vasodilator effect! The drink is frequently administered as a 'night cap'.

(ii) *Paravertebral injections of the Sympathetic Chain.* ('Chemical Sympathectomy').[1]—This is an alternative to surgical sympathectomy and like it rarely improves, and may even worsen, claudication. Injection can be given on more than one occasion. The procedure is contraindicated if anticoagulants are in use.

[1] The reader is referred to the book *Annapurna*, by Maurice Herzog (*Jonathan Cape, Ltd.*), who experienced intra-arterial and paravertebral injections for the treatment of frostbite.

Technique.—For the leg this implies the injection of 1% lignocaine beside the bodies of the second, third, and fourth lumbar vertebræ. Fig. 132 shows how to position the patient (a lumbar-puncture position is used for patients who cannot sit up). Fig. 133 shows how a long (15 cm.) needle is inserted, firstly to seek the side of the vertebral body and, secondly, to pass alongside it. Then 1% lignocaine (10–20 ml.) is injected. The effect of the injection (warmer feet and improved venous filling) may last for two or three days. Further injections may be given, but a permanent effect more easily achieved if 5 ml. of 5% phenol solution in water is injected (chemical sympathectomy). Care is needed to avoid penetrating the aorta or the vena cava.

FIG. 132. — Sitting position for a paravertebral injection. The patient rests his head and arms on a pillow.

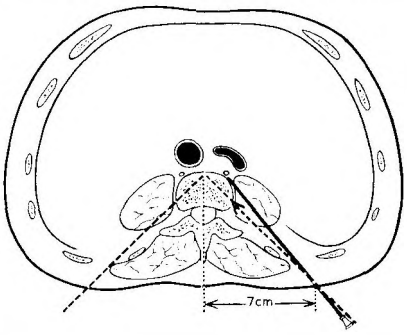


FIG. 133.—Lumbar paravertebral injection.

Precision injection.—With the patient in the lumbar puncture position, the employment of an X-ray T.V. image intensifier, used in two planes, assists greatly in the accurate positioning of the needles. Injection of a radiopaque contrast (diatrizoate) may be used in order to be sure that the local anæsthetic or the phenol is not going to be injected into the paravertebral veins or the vena cava.

For the arm a shorter needle is introduced vertically to the skin, 4 cm. lateral to the spines of the second, third and fourth thoracic vertebræ. The needle seeks the back ends of the ribs, and is then passed over or under them for another 1 cm. while the bolus of lignocaine is injected. Special care is needed to avoid puncturing the lung and thereby causing a pneumothorax.

(iii) *Reflex heating.* The affected limb is nursed exposed to room temperature, while the trunk and remaining limbs are warmed by an electric pad or blanket. Reflex vasodilatation of the exposed limb usually results, and may be a helpful adjunct, especially in acute ischæmia where improvement may be expected as collateral circulation develops.

Cooling the exposed limb with a fan as often practised may reduce local metabolism but will counteract the effect of reflex heating. Direct heating or freezing of an ischæmic limb should never be employed.

(iv) *Buerger's position* (fig. 134). The head of the bed is raised 15 cm. The hydrostatic congestion is associated with a slight increase in blood flow and may afford some relief to rest pain.

(v) *Buerger's Exercises* (fig. 135). — The leg is elevated on cushions or an upturned chair for two minutes, and lowered beside the bed for two minutes. Elevation and lowering are repeated for séances of about thirty minutes, twice or thrice daily.

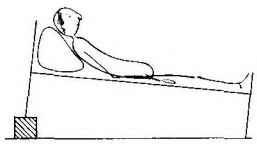


FIG. 134.—Buerger's position.

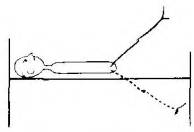


FIG. 135.—Buerger's exercises.

Intermittent venous occlusion devices are logical developments of (iv) and (v).

Protection of Ischæmic Parts.—Steps must be taken to prevent gangrene occurring in the areas likely to be subject to pressure (e.g. the heel, p. 168). All cases of peripheral vascular disease need careful chiropody; gangrene often follows the trauma of a badly cut toe nail.

Muscular Activity.—Exercise training to improve general physical fit-

ness and promote muscular activity in the involved limbs is associated with an increase in claudication distance of 100% or more in most patients. Similar improvements in angina pectoris can be obtained by exercise training.

Pain Relief.—Pain relief for intermittent claudication is not required since the occurrence of pain remains within the patient's control. Relief of rest pain is difficult and may require strong analgesic drugs which frequently fail to give complete relief. Simple drugs such as paracetamol should be tried first. Stronger analgesics such as oral dihydrocodeinone ('D.F. 118') 30 mg are often required. Strong analgesics causing dependence such as morphine and pethidine should be avoided if possible.

Heel Raise.—Raising the heel of the shoes has been advocated to diminish the load on the gastrocnemius muscle.

Other Forms of Drug Treatment

(i) **Low molecular weight dextran**—given intravenously this is helpful in the temporary acute ischæmic phase. It may also be valuable in vasospastic disease (p. 163).

(ii) **Anticoagulant drugs.**—Agents such as 'Dindevan' or 'Warfarin' given in the long term may reduce the incidence of thrombosis. Alternatively, soluble aspirin, 300 mg, taken at breakfast time is being tried as an inhibitor of platelet adhesion in the prophylaxis of further arterial thrombosis (e.g. coronary thrombosis).

(iii) **Fibrinolysis.**—Fibrinolytic agents such as streptokinase may be useful, especially in acute recent arterial thrombosis, but the effect is capricious, sometimes dramatically reopening blocked arteries and at other times a complete failure.

(iv) **Alpha-tocopherol (Vit. E.).**—Long-term administration may be of some help in increasing claudication distance. The effect may not appear for 6 months.

2. **Reduction in the Rate of Progression of the Disease.**—Many failures in direct arterial surgery for atherosclerosis are due to progression of the disease. The risk factors for atherosclerosis include smoking, diabetes, hypertension, raised serum lipids (cholesterol and triglycerides), obesity, and lack of exercise. In the hope that some reduction in rate of deterioration can be achieved these risk factors should be contended with:—

(i) **Smoking.**—Tobacco smoking should be strongly discouraged and every effort made to get the patient to stop smoking. Symptomatic peripheral vascular disease due to atherosclerosis is very rare in non-smokers (Eastcott).

(ii) **Obesity.**—Overweight patients should be encouraged to lose weight and weight increase on stopping smoking must be avoided.

(iii) **Lipid abnormalities.**—Serum lipoproteins contain cholesterol and triglyceride. β lipoproteins are cholesterol rich and pre-β lipoproteins are triglyceride rich. The commonest abnormalities in peripheral vascular disease are Frederickson Type II hyperlipoproteinæmia (raised β lipoproteins) and Type IV hyperlipoproteinæmia (raised pre-β lipoproteins). Low cholesterol diet or drugs such as cholestyramine are useful for Type II abnormalities. Weight reduction will frequently correct Type IV abnormality.

(iv) **Diabetes and hypertension.**—Should be treated by standard methods.

(v) **Exercise training.**—Has been discussed (p. 144).

Harry Hubert Grayson Eastcott Contemporary. Surgeon, St. Mary's Hospital, London.

B. Operative Methods

1. **Direct arterial surgery** is used either to restore the continuity of blood flow, or to provide an alternative channel (by-pass).

Arterial Repair.—For closure of a simple wound or arteriotomy after embolectomy, a continuous suture is employed, using 0000 silk or polypropylene on a 16 to 20 mm. atraumatic needle. A patch of vein or Dacron is frequently used for repairs in order to prevent narrowing the lumen of the vessel (fig. 136). If possible, small sections of the long saphenous vein should not be used as it may be required for by-pass grafting at a later date.

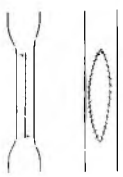


FIG. 136.—Where arterial suturing may reduce the lumen, a vein patch (right) is useful.

Artery By-pass Grafting (figs. 137 and 138).—*Veins* are commonly used for by-pass grafting of peripheral vessels, especially the femoro-popliteal artery segments. The long saphenous vein is usefully placed for by-passing a femoro-popliteal obstruction. A suitable length of long saphenous vein is excised, after the careful ligation of all its tributaries with fine silk. The vein is then distended with warm heparinised saline or Hartmann's solution, reversed to prevent obstruction of blood flow due to its valves and sutured end to side to the patent artery above and below the occlusion (see p. 160).

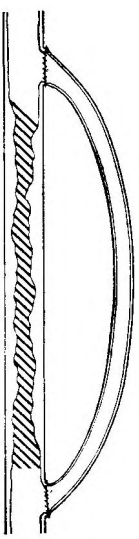


FIG. 137.—By-pass grafting.

Synthetic grafts of knitted or woven Dacron or Teflon are used mainly for aorto-iliac replacement (fig. 144). They are less suitable for peripheral work, as those of small calibre tend to thrombose more easily than vein grafts. As they are crimped in concertina fashion, they require to be stretched before insertion. Very little blood oozes through the interstices of the woven variety. After insertion, these grafts are incorporated by an invasion of fibroblasts, and within the lumen they become lined with pseudoendothelium of uncertain origin.

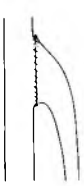


FIG. 138.—End of graft widened and sutured as a 'sucker foot' to the side of the parent artery.

Thrombo-endarterectomy ('**disobliteration**' or '**rebore**') is described on p. 160.

Embolectomy and thrombectomy are described on p. 156.

2. **Indirect Surgery.—Sympathectomy.**—This is employed (a) for the treatment of vasospastic conditions (p. 164), (b) in Buerger's disease and other cases of peripheral arteritis (p. 163) and (c) in those cases of atherosclerosis where the disease is restricted to the most distal vessels, e.g. plantar or digital arteries, or where its vasodilator effect in improving skin lesions, e.g. small ischæmic ulcers, is required. See vasodilator methods (p. 143). The techniques are described on p. 165.

DISEASES OF ARTERIES

Injury

Injuries to arteries usually take the form of *division*, *rupture*, or *contusion*.

Division occurs in penetrating wounds. A classical example is ' butcher's thigh ', which is a penetrating wound in the femoral triangle, due to the knife slipping while boning meat. Any of the femoral arteries may be severed, sometimes with an immediate fatal result. *Rupture* of an artery can be caused by severe trauma, e.g. the popliteal, in the case of a dislocated knee. *Contusion* of the arterial wall also follows violence (e.g. a severe blow).

Clinical Effects of Injury.—Primary arterial hæmorrhage is usually obvious (p. 70), but the effects of a closed rupture or contusion may be less apparent. In cases of rupture there will be a swelling which may pulsate (pulsating hæmatoma). Contusion usually causes a local thrombus (fig. 139) if a main artery is involved. In all three forms of injury the life of the limb may be threatened by ischæmia.

Late Effects of Injury.—An arteriovenous fistula may be caused by a penetrating wound. A false aneurysm may follow penetration or rupture, while a saccular aneurysm may follow contusion (fig. 140).

FIG. 139. — Thrombosis of the popliteal artery caused by a contusion. The foot was gangrenous. (Actual size.)

Treatment includes arresting hæmorrhage (p. 74), and reconstruction of the artery (p. 146), interposition of a vein graft, or thrombectomy (p. 156), as the case may be. The possibility of injury to other structures (e.g. nerves) must not escape the surgeon's notice, as these also will require appropriate treatment. In cases where a main artery has to be ligated to save life, or reconstruction is impossible, or when a local thrombosis becomes extensive, the limb will become severely ischæmic. Gangrene may occur or a group of muscles may necrose (e.g. Volkmann's ischæmia, Chap. 23). However, the rapid opening up of an efficient collateral circulation especially in the young, may save a limb from amputation. In the case of the leg there will be a period of intermittent claudication which disappears slowly over a matter of a few months. Conservative measures, as outlined on p. 143, are generally employed. Arteriography will show if an operation is likely to be successful.

FIG. 140. — Saccular palmar aneurysm in a hæmophiliac, due to the repeated trauma of striking a nail-removing tool with his palm. (Actual size.)

Aneurysm

An aneurysm is a sac filled with blood in direct communication with the interior of an artery.

Aneurysms may be:— → True { fusiform, saccular, dissecting.
↓ → False

Arteriovenous (fistula), congenital and acquired.

A *true aneurysm* is due to dilatation of an artery, whereas a *false aneurysm* is a sac lined by condensed cellular tissue which communicates with the artery through an opening in its wall.

A *fusiform aneurysm* (the commonest type), is one in which the lumen is more or less equally expanded. The artery wall is usually weakened by severe atheroma and hypertension. In syphilis, the media of the arch of the aorta may be weakened by endarteritis of the vasa vasorum (fig. 141). In a *mycotic*[1] *aneurysm*, the artery wall is weakened by infection, either in the form of a local abscess, or because an infected embolus has come to rest at that point.

FIG. 141. — An aortic aneurysm has eroded the sternum and is about to rupture. (*Raymond Helsby, F.R.C.S., Liverpool.*)

A *saccular aneurysm* is due to stretching of part of the arterial wall and commonly follows injury to superficial arteries (fig. 142).

Clinical Features of an Aneurysm.—(*a*) *Intrinsic.*—A swelling exhibiting expansile pulsation is present in the course of an artery. The pulsation diminishes if proximal pressure can be applied, and the sac itself is compressible,[2] filling again in two or three beats if proximal pressure is released. A thrill may be palpable, and auscultation sometimes reveals a bruit.

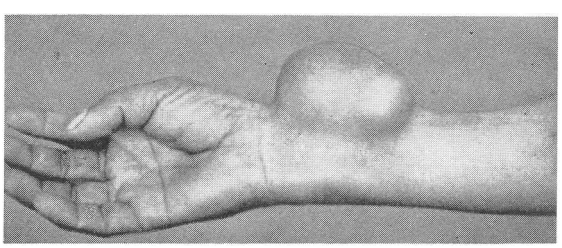


FIG. 142.—Saccular aneurysm of the radial artery. (*Professor A. K. Toufeeq, F.R.C.S., Lahore, Pakistan.*)

(*b*) *Extrinsic.* — Neighbouring or distal structures are affected. Thus pressure on veins or nerves causes distal œdema or altered sensation. In the popliteal fossa pressure eventually will cause gangrene. Bones, joints, or tubes (such as the trachea or œsophagus), are sometimes affected, but structures which are resilient, such as the intervertebral discs, often withstand prolonged pressure.

[1] 'Mycotic' is a misnomer, as the disease is not due to a fungus but to bacterial infection.
[2] Large aneurysms are frequently full of mural thrombus and may not be compressible.

Differential Diagnosis.—1. *Swelling Under an Artery.*—An artery may be pushed forwards, e.g. the subclavian by a cervical rib, and thus rendered prominent. Careful palpation distinguishes this condition.

2. *Swelling Over an Artery.*—Transmitted pulsation is liable to be mistaken for that caused by expansion. However, posture may diminish pulsation; thus a pancreatic cyst examined in the genupectoral position falls away from the aorta, and consequently pulsation is less definite.

3. *Pulsating tumours*, such as bone sarcoma, osteoclastoma, and a metastasis, especially from a hypernephroma.

4. *An Abscess.*—Before making an incision into a swelling believed to be an abscess, e.g. of the chest wall (fig. 141) in the groin, in the axilla, or in the popliteal fossa, it is essential to make sure that it does not pulsate! This mistake has been made many times.

5. *A serpentine artery* (e.g. innominate, carotid).

Natural Terminations.—1. *Extension and Rupture.*—This occurs either slowly as a leakage, or suddenly, in which case death may follow if a large vessel is involved (fig. 141).

2. *Spontaneous Cure* (Consolidation).—This sometimes occurs in small peripheral saccular aneurysms. In large aneurysms laminated mural thrombus is common but this does not prevent progression to rupture.

3. *Infection.*—Usually arises from organisms in the blood-stream. Signs of inflammation supervene and, if untreated, suppuration and rupture follow.[1]

Treatment.—This is surgical. The scope of the operation required may be indicated by arteriography, which will show the state of the arteries above and below the lesion but due to the presence of mural laminated blood-clot, the aneurysmal dilatation is seldom visualised.

Fig. 143 is designed to itemise the procedures which are available:—

Hunterian Ligation (fig. 143A).—This operation was designed by Hunter for the treatment of popliteal aneurysm, but the principle can be applied to the treatment of other limb aneurysms, when a simple and quick measure is needed.

The ligature must be placed above the main collateral nearest the aneurysm (e.g. above the descending geniculate artery). In this way the blood flow through the aneurysm is not immediately cut off. The collateral circulation is given time to open up before consolidation occurs.

Exclusion and By-pass Grafting (fig. 143B).—Excision is not always necessary. It may be undesirable, as neighbouring vital structures may be damaged. The excluded aneurysm consolidates with clot, and shrinks into a fibrous mass.

Excision and Grafting (fig. 143C).—Veins, or knitted or woven Dacron tubes, are used. The graft can be inserted 'end to end' or 'end to side'.

Excision of the sac and arterial reconstruction (Matas' reconstructive aneurysmorrhaphy) fig. 143D, is a very useful procedure for small saccular aneurysms of important arteries (e.g. femoral or popliteal).

[1] Salmonella organisms may sometimes be isolated from ruptured aortic aneurysms.

Rudolph Matas, 1860–1957. Professor of Surgery, Tulane University, New Orleans, Louisiana, U.S.A. (1894–1923). Introduced Endo-Aneurysmorraphy in 1888.

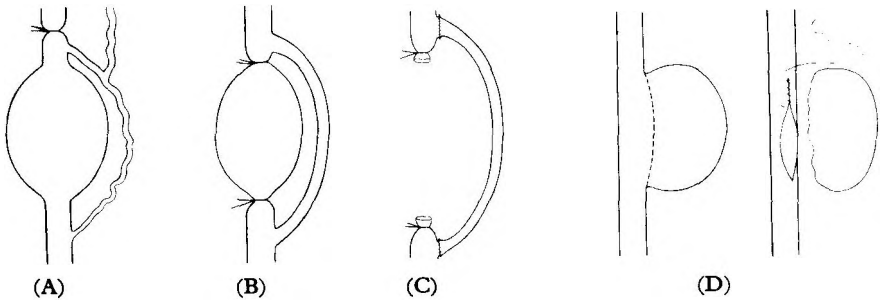


FIG. 143.—Operations for aneurysm.

(A) Hunterian ligation.
(B) Exclusion and by-pass graft.
(C) Excision and graft.
(D) Reconstruction (Matas).

Ligation and Excision.—Aneurysms of secondary arteries are treated by ligation of the proximal and distal vessels. Excision is not obligatory, save in special anatomical or physiological circumstances (fig. 140).

Abdominal aortic aneurysms are usually amenable to surgery. The patient complains of abdominal or back pains, and may be aware of the pulsatile abdominal mass. Left alone, the average expectation of life before rupture is about three years. When the aneurysm ruptures, the patient presents with abdominal pain and hæmorrhagic shock. The diagnosis may be difficult, as the clinical features may mimic some other abdominal emergency (e.g. perforated diverticulitis, acute pancreatitis, etc.). Emergency surgery has a high mortality, due usually to an unstable cardio-vascular system or renal failure resulting from prolonged hypotension. Modern intensive care wards have improved the prospects. Elective surgery, with a mortality of 5%, is therefore advised in otherwise fit patients, and when the aneurysm is believed to lie below the level of the renal arteries (usually the case). *An ultrasonogram* may be more helpful than an aortogram in locating the position of the renal arteries.

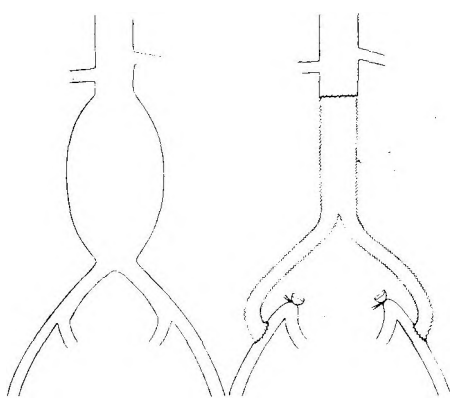


FIG. 144.—Grafting for an abdominal aneurysm. The remains of the aneurysm are omitted for the sake of clarity.

Exposure is through a long mid-line, a curved transverse,[1] or a flank incision, the latter using an extraperitoneal approach. The aorta is curiously amenable to encirclement with the finger and the application of a clamp just

[1] Post-operative wound dehiscence and incisional hernia are less common if a transverse incision is employed.

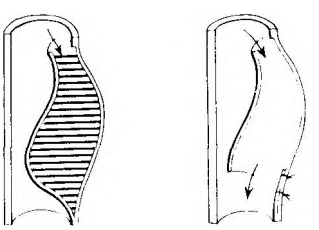
below the renal arteries.[1] The aneurysm may simply be excluded or evacuated and obliterated by suturing. The aneurysm is opened and evacuated, and a Dacron bifurcated graft is inserted (fig. 144). The walls may be wrapped round the graft.

Dissecting aneurysms are frequently rapidly fatal and occur in the ascending aorta (65%) or near the origin of the left subclavian artery (35%). The patients are hypertensive, usually markedly so, and the dissection results from a spontaneous tear in the intima which allows blood to insinuate itself between the inner and outer layers of the media in an aorta where medial degeneration has occurred, e.g., in hypertension and in Marfan's syndrome. The dissection may spread proximally into the aortic valve ring, resulting in acute aortic incompetence (ascending aortic tears) or distally. The distal dissection may be extensive reaching even into the iliac and femoral vessels where obstruction of the true lumen may mimic embolism or thrombosis. Alternatively, death may occur from external rupture. The diagnosis is frequently difficult because symptoms vary considerably depending on which arteries are obstructed. The dissection itself causes severe pain—anterior chest pain suggesting myocardial infarction and/or pain in the back (chest and/or lumbar region). The combination of severe chest and back pain in a markedly hypertensive patient is suspicious of a dissecting aneurysm, and the diastolic murmur of aortic incompetence may help to clinch the diagnosis. Chest X-ray may show an expanded aortic shadow and emergency aortography will usually demonstrate the double lumen.

Conservative Treatment of Dissecting Aneurysms (Wheat).—The mortality from dissecting aneurysms in the acute stage has been significantly reduced by vigorous conservative measures. These are now the treatment of choice for the acute stage after dissection. The aim is to reduce the systolic blood pressure quickly to levels of 100–110 mmHg (13·3–14·6 kPa).[2] This is achieved using trimetaphan (Arfonad) in an intravenous drip of 1–2 mg per ml. The rate of the drip is adjusted to produce the desired level of blood pressure. Simultaneously reserpine 1–2 mg is administered i.m. or i.v. and guanethidine 50 mg commenced twice daily. The trimetaphan drip is gradually withdrawn as the blood pressure comes under the control of the other drugs. The urine output and peripheral pulses are monitored.

Surgical Treatment of Dissecting Aneurysms.—The 're-entry operation' (fig. 145B) consists of incising the artery and opening a 'window' in the false sac so that blood can flow freely back into the true lumen so relieving obstructive symptoms. Resection of the aorta at the site of the tear, with grafting, carries a very high mortality in the acute stage, but has a place in management once a patient has been stabilised by conservative treatment. Cardio-pulmonary bypass (Chap. 41) will usually be required and replacement of the incompetent aortic valve may also be necessary.

Aorto-duodenal fistula is an uncommon but eminently treatable complication of abdominal aortic replacement surgery. It should be suspected whenever hæmatemesis or melæna occurs in the months or years after operation. A successful outcome is only achieved by prompt operation, separating aorta from duodenum, closing the holes, and also interposing some omentum.



(A) (B)

FIG. 145.—Dissecting aneurysm. (A) The dissection may obstruct the lumen. (B) The re-entry operation.

False aneurysms are traumatic in origin, and the extravasated blood is enclosed in a sac of condensed cellular tissue. Fig. 146 is a typical example, and the caption recounts the method of repair.

[1] The encirclement of the aorta with the finger below the renal arteries was demonstrated by Sir Astley Cooper, Surgeon, Guy's Hospital, London, and President of the Royal College of Surgeons of England 1827.

[2] Hypotensive therapy for aneurysms was in fact used empirically in the 18th century by employing absolute bed-rest, starvation and blood letting.

Myron W. Wheat, Jr., Contemporary. Professor of Thoracic Surgery, University of Florida College of Medicine, U.S.A.

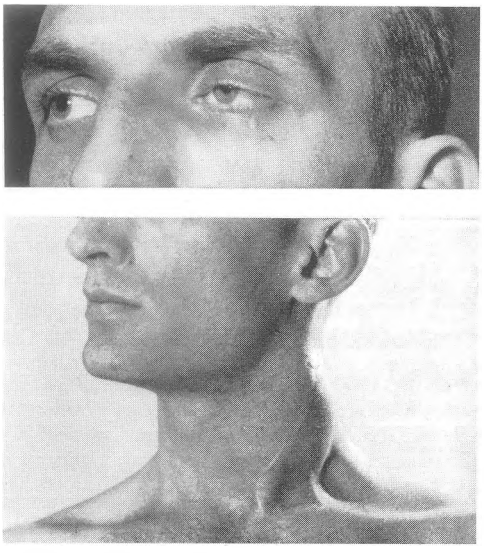


FIG. 146.—A false, and rapidly extending aneurysm of the common carotid caused by a stab wound. Note (above) the ptosis, myosis, and enophthalmos of a Horner's syndrome, due to concomitant severance of the cervical sympathetic nerve.

The scales of justice hung in the balance while the carotid artery was repaired and the sac evacuated (not excised), with a good result.

an enhanced venous return and venous pressure result in an increase in pulse rate and cardiac output. The pulse pressure is high if there is a large and persistent shunt. Left ventricular enlargement and, later, cardiac failure will occur. A congenital fistula in the young may cause overgrowth of a limb. In the leg, indolent ulcers may result from relative ischæmia below the short circuit.

Clinically, a pulsatile swelling may be present if the lesion is relatively superficial. On palpation, a thrill is detected and auscultation reveals a buzzing continuous bruit. Dilated veins may be seen, in which there is a rapid blood flow. Pressure on the artery proximal to the fistula causes the swelling to diminish in size, the thrill and bruit to cease, the pulse-rate to fall[1] and the pulse pressure to return to normal.

Arteriovenous Fistula. — Communication between an artery and a vein (or veins) may be either a congenital malformation, or acquired by the trauma of a penetrating wound or a sharp blow. Arteriovenous fistulas are also created surgically in the forearms or legs of patients undergoing renal dialysis. Any arteriovenous communication has a structural and a physiological effect.

The *structural effect* of the arterial blood flow on the veins is characteristic, as they become dilated, tortuous, and thick walled (arterialised) (fig. 147), and they also make the lesions diffuse and so render surgical procedures difficult.

Physiological Effect. — The combination of a leak from the high pressure arterial system and

FIG. 147.—Post-traumatic arteriovenous aneurysm at the wrist. Note the prominent arterialised veins.

[1] Known variously as Nicoladoni's (1875) or Branham's sign (1890).

Johann Friedrich Horner, 1831–1886. Professor of Ophthalmology, Zürich, Switzerland. Described the syndrome which bears his name in 1869. The effects produced by section of the cervical sympathetic chain had been noted previously by François Pourfour Du Petit (1664–1741) in 1710.
Carl Nicoladoni. 19th century Surgeon, Vienna.
H. H. Branham. 19th century Surgeon, U.S.A.

Arteriography confirms the lesion, which is noteworthy for the speed with which venous filling occurs. It is often difficult to pinpoint the actual site of the fistula.

Treatment.—*Congenital* lesions are usually stationary. Excision is advocated only for severe deformity or recurrent hæmorrhage. It is often wise to enlist the aid of a plastic surgeon in order that proper ablation and reconstruction can be effected. Ligation of a 'feeding' artery is of no lasting value.

The *acquired* lesions tend to be progressive, and operation is indicated. The vessels are separated, and, if possible, repaired by suturing, any intervening sac being excised. Failing this, ligation of the involved artery and vein is required both above and below the lesion (quadruple ligation). Vein or Dacron grafts may be required.

EMBOLISM AND THROMBOSIS

An embolus is a body which is foreign to the blood-stream, and which may become lodged in a vessel and so cause obstruction.

Simple emboli are due to blood-clot, vegetations from cardiac valves, or fragments from an atheromatous plaque. In the heart, mitral stenosis or auricular fibrillation predisposes to their formation, which is also encouraged if fibrillation is treated with quinidine. An endocardial mural thrombus, following a myocardial infarct, accounts for about one third of cases.

In some situations the results of embolism are characteristic, e.g. :

BRAIN.—The middle cerebral artery is most commonly affected, resulting in hemiplegia, temporary or permanent.

RETINA.—Amaurosis fugax is fleeting blindness caused in this context by the passage into the central artery of the retina of a minute thrombus emanating from an atheromatous plaque in the carotid artery. A complete obstruction causes total and permanent blindness.

MESENTERIC VESSELS.—Causing engorgement and possibly gangrene of the corresponding loop of intestine.

SPLEEN.—Commonly affected, with local pain and enlargement.

KIDNEYS.—Resulting in pain in the loin and hæmaturia.

LUNG.—Pulmonary embolism (Chap. 40) is a catastrophe which may fatally interrupt convalescence after operation.

LIMBS.—An embolus is arrested at the bifurcation of a main vessel (fig. 148), causing *pain*,[1] *pallor*, *paresis*, *loss of pulsation*, and *anæsthesia*, followed by acute ischæmia, sometimes progressing to gangrene but often going on to recovery with varying degrees of chronic ischæmia. The more distal in the limb an embolus lodges, the more likely is gangrene to occur. Popliteal emboli lead to gangrene much more commonly than femoral or iliac emboli. Aortic

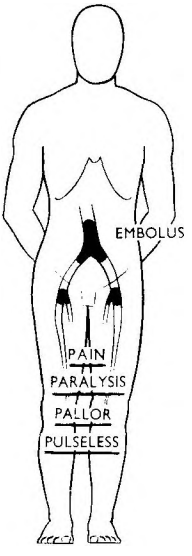


FIG. 148.—The symptoms and signs of embolism.

[1] In some cases of peripheral embolism pain is absent and the incident may pass unnoticed by a bedridden patient until gangrene is obvious.

bifurcation saddle emboli usually cause bilateral gangrene. In the arm emboli usually lodge at the brachial artery bifurcation and may cause gangrene of the hand.

The danger of extension thrombosis (consecutive or secondary thrombosis) is present immediately after an embolus becomes arrested. The presence of the embolus and the consequent stasis of blood favours the formation of blood-clot which, in a matter of hours, extends proximally as far as the nearest branch and distally down the main vessels and its branches (fig. 149). Should a collateral circulation not open up, the peripheral stasis, and therefore the thrombosis, extends still further, and gangrene occurs.

FIG. 149.—Extension-thrombosis subsequent to the arrest of an embolus.

Other types of acute arterial thrombosis are conveniently mentioned here, because clinically they mimic an embolus, and they are equally complicated by extension-thrombosis. Trauma causes arterial contusion (p. 147), which results in a local thrombus. The popliteal, external iliac, and axillary arteries (fig. 150) are specially susceptible. In axillary artery thrombosis, extension-thrombosis rapidly descends to obstruct the bifurcation of the brachial artery, a fact of great practical importance (see embolectomy). Acute arterial thrombosis, and this dangerous sequel, may also occur in an artery affected by atheroma (e.g. coronary thrombosis, cerebral thrombosis, femoral thrombosis, p. 158).

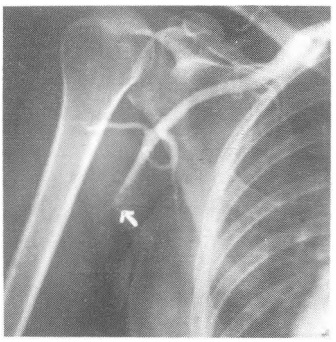


FIG. 150.—Arteriogram showing axillary artery thrombosis in a woman following trauma to the axilla over the headboard of a bed during the second stage of labour.

Treatment. — *Conservative treatment* is employed (*a*) if the embolism is mild and signs of recovery of the circulation are evident (loss of pain, return of movement), (*b*) in cases where the limb is showing early gangrene—too late for embolectomy! (*c*) if the patient is unfit for operation (heart failure, etc.). This is a rare contraindication since operation may be performed under local anæsthesia.

Embolectomy is advised if a main artery is involved, and, generally speaking, within ten hours of the onset of the emergency (see below).

Conservative treatment consists of (1) anticoagulant therapy, (2) treating any cardiac failure or arrhythmia, and (3) employing those vasodilator and other conservative measures which are described in the section on basic principles (p. 143).

Anticoagulant Therapy

Heparin.—This substance is present in body tissues, especially the liver and lung. It is produced by the mast cells, and prolongs the clotting time of blood. Heparin

is much the most effective anticoagulant for treating acute thrombo-embolism but must be given intravenously, usually in four-hourly doses of 5000–10 000 units (50–100 mg.), via an indwelling needle or polythene cannula. Continuous infusion by a pump (fig. 151) may be employed.

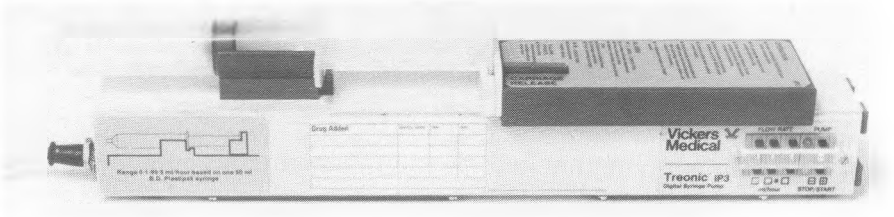


FIG. 151.—Continuous infusion pump for administering small volumes of a drug accurately and as a continuous infusion.

Overdosage leads to hæmorrhage, but the effect can immediately be neutralised by an intravenous injection of protamine sulphate. Each ml. of a 1 per cent. solution neutralises 1000 units of heparin.

Synthetic Anticoagulants.—Phenindione B.P. (e.g. Dindevan) and dicoumarol[1] derivatives (e.g. Warfarin or Marevan) depress the blood level of the prothrombin complex. They exert a full effect thirty-six to forty-eight hours after starting therapy, and for twenty-four hours after stopping. A loading dose is given in divided doses (loading dose of phenindione is 200 to 300 mg., Warfarin 30 to 50 mg.). The prothrombin level is estimated on the third day, and the amount given is then revised if necessary. Control is effected by estimation of the prothrombin time, which should be prolonged to two to three times the normal. The interval between estimation may be lengthened when a stable dose is found. These drugs may be useful for long-term administration to reduce incidence of further episodes of thrombo-embolism. They are much less effective for managing the acute episode.

Overdosage is shown by hæmorrhage, especially hæmaturia. Vitamin K_1 (Phytomenadione B.P., *not* the analogue) very quickly reverses the effects of synthetic anticoagulants. It is usually enough to give 10 mg. by mouth, but if bleeding persists, it should be administered by intravenous injection.

Contraindications to the use of anticoagulants include pregnancy, hepatic or renal deficiency, hypertension, or when there is a risk of hæmorrhage, e.g. in cases of peptic ulceration.

Combined Anticoagulant Therapy.—Heparin is injected for its rapid effect, and at the same time phenindione is started. When it is judged that the synthetic compound has exerted a full effect, the heparin is discontinued and clotting is controlled by prothrombin estimations (which, incidentally, are not valid within six hours of giving heparin).

Many surgeons prefer to rely upon heparin only, for four to six days before using the synthetic compounds, especially in cases of embolism.

Fibrinolysins.—Fibrinolytic agents (e.g. streptokinase) are useful and may lyse an acute thrombus or embolus without resorting to surgery. They are more effective in acute thrombosis than in embolism and are usually given in an intravenous infusion. Infusion intra-arterially through a catheter whose tip is close to the obstruction is more effective but carries a higher complication rate from hæmorrhage and infection. Infusion of fibrinolysins through a catheter in the right ventricle or pulmonary artery may be very effective in certain cases of severe pulmonary embolism. Certain forms of venous thrombosis (e.g. ilio-femoral) may also be treated. Heparin should not be used concomitantly with fibrinolysins.

[1] Dicoumarol was originally discovered in spoiled clover causing hæmorrhagic disease in sheep.

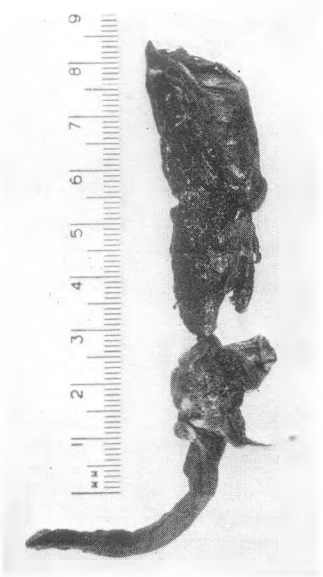


FIG. 152.—Aortic embolectomy. The extension-thrombosis extended proximally up to the renal, and distally into the iliac arteries.

Embolectomy and Thrombectomy

Local or general anæsthesia is used, dependent upon the patient's general condition and the scope of the proposed operation. The artery, bulging with clot, is exposed and held up by slings of fine rubber tubing. Through a longitudinal or transverse incision the clot begins to extrude and is removed, together with the embolus (fig. 152). Arterial clamps are applied as bleeding occurs, special note being made of the degree of retrograde bleeding (back bleeding). *Fogarty Catheterisation.* This is the most effective method of removing proximal and distal extension thrombus and also allows an embolus or thrombus to be removed from a vessel remote from the arteriotomy. The Fogarty catheter is like a ureteric catheter, with a balloon tip, and is introduced until it is deemed to have passed the limit of the thrombus. The balloon is inflated and the catheter withdrawn slowly, together with the clot. The procedure is repeated until bleeding occurs. The method is valuable in patients with an aortic bifurcation embolus, since the clot and embolus can be extracted by insertion of balloon catheters via the common femoral arteries in the groin and the patient is saved from a laparotomy. Post-operatively, anticoagulant therapy is commenced. *Other special measures* may sometimes be required to extract extension thrombus: (a) application of a spiral rubber bandage from below upwards, (b) retrograde flushing with heparinised Hartman's solution (1,000 units in 150 ml.).

In *axillary artery thrombosis*, it is often necessary to expose and even open the brachial artery at the elbow to be sure that it is free of clot or another embolus.

Papaverine sulphate (1% solution) painted on to the arteries may help to relieve post-operative spasm (Kinmonth).

Air Embolism—Air may be accidentally injected into the venous circulation (e.g. artificial pneumothorax), or sucked into an open vein. Thus venous air embolism occasionally complicates operations on the neck or axilla if a large vein is inadvertently opened, or it may be an accessory cause of death following a cut throat. The risks associated with intravenous infusion are reduced by the use of a drip chamber containing a spherical plastic float which plugs the exit when the fluid falls to a dangerous level (fig. 78).

When air enters the right auricle it is churned up, and the foam enters the right ventricle and causes an air-lock in the pulmonary artery, which may end in right-sided heart failure.

Treatment.—Trendelenburg's position encourages air to pass into the veins of the lower half of the body, and the patient is placed on the left side so that air will float into the apex of the ventricle, away from the pulmonary artery. Oxygen is administered to counteract anoxæmia, and to assist in the excretion of nitrogen. In serious cases the right ventricle should be aspirated by a needle passed upwards and backwards from below the left costal margin. If this fails, the heart is rapidly exposed for aspiration under direct vision.

Air may occasionally enter the left side of the heart, e.g. at open heart surgery, following puncture of a pulmonary vein during artificial pneumothorax or through a patent foramen ovale (paradoxical embolism). It may from there embolise coronary or cerebral arteries. Treatment is along similar lines to venous air embolism.[1]

Fat Embolism.—This condition, which is more common than generally supposed, usually follows severe injuries with multiple or major fractures. Cases have also

[1] Air embolism is also a risk following Fallopian tube insufflation, and following illegal abortion. The air may travel to the brain via the paravertebral veins.

Thomas J. Fogarty, Contemporary. Surgeon, University of Oregon Medical School, Portland, U.S.A.
John Bernard Kinmonth, Contemporary. Professor of Surgery, St. Thomas's Hospital and University of London.
Gabriele Fallopio, 1523–1562. Professor of Anatomy, Surgery and Botany, Padua, Italy.

been recorded following convulsive therapy. The fat may be derived from bone marrow or adipose tissue but recent work suggests that it is metabolic in origin, perhaps by aggregation of chylomicrons. Symptoms are evident a day or so after injury, and two more or less distinct types, cerebral and pulmonary, are recognised. In the cerebral type, the patient becomes drowsy, restless, and disorientated (delirium tremens may be suspected). Subsequently he is comatose, the pupils become small and pyrexia ensues. The pulmonary type is ushered in with cyanosis, which increases in intensity, and signs of right heart failure. White froth may occur at the mouth and nostrils. It may be mistaken for broncho-pneumonia or left ventricular failure.

One of the earliest signs may be emboli in the retinal arteries, which cause striate hæmorrhages and ' fluffy ' patches of exudate. The sputum should be examined for fat droplets, and fat may be excreted in the urine. A fall in the hæmoglobin value of the blood is a constant sign. Petechial hæmorrhages often occur.

Treatment consists of oxygen, early heparinisation, and intravenous low molecular-weight dextran (Rheomacrodex, p. 81). Intravenous 5 per cent. dextrose solution with 5 per cent. alcohol is supposed to increase the emulsifying power of the blood and reduce the size of fat globules. Hypothermia may be of use.

Other forms of emboli include *Infective emboli* of masses of bacteria or infected clot, which may cause mycotic aneurysms (p. 148), pyæmia, or infected infarcts, *Parasitic emboli* due to the ova of *Tænia echinococcus*—and *Filaria sanguinis hominis* (p. 49), and emboli of *malignant cells* (e.g. hypernephroma, Chap. 56).

Therapeutic Embolisation.—Has been used to arrest hæmorrhage from the gastrointestinal and urinary tracts, to treat arteriovenous malformations by blocking their arterial supply and to control the growth of unresectable tumours. Arterial embolisation requires accurate selective catheterisation using the Seldinger technique (p. 128). The left gastric or gastroduodenal artery may be occluded to treat a bleeding ulcer, occlusion of the hepatic artery will often retard the growth of liver metastases, and obstructing the renal artery has been used to infarct a renal tumour prior to surgery, and to arrest persistent hæmorrhage from an unresectable tumour. In patients with bleeding œsophageal varices, the portal system can be entered by percutaneous catheterisation through the liver and the veins supplying the varices can be embolised.

A wide range of materials have been used and they include blood clot, gel foam sponge, human dura, plastic microspheres, balloon catheters, quick setting plastics and mechanical devices made of stainless steel coils and wool. The advantages claimed for these procedures are that the morbidity is low particularly in very ill patients as a general anæsthetic is not required, and that they cause a minimum of upset in patients with advanced malignant disease where the aim of the procedure is palliation.

Caisson and Decompression Disease.—These similar conditions may affect divers, those who work in compressed air chambers, or who ascend in open aeroplanes to above 25,000 feet (7,620 m). If decompression is too rapid, bubbles of nitrogen are set free in the tissues and blood-stream, and occlude small vessels. Symptoms include pain in the muscles or joints, which may be excruciating (the ' bends '), and neurological disturbances; if the spinal cord is affected the patient suffers from weakness of the legs and sphincters. In severe cases, the lungs may be affected, and the patient complains of tightness of the chest and a dry cough (the ' chokes '). Caisson disease requires recompression and gradual decompression. The high altitude flyer is relieved by gradual descent. Inhalation of oxygen assists the excretion of nitrogen. If the spinal cord is not permanently damaged the prognosis is good, but hypertrophic changes may persist in the ends of long bones.

ATHEROSCLEROTIC OBSTRUCTION

The reader is advised to refer to the section on basic considerations and principles of arterial surgery (pp. 135–145) in conjunction with the following.

The Legs.—Patients with atherosclerotic obstruction of the arteries to the legs comprise the majority of cases of peripheral vascular disease. It is to

be remembered that the condition is part of a generalised disease, so that while the patient may present with symptoms in the legs, there may be manifestations in the coronary, carotid, or cerebral vessels. The patient is usually fifty years old or over, but a ' pre-senile ' variety may occur in early adult life, which must be distinguished from Buerger's disease (p. 162). Non-smokers are less affected. The incidence in males is much greater than that in females but this is changing. The increasing number of females may be associated with the increase in the smoking habit among women.

The symptoms are described on p. 136. They are caused by ischæmia, so they occur when the lumen of the artery (aorta, iliac, femoral, or popliteal) becomes severely diminished, or finally obstructed. As stenosis and occlusion occur, the collateral circulation usually has time to develop, and any episode of worsening of symptoms may be followed by a phase of improvement. Eventually, even the ostia of the collateral branches become obstructed, and should further thrombosis occur, rest pain and gangrene will follow (about 10 per cent. of patients). Sixty per cent. of the patients ultimately die from coronary thrombosis, 12 per cent. from congestive cardiac failure, and 12 per cent. from cerebral thrombosis or hæmorrhage.

The signs are described on p. 137 and the relationship of these clinical findings to the sites of disease are given on p. 140.

Arteriography (p. 140) is essential to show the exact site and the extent of the obstruction, as well as the presence of multiple lesions. It should, however, be performed only if surgical treatment is likely.

The treatment policy depends upon (1) the degree of disability, (2) the general condition of the patient, and (3) the feasibility of an operation as shown by arteriography.

(1) *The Degree of Disability.*—Rest pain and early gangrene are severe degrees of disability, and demand an attempt to salvage the leg. The disability of claudication is, however, very variable. Some patients with a walking distance of fifty yards may be content to restrict their activities within such a limit. Others, such as business executives, keen walkers, golfers, and gardeners complain that a distance of 300 to 400 yards (275–365 m) is intolerable, and they are reluctant to sacrifice either position or enjoyment.

(2) *The General Condition of the Patient.*—The absolute contraindications to operation are heart failure, severe bronchitis and emphysema, open tuberculosis, and advanced malignant disease. A recent coronary thrombosis or cerebral thrombosis (within 6 months), are temporary contraindications. Special care is required if a patient has an active peptic ulcer, since the stress occasioned by the operation (or even the arteriogram) may cause it to bleed. The ulcer requires priority treatment.

(3) *The Feasibility of Direct Arterial Surgery* to restore an adequate circulation depends upon the evidence given by arteriography of the site and extent of the obstructive disease. There should be no major stenosis proximal to the proposed upper limit of operation (e.g. in the iliac arteries when femoropopliteal reconstruction is contemplated). There should preferably be a good ' run off ' below the distal limit of reconstruction. Obstruction to the

calf arteries below a femoro-popliteal reconstruction will reduce the success rate of the operation in both short and long term.

Conservative Treatment as outlined on p. 143 is advised if the disability is mild, and/or the general condition of the patient is poor. It is also advised when an operation is not technically feasible or following the failure of a previous operation.

Operation

Aortic or Aortic-iliac Occlusion.—Thrombo-endarterectomy or Dacron by-pass grafting are both effective procedures. Thrombo-endarterectomy may be used for short localised lesions and Dacron by-pass grafts for more extensive blocks. A knitted Dacron bifurcation graft is a commonly used prosthesis. End-to-end or end-to-side anastomoses may be used but the latter are usually the simpler, and more convenient. The distal anastomoses should be to the external iliac arteries within the pelvis if possible, but if these arteries are involved by disease the distal anastomoses can be made to the common femoral arteries below the inguinal ligaments or even direct to the profunda femoris arteries.

Femoro-popliteal Artery Occlusions.—Vein by-pass grafting or thrombo-endarterectomy are suitable. Vein by-pass grafting is preferred, utilising thrombo-endarterectomy for short localised obstructions of 2–3 cm or longer blocks when a suitable vein is not available. Whichever method is used its distal limit should not be exactly behind the knee-joint since arteriotomies at this point usually fail rapidly. When the distal limit of the reconstruction is above the knee-joint an 8–10 mm Dacron prosthesis may be used for by-pass grafting instead of a vein. When the distal anastomosis is below the knee-joint vein by-pass grafting is much superior to either thrombo-endarterectomy or Dacron prosthetic grafting. Vein grafting should always be used if the distal anastomosis must be made to one of the calf arteries.

Aorto-iliac Endarterectomy.—Incision (p. 150). The intestines are temporarily eviscerated into a polythene bag, the aorta and iliac arteries are exposed and lifted up by slings of fine rubber tubing so that the clamps can be applied. The aorta is incised, and after any red jelly-like mural thrombus has been extracted, the inner atheromatous layer is peeled out, leaving an outer layer of media and adventitia. This procedure is carried down the iliac vessels until a satisfactory lumen and ' bleed-back ' is obtained. Should an intraluminal thrombus extend up to the level of the renal arteries (as in Leriche's syndrome, p. 139), it is milked or scraped out while pressure from a thumb occludes the aorta and protects the renal vessels. The arteriotomies are closed with a continuous 000 polypropylene suture after carefully tacking down the divided intima with one or two interrupted 0000 sutures at the lower limit of the reconstruction. *As a precaution against extension thrombosis* (p. 154) below the clamped-off iliac arteries many surgeons give intravenous heparin 2,500–5,000 units just before clamping the aorta. It is also a wise precaution to pass a Fogarty catheter (p. 156) down the distal arteries to remove any extension thrombus which may have formed, just prior to closure of the arteriotomies. The midline incision in the abdomen is best closed by interrupted non-absorbable sutures.

Aorto-iliac By-pass (p. 150).—The abdomen is opened and the aorta and iliac arteries dissected as above. Heparin 2,500–5,000 units is given i.v. and the aorta and iliac arteries cross-clamped. The aorta is opened at a convenient point below the renal arteries, by a 5 cm longitudinal incision. After removal of mural thrombus a suitably sized (usually 16 × 8 mm) knitted Dacron bifurcation graft is selected.

The stem of the graft is stretched and divided so that the bifurcation of the graft will correspond with the bifurcation of the patient's aorta when the graft is inserted—this avoids any ' concertina-effect ' from too long a graft. The cut in the stem of the graft should be made obliquely to produce a ' comfortable lie ' and is sutured end-to-side with a continuous 000 silk or polypropylene suture to the aortic incision. A knitted graft is porous so it should be ' clotted ' by releasing the aortic clamp to fill it with blood, the clot being milked out thereafter. Disease-free segments of the external iliac arteries are selected and each is opened by a 1·5–2 cm longitudinal incision. When the graft and its proximal anastomosis are fully blood-tight, each limb of the graft is stretched and divided obliquely, so that the completed anastomoses are not under tension. The limbs of the graft are taken retro-peritoneally and anastomosed to the iliac arteriotomies by a 0000 continuous suture. On the left side this will necessitate taking the graft behind the sigmoid mesocolon. Fogarty catheterisation (p. 156) of the distal vessels may be performed just prior to completion of the anastomoses. If the external iliac arteries are diseased the limbs of the graft may be taken behind the inguinal ligament and anastomosed to the femoral arteries in the groin, which will have been displayed through separate inguinal incisions.

Femoro-popliteal vein by-pass grafting.—The patient is positioned and the incision made according to fig. 153 so as to expose the long saphenous vein, the femoral artery and its branches in the groin, and the popliteal artery below the obstruction (either above or below the knee). Once the limits of the arterial occlusion have been confirmed the long saphenous vein is divided at the sapheno-femoral junction. A suitable length of vein is then removed taking care to dissect and tie each tributary with fine silk ligatures. The graft should preferably be 5 cm or so longer than the length of the arterial obstruction to allow for wastage when fashioning the ends for anastomosis. The vein is fully distended to its maximal diameter using warm heparinised Hartmann's solution. A good vein will be 5 mm or more in diameter—veins smaller than this diameter give poor long-term results. 2 500–5 000 units of heparin is given i.v. and the common femoral and profunda femoris arteries cross-clamped. The common femoral artery is opened by a 2–3 cm longitudinal incision opposite the mouth of the profunda femoris artery. This should be inspected since stenosis of its ostium is common and not always visible on the standard views taken at arteriography (Martin). Ostial stenosis should be treated by progressive dilatation or by a local endarterectomy. The distal end of the long saphenous vein is cut obliquely and anastomosed to the femoral arteriotomy with a 0000 silk or polypropylene suture. Special care must be taken not to narrow the vein at this point or early graft failure will result. The graft is laid subcutaneously and its reversed proximal end bevelled and sutured to a 2–3 cm longitudinal arteriotomy in a disease-free area of popliteal artery. A good back-bleed should be present. If the distal anastomosis is made below the knee joint it is best to tunnel the graft through the popliteal fossa alongside the occluded popliteal artery. The knee should be straightened when measuring the length of graft required since a short graft under tension will result in early failure.

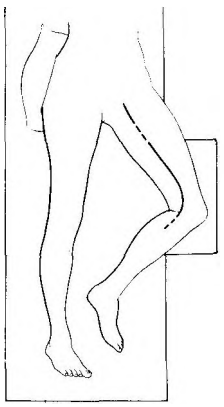


FIG. 153.—Position and incision for femoro-popliteal artery operations.

Femoro-popliteal thrombo-endarterectomy (disobliteration or rebore).—A longitudinal incision down to the outer layer of the media discloses a plane of cleavage and the core of atheroma and thrombus which can be dissected and lifted out. Short blocks can be removed by a single arteriotomy and the incision closed with a vein patch. Long blocks need to be freed with endarterectomy loops and dissectors via two or more incisions. Intimal tags and flaps must be tacked down.

Balloon Angioplasty.—Narrowed segments of atherosclerotic arteries can be dilated under radiological control using specially designed balloon catheters, with promising results, and the facility for repetition if required.

Results of Operation.—The long-term results of aorto-iliac reconstructive surgery are excellent. They are usually only marred by progressive disease

Peter Guy Cutlack Martin, Contemporary. Formerly Surgeon, Hammersmith Hospital, London.

producing femoro-popliteal occlusions at a later date. Femoro-popliteal surgery is less successful. The immediate post-operative success rate for vein by-pass approaches 90%. Many cases fail in the first eighteen months after operation and at the end of five years the success rate is usually only 50–60%. The results of thrombo-endarterectomy are less good in both short and long term and Dacron by-pass gives the poorest result with five year success rates of only 20–30%. The rather poor long-term results of femoro-popliteal surgery emphasise that these operations should only be used for clear indications.

Other Arterial Operations and Salvage Procedures

(i) **Profundaplasty.**—That the ostium of the profunda femoris artery is often stenosed is observed when oblique views are taken on arteriography. A combination of endarterectomy and a vein patch extending from the common femoral artery for some 3–5 cm along the profunda can be very effective (a) in enhancing a collateral circulation around an extensive femoropopliteal block and (b) in providing a good 'run off' below any procedure higher up when the superficial femoral artery is obstructed below. Thus it is a valuable routine and also a salvage procedure.

(ii) **Femoro-femoral crossover graft** is useful for relieving an iliac artery occlusion when the other iliac artery is patent with a strong femoral pulse. An 8 mm Dacron graft is tunnelled subcutaneously above the pubis and anastomosed end-to-side to the common femoral arteries on each side. Blood from the patent iliac system is then carried through this graft to vascularise the ischæmic limb.

(iii) **Axillo-femoral graft**—useful for salvaging a pre-gangrenous limb in a poor risk patient with bilateral iliac obstruction. A long 8 mm Dacron graft is tunnelled subcutaneously, from an end-to-side anastomosis with the axillary artery proximally, to reach the femoral artery of the involved limb in the groin where the distal anastomosis is made. The axillary artery will carry a sufficient volume of blood to maintain the circulation in the arm and re-vascularise the lower limb. The short-term results are usually good and in these patients with their poor general condition, the poorer long-term result is usually less important.

(iv) **Hitch-hike femoro-popliteal-tibial graft.**—A Dacron graft connects the common femoral artery to a rebored section of the upper popliteal artery. A vein graft connects the rebored section to arteries below the knee.

Salvage operations *should not* be performed for intermittent claudication alone. Gangrene and loss of limb may result if the operation should fail.

Adjuncts to Direct Arterial Surgery

(i) Lumbar sympathectomy in conjunction with direct arterial surgery, especially if the 'run-off' is poor. The other indications for sympathectomy are considered on p. 146.

(ii) Reserpine 1 mg by intra-arterial injection at the completion of the operation to promote vaso-dilatation and increased flow through the reconstruction during the first 2–3 days after operation.

(iii) Blood-flow estimations (by flowmeter), or on-table arteriography at the completion of the operation are useful techniques for ensuring that there have been no errors of surgical technique before the wound is closed.

Other Sites of Atherosclerotic Obstruction.—The principles of arterial surgery can be applied to other arteries which are stenosed by the disease.

Carotid stenosis causes transient, recurrent, and progressive 'strokes' (hemiplegia). On the contralateral side to the stroke, a carotid arteriogram will show either a local stenosis at the origin of the internal carotid artery, or a complete obstruction of that artery up into the skull, to where the ophthalmic artery enters as a collateral. Only a local stenosis is suitable for the disobliteration and vein-patch operation (fig. 154). Special measures may be necessary to prevent cerebral damage occurring while the

clamps are in place; postural hypertension (operating with the patient tilted head down) can be used, but a temporary silicone or polythene by-pass is more usual.

FIG. 154.—Carotid stenosis. A unilateral localised stenosis suitable for operation (see text). Also some narrowing of the external carotid. *N.B.* A rather similar double narrowing is seen in some carotid body tumours (p. 608).

Extra/Intracranial Bypass operations using microsurgical techniques to anastomose the superficial temporal to a superficial cerebral artery are now being used in selected cases. Neurological, psychological (psychometry), arteriographic, and flow studies are included in the location of the cerebral area most under threat of ischæmia.

Vertebral artery stenosis, causing basilar artery 'strokes' of giddiness and impairment of vision, may be demonstrated by arch arteriography (p. 140). Operation by widening of the lumen by vein-patch graft without removing the stenosis is sometimes performed.

Subclavian artery stenosis causes pain and a feeling of coldness in the arm and hand. Small patches of thrombus and atheroma break off as emboli, and occlude the arteries of the forearm, hand, and fingers. Resection and grafting, or disobliteration, may be performed.

Subclavian Steal Syndrome.—If the first part of the subclavian artery is obstructed, the vertebral artery provides collateral circulation to the arm, and its flow is reversed, as is demonstrated by arteriography. The cerebral circulation is diminished, hence the term 'steal'. Clinical features include syncopal attacks, visual disturbances, and diminished pulse and blood pressure on the affected side. A Dacron by-pass of the affected part of the subclavian artery, or thromboendarterectomy (rebore), relieves the symptoms. To avoid opening the chest the graft is usually taken from the common carotid artery of the affected side and anastomosed to the easily accessible third part of the subclavian artery.

Mesenteric Artery Stenosis.—Post-cibal central abdominal pain in a patient with atherosclerosis elsewhere may be shown by an arteriogram (lateral view) to be due to stenosis of the origin of the superior mesenteric artery. Usually the collateral circulation via the inferior mesenteric artery or cœliac artery has already been obstructed. The pain occurs so soon after food that the patient is afraid to eat. This classical syndrome is however a very rare cause of post-cibal pain. A by-pass graft is led from the aorta to the superior mesenteric artery, distal to the stenosis.

Renal artery stenosis may be responsible for some cases of hypertension (fig. 127). If the function of the kidney is satisfactory, the stenosis may either be disobliterated or by-passed (fig. 155).

Coronary artery disease.—See Chapter 41.

ARTERITIS

Thrombo-angiitis Obliterans (Buerger's disease):

This is a condition characterised by occlusive disease of the small and medium size arteries (plantars, tibials, radial, etc.), thrombophlebitis (fig. 167) of superficial or deep veins, and Raynaud's phenomena occurring in male patients in a young age group (usually under the age of 30 years). Usually one or two of the three manifestations are present and occasionally all three. The condition does not occur in women or non-smokers. It is not, as used to be stated, more common in Russian Jews. Cases are seen in different

Leo Buerger, 1879–1943. Professor of Urologic Surgery, New York Polyclinic Medical School, New York, U.S.A. Described Thrombo-Angiitis Obliterans in 1908.

races all over the world. Histologically, localised inflammatory changes occur in the walls of arteries and veins leading to thrombosis.

The usual symptoms and signs of arterial occlusive disease will be present. Progression to gangrene of toes and fingers is common.

Arteriography sometimes shows a characteristic 'corrugation' of the femoral arteries as well as the distal arterial occlusions and helps to distinguish the condition from pre-senile atherosclerosis.

Other forms of arteritis (e.g. polyarteritis nodosa) must be excluded.

Treatment.—The treatment is total abstinence of smoking, i.e. 'You can have your cigarettes or your legs, but you can't have both'! This will arrest the disease but not, or course, reverse established arterial occlusions. Reduction in smoking is not sufficient to prevent the otherwise relentless progression of this disease. Established arterial occlusions must be treated along the usual lines. Direct arterial surgery is not usually applicable and sympathectomy is the most useful surgical procedure. It often results in the healing of ischæmic ulcers and improvement in skin nutrition, with relief of pain. Amputations, conservative if possible, may be required.

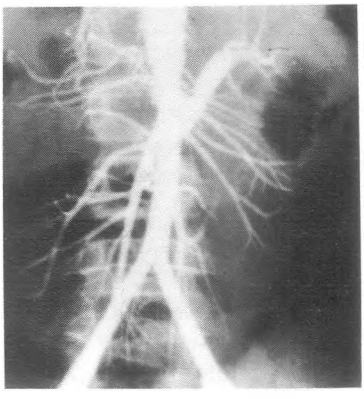


FIG. 155.—A by-pass vein graft from the aorta to the left renal artery in a little girl with bilateral renal artery stenosis, causing hypertension. The subclavian and other arteries were also occluded by Takayasu's arteriopathy (see text).

Other types of arteritis are encountered in rheumatoid arthritis, diffuse lupus erythematosis, and polyarteritis. Treatment is similar. *Diabetes* is discussed on p. 170. *Temporal arteritis or occipital arteritis*, which is a local collagen disease causing headache and sometimes retinal vessel changes, may be relieved when the tender thrombosed artery is excised for biopsy purposes. *Takayasu's arteriopathy* (obliterative arteritis of females, pulseless disease) causes narrowing and obstruction of major arteries and usually pursues a relentless course (fig. 155).

Cystic Myxomatous Degeneration.—Instances of an accumulation of clear jelly (like a synovial ganglion) in the outer layers of a main artery have been reported, e.g. the popliteal artery. The lesion so stiffens the artery that pulsation disappears, and claudication occurs when the limb is flexed (as on walking up stairs). Arteriography shows a smooth narrowing of an otherwise normal artery, and a sharp kink, or buckling, when the knee is flexed. Decompression, by removal of the myxomatous material, is all that is required, but the 'ganglion' may recur and require excision of part of the arterial wall and a vein-patch repair.

VASOSPASTIC CONDITIONS

Raynaud's disease usually occurs in young women, and affects the upper extremities more than the lower. The peripheral pulses are normal. The condition is attributable to abnormal sensitivity in the direct response of the arterioles to cold. When cooled, these vessels go into spasm, and as a result the part becomes blanched and incapable of finer movements. The decreased blood flow causes an accumulation of metabolites in the capillary

M. Takayasu, 1871– . A Japanese Ophthalmologist who studied in Tokyo, St. Thomas's Hospital, London and Breslau. (Conjoint M.R.C.S., L.R.C.P., 1896). Published paper on Takayasu's Disease in 1908.
Maurice Raynaud, 1834–1881. Physician, Hôpital Lariboisière, and Professor Agrégé, Paris. Published his thesis 'De L'Asphyie Locale et La Gangrène Symetrique des Extrémités' in 1862.

circulation. The capillaries dilate and become filled with slowly flowing deoxygenated blood, the part therefore becomes swollen and dusky. As the attack passes off, the arterioles relax, oxygenated blood returns into the dilated capillaries, the hands become red and a burning sensation or pain is produced by increase in tissue tension. Thus the condition is recognised by the characteristic sequence of blanching, dusky cyanosis and red engorgement. Eventually, in a minority of patients, obliterative changes may occur in the peripheral vessels, superficial necrosis occurs, the tips of the fingers undergo dry gangrene, and the distal parts of the terminal phalanges are absorbed. Early cases of Raynaud's disease must be distinguished from chilblains and vascular disturbances which are sometimes associated with the costoclavicular (cervical rib and the scalene) syndrome (Chap. 34).

Treatment.—*Conservative.*—Protection from cold, avoidance of pulp and nail-bed infections, and the use of vasodilator drugs are part of the conservative regimen that is advised for mild cases.

Sympathectomy.—The immediate results of sympathectomy are good, but after a few months the susceptibility to cold returns, although cyanosis is not so severe as before the operation, and subjective symptoms are less marked. This partial relapse is an indication that the underlying cause of Raynaud's disease is not in the sympathetic system, but is due to some abnormality in the smaller arteries and arterioles; sympathectomy apparently raises the threshold at which spasm occurs. From the practical point of view preganglionic section of the thoracic sympathetic chain can be recommended as a palliative procedure in Raynaud's disease, especially if performed before the onset of ulceration and absorption of the terminal phalanges.

Raynaud's Phenomenon.—Peripheral vasospasm also occurs secondarily to other organic diseases such as atherosclerosis, collagen diseases—especially scleroderma, Buerger's disease, cervical rib, etc. It also follows the use of industrial tools, e.g. pneumatic road drills and chain saws, which vibrate at certain frequencies. Also, in the arm, atherosclerotic stenosis of the subclavian artery (p. 162), scleroderma, Buerger's disease (p. 162), or cervical rib and the scalene syndrome (Chap. 34) may be responsible. Treatment is directed primarily to these causal lesions, though the conservative measures outlined above are often helpful. Sympathectomy may occasionally be required but it is not very helpful in scleroderma.

Raynaud's phenomena may also occur secondarily to atrophic changes in a limb, e.g. *after poliomyelitis, following trauma*, e.g. arthrodesis of a peripheral joint, crushing injuries, etc., in cases of *severe chilblains* (perniosis) and *following frostbite*. The mechanism of the cold sensitivity in these cases is obscure but in most instances good results follow sympathectomy.

Acrocyanosis—*crurum puellarum frigidum*[1] may be confused with Raynaud's disease, but it is painless and is not paroxysmal. Affecting young females, the cyanosis of the fingers, and especially the legs may be accompanied with paræsthesia and chilblains. In severe cases sympathectomy may be necessary, and gives good results. If merely affecting the calves, a differential diagnosis is Bazin's disease (p. 67).

[1] Puellarum = of girls, frigidum = ? frigid or just cold.

Sympathectomy

Preganglionic Cervico-dorsal Sympathectomy.—(*a*) *Supraclavicular Method.*—Through a supraclavicular incision, the clavicular part of the sternomastoid, the posterior belly of the omo-hyoid, and the scalenus anterior muscles are divided, the phrenic nerve being displaced inwards. The subclavian artery is exposed, and the thyro-cervical trunk is divided. The subclavian artery is depressed and the supra-pleural fascia is divided, so that the dome of the pleura can be displaced downwards. The stellate ganglion is identified as it lies on the neck of the first rib (fig. 156). The sympathetic trunk is traced downwards and divided below the third thoracic ganglion.

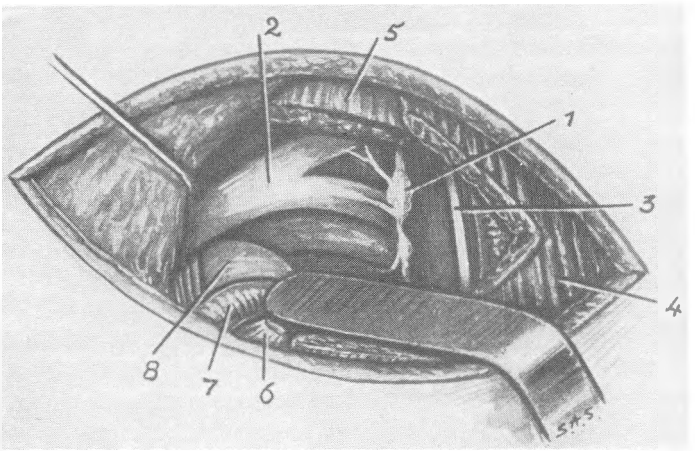


FIG. 156.—Exposure of the right cervicodorsal sympathetic chain from the front.

1. Stellate ganglion.
2. Lower trunk of brachial plexus.
3. Phrenic nerve displaced inwards.
4. Partially divided sternomastoid muscle.
5. Divided scalenus anticus muscle.
6. Divided posterior belly of the omo-hyoid.
7. Subclavian artery displaced downwards.
8. Dome of the pleura.

All rami communicantes associated with the second and third ganglia and the nerve of Kuntz, a grey ramus running upwards from the second thoracic ganglion to the first thoracic nerve, are meticulously divided. Occasionally the approach is under a high arching subclavian artery.

(*b*) *Transthoracic Method.*—This gives a greater exposure and facilitates the removal of the sympathetic chain from the fifth ganglion up to the lower fringe of the stellate ganglion. It tends to give better results than (*a*), and can be employed when (*a*) has failed. In women where cosmetic effects are a consideration, the approach can be made via an axillary incision through the third space (Hedley Atkins). The sympathetic chain is easily seen and after dividing the pleura, it is dissected out, care being taken to avoid damage to the intercostal vessels, which may cause tedious hæmorrhage. Care should also be taken, when making and suturing the approach wound, to avoid damage to the nerve to serratus anterior, giving rise to 'winging' of the scapula.

Lumbar Sympathectomy.—Using a transverse loin incision, an extra-peritoneal approach is used in which the colon and peritoneum, to which the ureter clings, are stripped inwards so as to expose the inner border of the psoas muscle (fig. 157).

The sympathetic trunk lies on the sides of the bodies of the lumbar vertebræ, and on the right side is overlapped by the vena cava. Lumbar veins are apt to cross the trunk superficially. The sympathetic trunk is divided on the side of the body of the fourth lumbar vertebra, and is traced upwards to be divided above the large second lumbar ganglion, which is easily recognised by the number of white rami which join it. Care should be taken not to mistake small lymph nodes, lymphatics, the genito-

Albert Kuntz, 1879–1957. Professor of Anatomy, St. Louis, Missouri, U.S.A.
Sir Hedley John Barnard Atkins, Contemporary. Past President, The Royal College of Surgeons of England. Emeritus Professor of Surgery, Guy's Hospital, London.

femoral nerve or the occasional tendinous strip of the psoas minor for the sympathetic chain.

Occasionally both sides are done at one session.

GANGRENE

Gangrene implies death with putrefaction of macroscopic portions of tissue. It is commonly seen affecting the distal part of a limb, the appendix, a loop of small intestine, and sometimes organs such as the gall-bladder, the pancreas, or the testis. Note that the term 'necrosis' applies mainly to the death of groups of cells, though it is extended to include bone, i.e. a 'sequestrum'. A 'slough' is a piece of dead soft tissue, e.g. skin, fascia, or tendon.

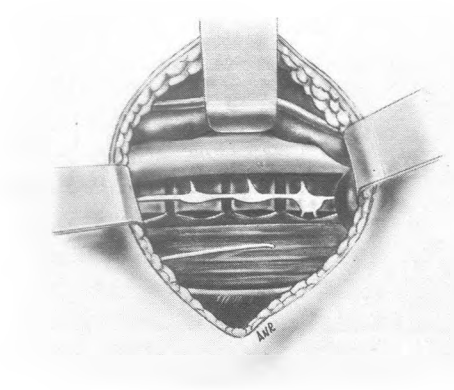


FIG. 157.—Left lumbar sympathectomy. The central retractor is holding back the peritoneum and ureter to expose the aorta and the psoas muscle, between which lies the sympathetic chain. Note that the second lumbar ganglion, adjacent to the lower pole of the kidney, has connecting rami to the spinal cord. The genitofemoral nerve is seen emerging through the fibres of the psoas.

VARIETIES OF GANGRENE ACCORDING TO CAUSE

1. **Secondary** to arterial obstruction from disease,[1] e.g. thrombosis of an atherosclerotic artery; embolus from the heart in auricular fibrillation or after coronary thrombosis; arteritis with neuropathy in diabetes; Buerger's disease; arterial shutdown in Raynaud's disease, and ergotism (fig. 160); the effect of intra-arterial injections—thiopentone and cytotoxic substances.

2. **Infective:** Boils and carbuncles, gas gangrene, gangrene of the scrotum.

3. **Traumatic:**

(*a*) *Direct*, such as crushes, pressure sores, and the constriction groove of strangulated bowel.

(*b*) *Indirect*, due to injury of vessels at some distance from the site of gangrene, e.g. pressure on the popliteal artery by the lower end of a fractured femur.

4. **Physical**, e.g. burns, scalds, frostbite, chemicals, irradiation and electricity.

5. **Venous Gangrene** (p. 173).

CLINICAL FEATURES OF GANGRENE (figs. 125 and 126)

A gangrenous part lacks arterial pulsation, venous return, capillary response to pressure (colour return), sensation, warmth, and function. The colour of the part changes through a variety of shades according to circumstances

[1] A classical mnemonic (aid to memory) for these causes is the word 'RESTED', signifying: Raynaud, Ergot, Senile, Thrombosis, Embolism, Diabetes.

(pallor, dusky grey, mottled, purple) until finally taking on the characteristic dark brown, greenish black, or black appearance which is due to the disintegration of hæmoglobin and the formation of iron sulphide.

Clinical Types.—Dry gangrene and moist (wet) gangrene.

Dry gangrene occurs when the tissues are desiccated by gradual slowing of the blood-stream, and typically occurs as a result of atherosclerosis, e.g. senile gangrene. The affected part becomes dry and wrinkled, discoloured from disintegration of hæmoglobin, and greasy to the touch.

Moist gangrene occurs when venous as well as arterial obstruction is present, when the artery is suddenly occluded, as by a ligature or embolus, and in diabetes. Infection and putrefaction are always present, and the affected part becomes swollen, discoloured, and the epidermis may be raised in blebs. Crepitus may be palpated, due to infection by gas-forming organisms. Moist gangrene is manifest also in such conditions as acute appendicitis and strangulated bowel.

SEPARATION OF GANGRENE

Separation by Demarcation.—A '*zone of demarcation*', between the truly viable and the dead or dying tissue, appears first. It is indicated on the surface by a band of hyperæmia and hyperæsthesia. Separation is achieved by the development of a layer of granulation tissue which forms between the dead and the living parts. These granulations extend into the dead tissue, until those which have penetrated farthest are unable to derive adequate nourishment. Ulceration follows, and thus a '*final line of demarcation (separation)*' forms which separates the gangrenous mass from healthy tissue.

In dry gangrene, if the blood supply of the proximal tissues is adequate, the final line of demarcation appears in a matter of days and separation begins to take place neatly and with the minimum of infection (so-called separation by aseptic ulceration). Where bone is involved complete separation takes longer than when soft tissues alone are affected, and the stump tends to be conical as the bone has a better blood supply than its coverings.

In moist gangrene there is more infection, and suppuration extends into the neighbouring living tissue, thus causing the final line of demarcation to be higher than in dry gangrene (separation by septic ulceration). This is why dry gangrene must be kept as dry and aseptic as possible, and every effort should be made to convert moist gangrene into the dry type.

Vague Demarcation. Spread of Gangrene.—In many cases of gangrene from atherosclerosis and embolism, the line of final demarcation is very slow to form or does not develop. Unless the arterial supply to the living tissues can be improved (pp. 135–145), the gangrene will spread to adjacent tissues or toes, or will suddenly appear as 'skip' areas further up the limb. Signs of skipping should always be carefully looked for. Black patches suddenly appear, perhaps on the other side of the foot, on the heel, on the dorsum of the foot, in front of the ankle, and in the calf. *Infection*, being another cause of the spread of gangrene, may spread upwards beyond the line of separation along the lymphatic vessels or

cellular tissue into healthy parts, and extensive inflammation then results. Except in diabetic gangrene without concomitant atherosclerotic obstruction, these forms of spread do not usually respond to efforts to save the limb, and an above-knee amputation is then necessary. To attempt local amputation in the phase of vague demarcation is to court failure, as gangrene reappears in the skin-flaps (die-back).

TREATMENT OF GANGRENE

General Principles.—*A limb-saving attitude* is needed in most cases of symptomatic gangrene affecting hands and feet. The surgeon is concerned with how much can be preserved or salvaged.[1] With arterial disease, so much depends upon there being a good blood supply to the limb above the gangrene, or whether a poor blood supply can be improved by such measures as direct arterial surgery (p. 146) or interruption of the sympathetic nerve supply (p. 146). A good or improved blood supply indicates that a conservative excision is likely to be successful and a major amputation may be avoided. *A life-saving amputation* is required for a badly crushed limb, rapidly spreading symptomatic gangrene, and gas gangrene.

General treatment includes that of cardiac failure, atrial fibrillation, and anæmia, in order to improve the tissue oxygenation. A nutritious diet, essential in all forms of gangrene, and the control of diabetes when present, are additional items of care. Pain, especially night pain, may be difficult to relieve. Non-addictive drugs should be used whenever possible.

Local Treatment.—(*a*) *Care of the affected part* includes keeping it absolutely dry. Exposure, and the use of a fan may assist in the desiccation and may relieve pain. *The limb must not be heated.*

(*b*) *Protection of local pressure areas*, e.g. the skin of the heel or the malleoli, otherwise fresh patches of gangrene are likely to occur in these places. A bed-cradle, padded or Sorbo rings, foam P.V.C. blocks, or a 'sheepskin', (Acrilan)[2] are useful preventive aids.

(*c*) *Minor Surgical Toilet.*—Careful observation of a gangrenous part will show whether the lifting of a crust, or the removal of hard or desiccated skin, will assist in demarcation, the release of pus and the relief of pain.

Amputations of the Leg for Gangrene.—These may be conservative, radical (definitive) and semi-conservative (compromise), and as stated above, the indications depend upon the blood supply of the part of the limb immediately adjacent.

(*a*) *Conservative.*—There need be no undue haste to remove a dry, painless, gangrenous toe when the final line of demarcation is satisfactory. A diabetic or frostbitten part can in theory be left to separate. An excision through the line of demarcation is usually performed when separation is proceeding well. These wounds are left open to heal by granulation (fig. 158). If bone and tendons are to be sectioned at a higher

[1] Sir William Fergusson (1808–1877), Surgeon, Royal Infirmary, Edinburgh, later Professor of Surgery, King's College Hospital, London, said that amputation is 'one of the meanest, and yet one of the greatest operations in surgery: mean, when resorted to where better may be done—great, as the only step to give comfort and prolong life'.

[2] A sheepskin or a large pelt made of washable fibre ('Acrilan' pad made by Northpads, London). The air pockets in wool make it specially resilient to pressure, so these pads may be used wherever areas need protection. The idea stems from cattle and sheep ranches where saddle-sores on horses are prevented by the interposition of a sheepskin.

level, this can be done through the cuff of skin and soft tissue. Special flaps are not fashioned in conservative amputations unless a portion of a foot or the forefoot is to be removed. A trans-metatarsal amputation (fig. 159) is only possible when there is

FIG. 158.—Conservative amputation for diabetic gangrene.

FIG. 159.—Trans-metatarsal amputation for diabetic gangrene of the toes.

a viable long plantar flap extending almost to the base of the toes. This must be wrapped round the severed forefoot and sutured to the dorsal skin. A long dorsal flap cannot withstand the subsequent trauma from a shoe.

(*b*) *Radical* (*Definitive*).—Where conservative amputation is impossible because of failure of demarcation, spread of gangrene, failure of direct arterial salvage surgery or sympathectomy, a mid-thigh amputation is the classical method of treatment. There is no statutory length of the stump; it is, in fact, essential to amputate high enough to ensure that the wound will heal soundly. This usually means a bony stump of some 15 to 20 cm. below the greater trochanter. Equal anterior and posterior flaps of skin and subcutaneous tissue and deep fascia are used. The muscles are divided just below the level of bone section (quadriceps divided with the knee flexed, and hamstrings with the knee extended). A tourniquet is not used to control hæmorrhage in atherosclerotic and embolic gangrene as there is surprisingly little bleeding owing to the obstructed main vessels. The sciatic nerve is cut cleanly across. It should not be ligated, though usually there is brisk bleeding from the artery to the nerve, which requires a ligature.

After sawing across the bone the leg is removed and full hæmostasis is secured. The *myoplastic* technique consists of suturing a flap of periosteum across the severed bone, and separating the muscles into anterior, posterior, medial and lateral bunches. The anterior and posterior groups are sutured to each other across the bone end and the medial and lateral groups to each other across these. A subcutaneous catgut suture may be inserted and the skin closed by a subcuticular suture of catgut or Dexon. The stump should be drained, the 'Readivac' suction method being convenient.

(*c*) *Semi-conservative* (*Compromise*) *Amputations.*—In many cases amputations below the knee, through the knee, or supracondylar (Stokes-Gritti) will heal well. The functional result of these amputations is usually much better than the classical above knee amputation. Classically, below knee amputations were performed with equal length anterior and posterior skin flaps, and through knee and supracondylar amputations with a long anterior skin flap. It is now realised that in atherosclerotic limbs the posterior skin has a much better blood supply, and long posterior skin flaps are now used to close all three types of amputation with greatly improved results.

After-care.—The stump is immobilised between sandbags for forty-eight hours, and there should be adequate sedation for pain. Thereafter the patient is encouraged by a programme of promotion to a full and active participation in the community. This programme entails exercises, sitting up in a chair, getting up on crutches, and finally the measurement for, fitting of, and rehabilitation with, an artificial limb (prosthesis). Early dressing and the removal of the drain require sedation, and an

Sir William Stokes, 1839–1900 (*see p. 100*). *Rocco Gritti, 1857–1920. Surgeon, Milan.*

assistant to hold the limb gently but firmly between his hands. When the wound of an above- or below-knee amputation has healed, the patient holds his limb himself while he is taught how to bandage the stump in such a way as to mould it into the cone-shape necessary for fitting the prosthesis. Removal of the sutures on the fourteenth day depends upon satisfactory healing, and if there is any doubt on this score, they should be left in for three or even four weeks. Catgut or Dexon sutures have the advantage of not requiring removal.

Complications.—*Early*, include the following: reactionary hæmorrhage, which requires return to the theatre for operative hæmostasis; a hæmatoma, which requires evacuation; infection, usually from a hæmatoma. Any abscess must be drained. Depending upon the sensitivity reactions of the organisms cultured, the appropriate antibiotics are given. Gas gangrene can occur in a mid-thigh stump—the organisms coming from contamination by the patient's fæces (p. 33).

Wound dehiscence and gangrene of the flaps are due to ischæmia, and a higher amputation may well be necessary. Fat embolism (p. 156) is another but uncommon sequel, occurring within the first two days.

Late.—Pain is usually the presenting symptom, due to: unresolved infection, e.g. a sinus, osteitis, sequestrum (even a complete 'ring' sequestrum); a bone spur; a scar adherent to bone; an amputation neuroma from the outgrowth of nerve fibrils which become attached to skin, muscle or fibrous tissue; a phantom limb.

Phantom Pain.—Patients frequently remark that they can feel the amputated limb and sometimes that it is painful. The surgeon's attitude should be one of reassurance that these feelings will disappear. He should on no account foster the subject and talk about phantom-limb pain in front of the patient, as it is very refractory to treatment once it is established.

Other late complications include ulceration of the stump (pressure effects of the prosthesis or increased ischæmia). Rarely, an ulcer is artefacta (fig. 66). Some patients are troubled by cold and discoloured stumps, especially during the winter.

ASPECTS OF CERTAIN VARIETIES OF GANGRENE

Ergot, a cause of gangrene among dwellers on the shores of the Mediterranean Sea and the Russian steppes who eat rye bread infected with *Claviceps purpurea*, also occurs in migraine sufferers, who, for prophylactic reasons, unwittingly take ergot preparations over a long period. Overdosage can also occur following the ergot treatment of post-partum hæmorrhage. The fingers, the nose and ears may be affected (fig. 160).

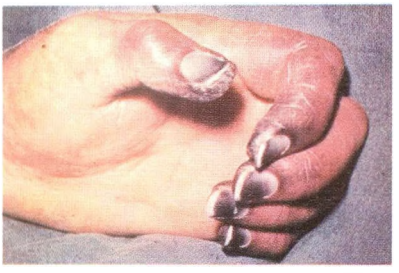


FIG. 160.—Gangrene due to ergot. The patient had taken repeated doses of ergotamine tartrate for 'migraine' while on a transatlantic flight. *(Professor Dame Sheila Sherlock, London.)*

Diabetic gangrene is due to three factors. These are (i) trophic changes resulting from peripheral neuritis, (ii) atheroma of the arteries resulting in ischæmia, and (iii) excess of sugar in the tissues which lowers their resistance to infection (fig. 161) including fungal infections.

The neuropathic factor impairs sensation, and thus favours the neglect of minor injuries and infections, so that inflammation and damage to tissues are ignored. Muscular involvement is frequently accompanied by loss of reflexes, and deformities. In some cases the feet are splayed and deformed (neuropathic joints). Thick callosities develop on the sole, and are the means whereby infection gains entry, often following amateur chiropody. Infection involving fascia, tendon, and bone can spread rapidly upwards via subfascial planes.

Clinical examinations and investigations include those on the urine and blood for diabetes. Palpable dorsalis pedis and posterior tibial pulses, and the absence of rest pain and intermittent claudication, imply that there is no associated major arterial disease (atherosclerosis). A bacteriological examination is made of any pus. An X-ray will reveal the extent of any osteomyelitis.

FIG. 161.—Diabetic gangrene.

TREATMENT.—The diabetes must be brought under control by diet and the appropriate drugs (e.g. Insulin, Tolbutamide, etc.). The gangrene is treated on the lines already described on the previous pages, the accent being on conservatism if there is no major arterial obstruction. A rapid spread of infection requires drainage of the area by incision and the removal of any obviously dead tissue. This may often involve free and extensive laying open of infected tissue planes. Adequate surgical drainage of pus and the control of infection due to bacteria and fungi may then be followed by rapid healing.

TRAUMATIC GANGRENE

(i) **Direct traumatic gangrene** is due to local injury, and may arise as a result of crushes, pressure (as in the case of splints or plasters), or bedsores.

Gangrene following severe injury, e.g. a street accident in which a heavy vehicle passes over a limb, is of the moist variety (p. 167) and removal without delay is usually indicated. Amputation may be performed as close to the damaged part as will leave the most useful limb.

Bedsores (*syn.* decubitus ulcers) are either acute or chronic. The acute or *trophic* variety is associated with disease or injury of the spinal cord, and often progresses with alarming rapidity in spite of every care and attention.

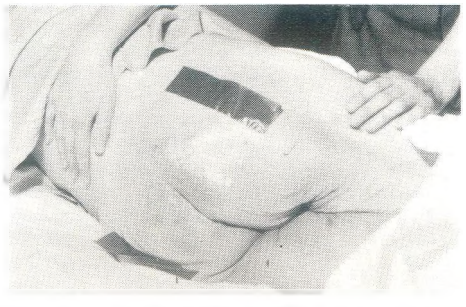


FIG. 162.—Opsite being applied to combat an incipient bedsore.

The chronic or *postural bedsore* is predisposed to by five factors—pressure, injury, anæmia, malnutrition, and moisture. Prophylactic treatment is of the utmost importance. Thus pressure over bony prominencies is counter-acted by a two-hourly change of posture and protection by foam P.V.C. blocks, or a 'sheepskin' (footnote, p. 168). A water-bed or a ripple bed is sometimes desirable. Injury due to wrinkled drawsheets and maceration of the skin by sweat, urine, or pus is

combated by skilled nursing and the use of an adhesive film such as Opsite (fig. 114 and p. 22).

A bedsore is to be expected if erythema, which does not change colour on pressure, appears. The part must be kept dry. An aerosol silicone spray may be used. Actual bedsores may either be treated by lotions (p. 65) or by exposure to keep them as dry as possible, and the appropriate antibiotic given systemically. The hæmoglobin of the patient should be maintained at a normal level by transfusions of packed cells if need be. If the patient is young and otherwise healthy, excision of the dead tissue and flap pedicle skin grafting (p. 133) is often successful.

(ii) **Indirect traumatic gangrene** is due to interference with blood-vessels:—

(*a*) From pressure by a fractured bone in a limb, or by strangulation (fig. 163) (strangulated hernia, Chap. 54).

(*b*) Thrombosis of a large artery, following injury.

(*c*) Ligation of the main artery of a limb, as after division by injury.

FIG. 163.—Indirect traumatic gangrene of a new-born infant's finger, accidentally caused by a thread of cotton.

The likelihood of gangrene depends upon the sufficiency of the collateral circulation (p. 135).

Treatment directed to the cause, e.g. closed or open reduction of a fracture together with direct arterial surgery for the damaged vessel, will usually prevent the onset of gangrene. The limb must be kept cool, so as to reduce metabolism to the minimum (p. 144). When gangrene is slow to develop, a line of demarcation will indicate the level of vitality. If moist gangrene occurs and spreads rapidly, amputation may be needed to save the patient's life.

PHYSICAL AND CHEMICAL CAUSES OF GANGRENE

Frostbite is due to exposure to cold, especially if accompanied by wind or high altitudes (e.g. explorers and airmen). It is also encountered in the elderly or the vagrant during cold spells (fig. 164). Pathologically, there is damage to the vessel walls, which is followed by transudation and œdema. The sufferer notices severe burning pain in the affected part, after which it assumes a waxy appearance and is painless. Blistering, and then gangrene, follows.

Treatment.—Frostbitten parts must be warmed *very gradually*. Any temperature higher than that of the body will be detrimental. Many frostbitten limbs have been either stewed or roasted in ignorance, and gangrene thereby encouraged. The part should be wrapped in cottonwool and kept at rest. Friction, e.g. rubbing with snow, may damage the already devitalised tissues. Warm drinks and clothing are provided and powerful analgesics are required to relieve the pain which heralds the return of circulation. Paravertebral injection of the sympathetic chain (p. 143) may be helpful in relieving associated vasospasm. Amputations should be conservative (p. 168). Hyperbaric oxygen (p. 85) is effective.

Trench foot is due to cold, damp, and muscular inactivity, and is predisposed to by tight clothing, such as garters, puttees, or illfitting boots. Prophylaxis is therefore

of paramount importance. Numbness is followed by pain, which is excruciating when boots are removed. The skin is mottled like marble, and in severe cases blisters containing blood-stained serum develop, and moist gangrene follows. The pathology is similar to that of frostbite, and the treatment is the same.

Ainhum (fig. 165), a disease of unknown ætiology, usually affects male Negroes, (but some females), who have run barefoot in childhood. Besides Central Africa there are reports from Central America and the East. A fissure appears at the level of the interphalangeal joint of a toe, usually the fifth. The fissure becomes a fibrous band, which encircles the digit and causes necrosis. The treatment is either early Z-plasty or, later, amputation.

Intra-arterial injection of thiopentone is an occasional accident when a high division of the brachial artery results in one of its two terminal branches, usually the ulnar, passing superficially downwards in the antecubital fossa. Pulsation of the vessel, and the withdrawal of bright red blood should prevent this calamity.

FIG. 164.—Frostbite in Fulham, London. An elderly patient, living without sufficient food or heating. Conservative amputations were successful.

FIG. 165.—Ainhum (see text).
(*Dennis Morrissey, F.R.C.S., Birmingham.*)

Injection causes immediate and severe burning pain and blanching of the hand, usually accompanied by an interjection from the patient! The needle should be left in position, and 5 ml. of 1 per cent. procaine and/or 2 per cent. papaverine sulphate, or 1 mg reserpine injected in order to obviate vascular spasm. Dilute heparin solution (p. 154) may also be given intra-arterially if the needle is in position. If the needle is not in position, and the facilities are available, the artery may be exposed, opened, and any clots flushed out with dilute heparin solution. Then vein-patch repair (p. 146), and/or peri-arterial papaverine, or intra-arterial fibrinolysins (p. 145) and intravenous low molecular weight Dextran (p. 81) may be employed. Brachial block must also be performed, and repeated if necessary. Even so, gangrene of one or more fingers may occur.

Chemical Gangrene.—Carbolic acid is the most dangerous, as anæsthesia masks the pain which occurs before the onset of gangrene. *Carbolic compresses should never be used*, for fingers have been lost by application of compresses even as dilute as 1 : 80. The gangrene is due to local arterial spasm. In addition there is danger of severe systemic effects from absorption of the carbolic. Local bicarbonate soaks should be applied. Later, excision of the slough and skin grafting are necessary.

VENOUS GANGRENE

A rare condition, due to extensive thrombosis in peripheral veins, there being normal arterial pulsations. It may occur in the toes and forefoot without any apparent cause, though it may be a manifestation of visceral neoplasm (Trousseau's sign, p. 175). It also occurs in peripheral thrombosis due to polycythæmia vera (fig. 166).

Treatment.—The limb is elevated and anticoagulant therapy commenced (p. 154). Low molecular weight Dextran i.v. to reduce the viscosity of the blood may be

FIG. 166.—Venous gangrene due to polycythæmia vera.

helpful. Conservative surgery is performed when the final line of separation appears. It may be found that the gangrenous tissue is relatively superficial and that the deeper layers are pink and viable. Skin grafting may be helpful in this conservative approach to treatment.

CHAPTER 11

VEINS

VENOUS THROMBOSIS

Thrombosis of veins is predisposed to by Virchow's triad:

(1) *Change in the vessel wall* with damage of the endothelium, e.g. inflammation or injury.

(2) *Diminished rate of blood flow*, as occurs during and after operations, and in debilitating conditions, such as typhoid fever.

(3) *Increased coagulability of the blood*, such as occurs in infections, after hæmorrhage and with visceral carcinoma (Trousseau's sign).

The results of thrombosis are as follows:

(1) *Locally*.—The clot may organise into fibrous tissue, or the vein becomes recanalised. Calcification in the form of a phlebolith is seen in pelvic veins. Infection may lead to abscess formation or pyæmia.

(2) *Distally*.—After a varying degree of œdema a venous collateral circulation soon opens up, as shown by the appearance of tortuous superficial veins (fig. 168).

(3) *Proximally*. — Extension into larger veins where portions of clot may become detached and, as emboli, cause pulmonary infarction (Chap. 40). Infected clot in the portal vein will cause liver abscesses (pylephlebitis, Chap. 45).

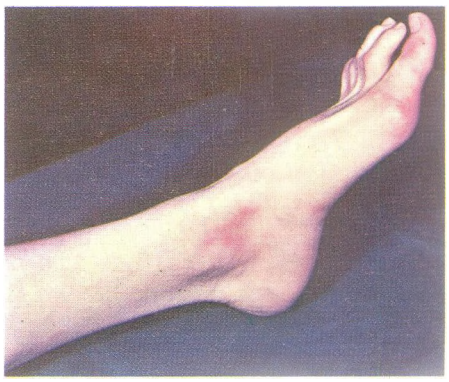


FIG. 167.—Spontaneous thrombophlebitis in Buerger's disease (p. 162).

Superficial Vein Thrombosis (**Thrombophlebitis**) commonly occurs in varicose veins or a vein which is cannulated for an infusion (p. 96). A painful and cord-like inflamed area is diagnostic. Spontaneous thrombophlebitis, including a migratory type (thrombophlebitis migrans), heralds the presence of such conditions as Buerger's disease (fig. 167), a visceral carcinoma, polyarteritis and polycythæmia. The treatment includes gentle support by means of a crepe bandage (and see p. 180), with a short 5-day course of penicillin or cotrimoxazole. An anti-inflammatory drug (e.g., aspirin or indomethacin) may be helpful.

As the thrombus is adherent to the wall of the vein an embolus is unusual but, if infection spreads rapidly up the vein, an emergency proximal ligation is necessary.

Rudolph Virchow, 1821–1902. Pathologist, Germany.
Armand Trousseau, 1801–1867. Physician, Hôtel-Dieu, and Professor of Medicine, Paris. Noted this sign as his own death warrant, as his own thrombosis confirmed his suspicion that he had carcinoma of the stomach.

Deep Vein Thrombosis (**Phlebothrombosis**[1]) follows childbirth, operations, muscular violence, local trauma of any kind, immobility, and any debilitating illness. Spontaneous thrombosis may also be indicative of the presence of a visceral neoplasm (see superficial vein thrombosis). Post-operative thrombosis, virtually unknown in equatorial regions such as Singapore, is believed to occur more frequently with the arrival of snow and ice.

The thrombus may commence in a venous tributary of a main vein, where there are eddying currents around a valve. It extends in a serpentine fashion into the main deep vein where the relatively faster stream may cause a portion to break off and so cause a pulmonary embolus (Chap. 40). The pelvic and calf veins are more commonly implicated than others. Occlusion of a length of the deep femoral vein will cause painful congestion and œdema of the leg. If in addition there is an associated lymphangitis, the swelling will increase and is likely to be protracted (e.g. ' white leg '—phlegmasia alba dolens). Extensive deep vein thrombosis of the iliac and pelvic veins may cause a ' blue leg ' (phlegmasia cærulea dolens) in which either venous gangrene or areas of infarction may threaten part of or the whole of the limb (p. 173).

Prophylaxis.[2]—*Before operation.* When possible, grossly over-weight patients should reduce weight. Those over forty, rendered immobile during a period of in-patient investigation or other treatment, are less at risk if they have a spell of 2–3 weeks activity at home before readmission for operation. Patients with cancer are particularly at risk.

During operation it is essential that the venous return from the lower limbs is not impeded in any way. Pressure of the calf on the operating table *must* be prevented by elevating the heel on a sandbag or sorbo-rubber pad. At the end of an operation it is a good plan to elevate and massage the legs.

Mechanical methods of prophylaxis include:—(i) *Graduated static compression* of the leg using specially constructed elastic stockings (Kendall's thrombo-embolic deterrent). These exert diminishing pressure on the leg from the ankle upwards to the thigh. Other methods of maintaining a satisfactory flow of blood in the deep veins include (ii) *Electrical stimulation* of the calf muscles and (iii) *Pneumatic compression.*

Dextran ' 70 ' inhibits platelet adhesion. A useful regimen is to give 500 ml i.v. during the operation, and 500 ml in the following 24 hours.

Prophylactic anticoagulants.—Subcutaneous heparin, 5 000 units, is given 2 hours before operation and continued 8 hourly for 7 days. It is of value in major surgical cases providing the operation does not involve leaving raw bleeding areas. It is contraindicated where a small postoperative hæmorrhage in a restricted area would be disastrous. Intravenous heparin may be used in cases where there is a high risk of thrombosis and a low to medium risk of bleeding (Browse).

After operation, conditions which predispose to a sluggish circulation such as immobility of the lower limbs, dehydration, and delayed venous return should be avoided by massage, leg movements, continued use of graduated

[1] As there is likely to be as much inflammation around a deep vein as a superficial vein, the distinction given by the terms thrombophlebitis and phlebothrombosis is really inappropriate.

[2] When there is no urgency, female patients on the ' pill ' should stop this method of contraception for one month before surgery.

Kendall Company, Chicago, Illinois, U.S.A.
Norman Leslie Browse, Contemporary. Professor of Vascular Surgery, St. Thomas's Hospital, London.

compression stockings, adequate hydration, and early ambulation. Patients should not be allowed to sit out of bed with their legs dependent. In fact, patients are often more mobile in bed than in a chair.

Detection of Thrombosis.—Phlebothrombosis is often symptomless,[1] but there may be a complaint of pain in the affected calf. Any unexplained elevation of temperature or pulse-rate should arouse suspicion of thrombosis, especially when occurring towards the end of the first post-operative week. In all cases the calves should be examined daily for tender areas of induration. Tenderness may be elicited along the course of the posterior tibial and peroneal veins. Slight swelling of the calf muscles is another important sign. Homans' sign—pain in the calf or dorsiflexion of the foot may be present but it is rather unreliable.

The Doppler principle (p. 142) is a useful and simple aid (Sonic-aid) in the diagnosis of deep vein thrombosis. The sensing probe, placed over the femoral vein in the groin normally transmits a venous hum (like wind in telegraph wires) and pressure on the calf, or calf muscle contractions, changes the hum into a roar, due to increased blood flow. If there is a thrombosis of the deep vein between calf and groin the roar does not occur. A deep venogram is used sometimes to show the site and extent of the obstructing thrombus (pp. 182, 183). Browse believes a venogram to be essential to diagnosis and to the formulation of the correct treatment policy. However, caution should be exercised in patients who are inveterate clotters, as venous gangrene has been known to occur after this investigation.

Treatment of Deep Vein Thrombosis.—(*a*) *Anticoagulants, Bandage and Rest.*—A suitable regimen includes (1) Giving anticoagulants, combining heparin and phenindione (or Warfarin, p. 155) and continuing the latter for three to six weeks taking care to tail-off slowly to prevent a ' rebound ' of thrombosis. (2) Bandage the whole limb with crepe or use a graduated compression stocking to increase the deep venous flow. (3) Rest the patient until the elevated temperature and the local signs abate, and then begin mobilisation.

(*b*) *Treatment based on venography.*— (i) *Fixed thrombus.*—If the thrombus is fixed to the vein wall, anticoagulants are given. Heparin 40 000 units per day for 7 days are given, if possible, by continuous infusion (fig. 79). Warfarin is begun (p. 155) and continued for 6 months. (ii) *Free thrombus.*—If the thrombus is loose at the top it should be either removed or ' fixed '. Removal is achieved by thrombectomy, using a Fogarty balloon venous catheter (p. 156), or by streptokinase (providing there is no bleeding and no heparin is given). Fixation is achieved in reality by blocking the vein. A ligature suffices for the superficial (not main) femoral vein, but for the vena cava a ligation, or the introduction of a filter, may be carried out. One such filter is the Kimray-Greenfield, introduced in a capsule, under X-ray control, via the neck and internal jugular vein into the inferior vena cava.

Axillary Vein Thrombosis.—Thrombosis of the axillary vein is not uncommon, and can occur after unaccustomed use of the corresponding arm (e.g. distempering a ceiling). The vein is damaged by the excessive movement

[1] Studies, using isotope-labelled fibrinogen, have indicated that symptomless thrombosis is relatively common.

John Homans, 1877–1955. Professor of Clinical Surgery, Harvard Medical School, Boston, Massachusetts, U.S.A. Described this sign in 1941.
Kimray Medical Associates, Oklahoma City, U.S.A.
Laser J. Greenfield, Contemporary. Surgeon, Oklahoma, U.S.A.

that occurs between the clavicle and the first rib.[1] 'Spontaneous' thrombosis may be due to a pyjama sleeve becoming twisted around the axilla during heavy slumber, with consequent compression of the vein. The thrombosis results in painful congestion and œdema of the arm (fig. 168). As a collateral venous circulation develops, the symptoms subside; this takes about three months. The extent of the thrombosis can be limited if anticoagulant therapy is given in the early stages. Fibrinolytic therapy may restore the circulation if given in the first 2 or 3 days (p. 155).

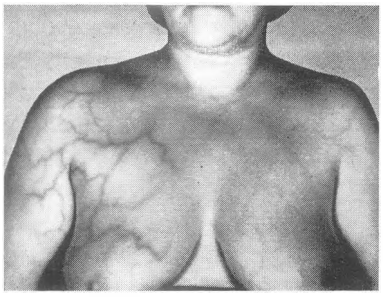


FIG. 168. — Infra-red photograph showing collateral venous circulation following thrombosis of the axillary vein. (*Max Pemberton, F.R.C.S., Enfield, London.*)

VARICOSE VEINS

A vein is stated to be varicose when it is dilated and tortuous. The condition commonly occurs in connection with the veins of the leg (fig. 169), but also the spermatic, œsophageal, and hæmorrhoidal veins may be affected. The three last conditions are dealt with elsewhere.

Ætiology.—Varicose veins are part of the penalty we pay for the adoption of the erect posture. Animals do not suffer from this condition.

The Venous Pump.—In the human, the return of venous blood from the lower limb to the heart requires a pump equipped with non-return valves. The pumping action is provided by the muscles. Their tone and contractions, acting within the strict confines of the encircling deep fascia, squeeze or milk the blood in the direction insisted upon by the valves, i.e. towards the heart. Therefore a congenital paucity of valves, muscle weakness and wasting, or deep fascia stretching, will impair the function of the pump. On standing, the whole weight of the column of blood from the legs to the heart is exerted on the valves (up to pressures of 90 mmHg (12·0 kPa)), particularly on those guarding the communications between the superficial and deep venous systems of the leg (blood in the superficial system normally flows into the deep veins). The main valves affected are the sapheno-femoral lying at the junction of the long saphenous and common femoral veins (leading to dilatation of the long saphenous system), and the sapheno-popliteal (leading to dilatation of the short saphenous

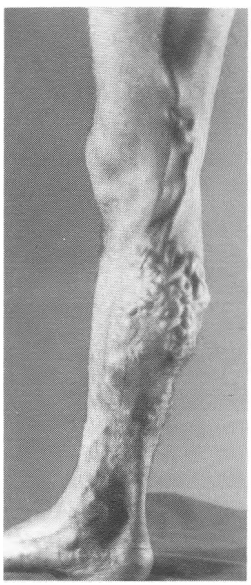


FIG. 169.—Varicose veins.

[1] Henry of Navarre, 1553–1610 (King Henry IV of France, 1589–1610), led his army into the battle of Ivry (1590), shouting 'Rally round the white plume of Navarre'. He used his sword to such good effect that he could not use his arm for six weeks, and probably had an axillary vein thrombosis.

system). However, varices may commence at the site of any incompetent communicating vein perforating the deep fascia, either alone or in conjunction with one of the above (fig. 170).

When these valves become incompetent, there is a high, intermittent pressure leakage of blood into the superficial system (fig. 171). The main superficial veins are partially protected by the superficial fascia and they possess muscular walls which in some cases prevent the varicosity which affects mainly the intercommunicating network of veins.

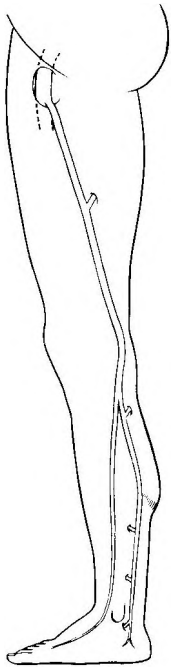


FIG. 170.—The sites of communicating (perforating) veins between the deep and superficial (long saphenous) veins. The short saphenous vein has similar communications on the lateral side of the Achilles tendon.

Varicosity is also predisposed to by any obstruction which hampers venous return, e.g. tumours and pregnancy, and by thrombosis of the deep veins. The predilection for females suggests a hormonal (? progesterone) factor.

Varicose veins occurring below the age of twenty may be due to congenital arteriovenous fistula, or an extensive cavernous (venous) hæmangioma.

In severe cases of varicose veins the blood volume is increased. It is reduced by operation, so there is a reduction of symptoms and an improvement of ability to perform physical work.

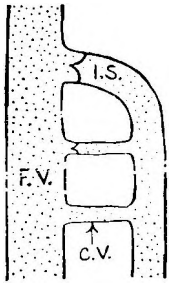


FIG. 171.—An incompetent communicating vein (C.V.), femoral vein (F.V.), long (internal) saphenous vein (I.S.).

Symptoms occur in connection with varicose veins only when there is a retrograde flow, and they depend on the extent of the back pressure. The commonest is a tired and aching sensation, felt in the whole of the lower leg, and especially in the calf, towards the end of the day. Sharp pains, when present, are localised to the site of the varices, and are especially noticeable in grossly dilated thigh veins. The ankle may swell towards evening, or the skin of the leg may itch. Some patients suffer from cramp in the calf shortly after retiring to bed[1].

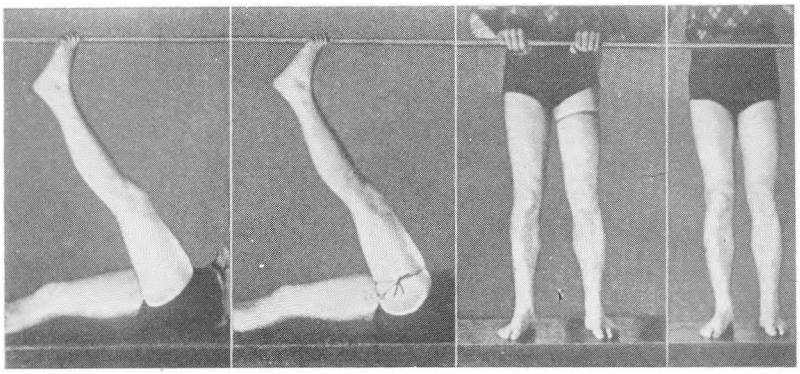
Examination.—The condition may be widespread in both legs, or a single varix is sometimes present. If this is situated close to the saphenous opening, it is called a 'saphena-varix',[2] and it is readily distinguished from a femoral hernia on account of the characteristic thrill when the patient coughs, or if the vein below is tapped with a finger.

[1] Quinine dihydrochloride (200 mg), taken on retiring, is often efficacious in relieving night cramps. Hexopal 500 mg has also helped some patients.

[2] The word 'saphenous' is of Arabic derivation and means 'seen easily'.

The examination of the varices is most important, for upon it depends the success, or failure, of treatment. The aim is to locate the site of the incompetent superficial-deep valves (fig. 170), remembering that if an incompetent valve is present the venous flow is retrograde, so that veins when emptied fill from above; normally they fill from below. The examination, based upon the test described by Brodie (1846) and Trendelenburg (1890), involves what has aptly been described as ' the intelligent use of the tourniquet '.

Brodie-Trendelenburg Test.—Briefly, the patient lies upon his back and raises his leg to empty the veins (fig. 172A). A venous tourniquet is applied just below the saphenous opening (fig. 172B), and he stands up (172C). The constriction is then released (172D). If the sapheno-femoral valve is incompetent, the veins fill immediately from



(A) (B) (C) (D)

FIG. 172.—The Brodie-Trendelenburg test (see text). (*Dr. S. M. Rivlin, London.*)

above; if not, the veins fill slowly from below. The veins may fill rapidly from above, even though the tourniquet has not been released; this means that the varices are in communication with a perforating vein (one is commonly present in the lower third of the thigh), or the sapheno-popliteal junction is incompetent. In this case, the tourniquet test must be repeated, with application at successively lower sites on the thigh and leg, until the point of origin is shown by prevention of the abnormal direction of flow.

Treatment.—(i) **Palliative treatment** is required for varicose veins in pregnancy, and for patients who do not wish for, who are unfit for, or who are waiting for, operation. The veins are supported by elastic stockings or crepe or elastic bandages.

Rowden Foote describes how a patient with an aching leg used to obtain relief by employing the Brodie-Trendelenburg manœuvre. If he lay down, elevated his leg, and then pressed hard in his groin, the veins remained empty when he stood up, and the aching was relieved.

(ii) **Injection-Compression.**[1]—The use of sclerosants, like ethanolamine oleate[2] 5 per cent. (Ethanolamine BPC), or sodium tetradecyl sulphate 3 per

[1] A word of warning is necessary in this era of the contraceptive pill. Caution should be exercised before giving sclerosant injections to a woman practising this form of family planning. Deep-vein thrombosis and fatal thrombo-embolism have been reported.

[2] Strange to relate, Mother Nature has, from the days of the primeval swamp, provided this substance from ? platelets in the actual chemistry of thrombosis.

Sir Benjamin Collins Brodie, 1783–1862. Surgeon, St. George's Hospital, London.
Friedrich Trendelenburg, 1844–1924 *(see p. 75).*
Robert Rowden Foote, Contemporary. Surgeon, Varicose Vein Dept., Harrow Hospital, Middlesex, England.

cent. (Sotradecol or Thrombovar) acts by damaging the intima of the vein so that a thrombosis, and later sclerosis, develops. Intimal damage will take place only whilst the sclerosant is able to act in sufficient concentration; as soon as it is diluted by the blood in the deep veins the effect is lost. If an excess of the sclerosant is injected at one site it may reach the deep veins in sufficient concentration to initiate a thrombosis before it becomes sufficiently diluted to render it harmless. *The minimum fully effective dose* for a sclerosant should always be known, and should not be exceeded at any one site. *Indications.*—Injection-Compression treatment is the treatment of choice for varicose veins confined to below the knee and for recurrent varicosities after operation. It is not an ideal cosmetic treatment because of the unpredictable staining which may occur.

Technique.—In order to make the venous occlusion permanent, injections must be given into an empty vein, so that wall adheres to wall with no intervening blood clot and thrombosis which will certainly recanalise. The needle is inserted into the vein with the patient sitting down, with the leg horizontal. A latex foam pad may be put over the site of the injection (not on to the skin directly), and along the length of vein (fig. 173), and it is maintained by pressure bandage and a stocking overall for six weeks from the last injection or until the pain has gone from the site (Fegan). Injections are also given at the site of the perforating veins as delineated by the Brodie-Trendelenburg test (above) and at the maximum site of tenderness.

The maximum dose at any one time and any one point is 1 ml. It is usual to commence treatment with a test dose of ½–1 ml. so that the extent of the local reaction may be gauged and the dose modified as necessary.

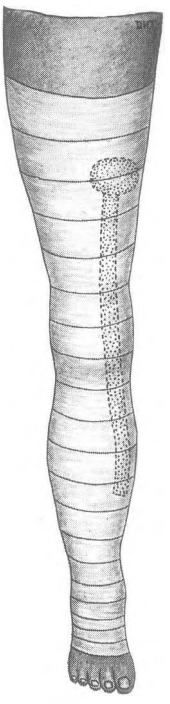


FIG. 173. — Bandage and pressure in the injection treatment of varicose veins (see text), and the ambulatory treatment of thrombophlebitis (p. 175).

(iii) Operations for Varicose Veins

Ligation Procedures.—The basic principle of the operative treatment of varicose veins is the ligation and division of those veins into which the high-pressure leak from the deep venous system has primarily occurred. Almost always this means ligation of the long saphenous vein where it enters the femoral vein (sapheno-femoral flush ligation). A flush ligation proximal to any tributaries is essential, otherwise a recurrence is inevitable (fig. 174).

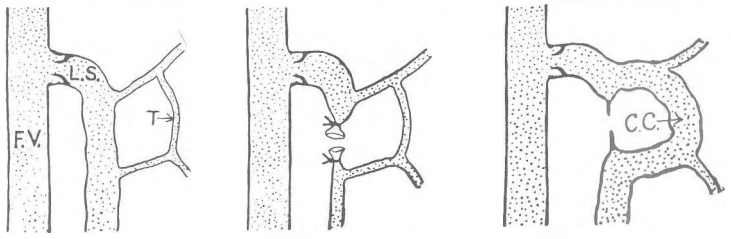


FIG. 174.—Tributaries (T.) must be ligated as well as the long saphenous vein (L.S.) flush with the femoral vein, otherwise a collateral circulation (C.C.) develops.

William George Fegan, Contemporary. Professor of Surgery, Trinity College, Dublin.

Should the short saphenous vein be the site of a high-pressure leak, it must be exposed in the popliteal fossa and ligated, proximal to any superficial tributaries, close to or flush with the popliteal vein.

In other instances of incompetent valves (or ' blow outs ') of the communicating veins (fig. 170), they must be ligated under the deep fascia (p. 184).

Technique.—An oblique incision is made just below the groin, commencing over the pulsation of the femoral artery and extending some 6 to 7 cm medially. The proximal portion of the long saphenous vein is exposed and traced to the femoral junction, which may lie 1·25 cm deep to the fossa ovalis, *dividing and ligating all tributaries* encountered on the way. It is then tied flush with the femoral vein. The presence of an accessory saphenous vein should be excluded. It may enter the femoral vein directly and escape the necessary attention.

Dealing with the varicosities below the groin includes additional ligatures as required for ' blow outs ' (see above). Other means of obliterating varicosities include multiple ligatures, injections, and minor and major avulsions (stripping). If a long saphenous vein is relatively normal (as opposed to its tributaries) it may be left since, in later years, it may well be needed for arterial bypass surgery.

Stripping.—After juxta-femoral ligation of the long saphenous vein and ligation of its tributaries the stripper is introduced into the vein in front of the internal malleolus (fig. 175). Care must be taken to preserve the long saphenous nerve which is close to the vein. The stripper is threaded upwards to emerge from the proximal end of the vein, the leg is elevated, and the vein avulsed by traction. Bleeding is controlled by pressure bandages applied from below upwards as the stripper is withdrawn. The wounds are sutured, and the patient is ambulatory the following day. An incision over the popliteal fossa permits stripping of the short saphenous vein, after it has been ligated at its entry into the popliteal vein. Care is also required to avoid stripping the sural nerve which is close to the vein behind the external malleolus.

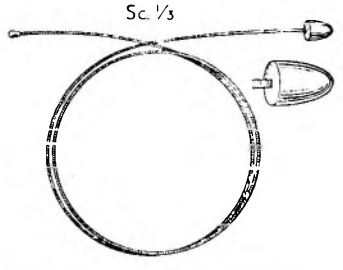


FIG. 175.—Myer's vein stripper.

Contraindications to Injection and Operative Treatment

(*a*) *Acute Infective Thrombophlebitis.*—At least three months should be allowed to pass, after this has completely subsided, before injecting.

(*b*) *Deep Thrombosis.*—Due to any cause or revealed by a history of prolonged confinement to bed with a painful swollen leg. Perthes' test is informative if doubt exists regarding the patency of the femoral vein:—The saphenous vein is occluded by a tourniquet applied immediately below the saphenous opening, and the patient walks 15 to 20 yards (13·5 to 18 metres). Normally, the veins below the constriction become less obvious, but if the communicating veins or the femoral vein are obliterated, these subcutaneous veins become engorged and the patient complains of ' bursting ' pain.

(*c*) *Pregnancy, Pelvic tumours and the ' Pill '.* See footnotes pp. 176 and 180.

COMPLICATIONS OF VARICOSE VEINS

Thrombophlebitis of superficial veins reveals itself as a reddened, tender cord in the subcutaneous tissues. Ambulatory treatment is safe and convenient. Strips of foam rubber or P.V.C., the edges of which are bevelled, are laid over the inflamed vein (over a layer of bandage), and 2·5 cm above it a double thickness is placed transversely. The leg is then bandaged with an elastic bandage (fig. 173). The strapping is removed after a fortnight. This procedure gives immediate relief, and it need be renewed only if tenderness persists.

Eczema (chronic dermatitis) may follow minor trauma or the patient scratching his skin which itches due to extravasation and break-down of red-cells in the affected area. It may be an allergic manifestation resulting from ointment or strapping applied for treatment. The condition is treated by the application, twice daily, of an ointment containing zinc oxide and coal tar.

Georg Clemens Perthes, 1869–1927. Professor of Surgery, Tübingen, Germany. Described this test in 1895.

Venous ulcers[1] occur either in connection with varicose veins (fig. 176), or follow deep-vein thrombosis in which recanalisation of the deep vein has occurred, but the valves are either destroyed or incompetent due to damage. Venous stasis is the underlying cause of both types as it results in local anoxia and œdema. It is important to be sure that the ulcer is not an ischæmic ulcer due to atherosclerotic arterial obstruction, arteritis (p. 162), or syphilis (p. 44). Also it is necessary to differentiate between the venous ulcers, for a varicose ulcer responds promptly to ambulatory treatment or ligation operations, but post-thrombotic ulcers are refractory to treatment, and may require bed-rest, curettage and skin grafting. Amnion may be tried (p. 65). Carcinomatous changes may occur in a chronic ulcer (fig. 47).

FIG. 176.—Venous ulcers with pigmentation of skin and talipes equinus deformity.

A deep venogram is particularly useful in difficult cases, as it will show the patency and the size of the lumen of the deep veins, the presence of valves, and the existence of high-pressure leaks in the calf:—

A fine rubber tourniquet is applied just above the malleoli to occlude the superficial veins. An injection of 20 to 30 ml. of 45 to 65 per cent. Hypaque (sodium diatrizoate) is given into a superficial vein of the foot, via a fine polythene cannula. (A test injection is, of course, given first, p. 141). The contrast medium is thus forced into the deep veins, and serial X-ray exposures are made of the whole leg. High-pressure conditions are simulated by tilting the patient into the semi-vertical position, or by his pinching the nostrils and endeavouring to expire forcibly.

Treatment.

(*a*) **Bisgaard method.** Almost any venous ulcer can be healed by elevation, bandaging, exercises, and massage. A physiotherapy department can be most helpful in applying the Bisgaard treatment, which consists of:

(1) Massage in elevation to the whole leg and particularly to soften the indurated area round the ulcer.

(2) Passive movements to maintain the mobility of the foot and ankle.

(3) Active movements to the calf muscles in elevation, and in standing (with bandages on).

(4) Teaching correct walking, placing heel down first and using the calf muscles to lift the heel of the back foot, giving 'spring' to the walk and therefore improving the venous pump (p. 178).

(5) A *firm* elastic (e.g. 'blue line') bandage is applied spirally from the base of the toes to the knee, so that movements in walking alternately stretch and relax the bandage and produce an added pumping effect.

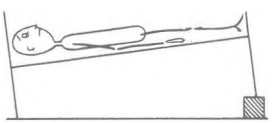


FIG. 177. — Sleeping with the foot of the bed raised.

(*b*) **Bandage and elevation.** Without facilities for Bisgaard treatment, reliance is placed upon the use of compression bandaging (e.g. wet 'Viscopaste' type, or a firm spiral of elastic bandage over a suitable dressing). Proper bandaging reduces the local œdema and aids the muscular component of the venous pump (p. 178). The patient should sleep with the foot of the bed raised (fig. 177) and periods of high

[1] *John Gay, 1812–1885. Surgeon, Great (now Royal) Northern Hospital, London. Was the first to use the term Venous Ulcer.*
Holger Bisgaard, 1881–1943. General Practitioner, Copenhagen. Published his method of treating varicose ulcers in 1939.

elevation of the leg are to be strongly encouraged (fig. 178). A 'wet' bandage cannot be wound spirally round the leg without the bias of the weave cutting into the skin. It must be applied in many encircling and overlapping strips. Often a piece of felt, with bevelled edges and cut to a size larger than the ulcer, is interposed in the bandage, over the ulcer, to reduce the local œdema which so often retards healing. A crepe bandage is put on over the 'wet' bandage. The patient is instructed to continue his usual work, and to wash off any discharge which may percolate through the bandage. Injection-Compression therapy may also be used as an ancillary treatment.

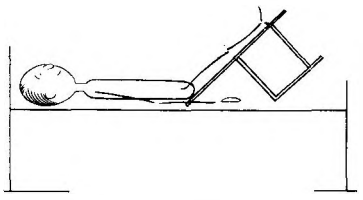


FIG. 178.—High elevation of the leg, using a chair.
(After Dickson Wright, F.R.C.S., London.)

The first application is removed after one week, and thereafter the bandage is renewed at fortnightly intervals until the ulcer is healed. The area of the ulcer should be measured at each visit, so that the decrease in size can be recorded.

(*c*) **Subfascial Ligation** (Cockett & Dodd Operation).—Most venous ulcers are due to incompetence of perforating veins on the inner side of the ankle (fig. 170). Therefore, when the ulcer has healed, the perforating veins should be identified and divided through an incision in the lower half of the leg 2·5 cm behind the posterior border of the tibia. The veins, which may be enormously dilated, are best secured by exposing them beneath the deep fascia. If necessary the saphenous veins are also dealt with, as already described.

Hæmorrhage from a ruptured varicose vein is usually profuse. Elevation of the leg and the application of a firm pad and bandage easily control the bleeding. *On no account* should a tourniquet be used (p. 74).

Calcification occasionally occurs in veins which have been varicose for many years.

Periostitis occurs in long-standing cases if the ulcer is situated over the tibia.

Equinus deformity may result from a long-standing ulcer. The patient finds that walking on the toes relieves the pain, and after some years the Achilles tendon becomes contracted (fig. 176). Cases usually respond to remedial exercises. The Bisgaard treatment (p. 183) should prevent this complication.

Harold Dodd, Contemporary. Emeritus Surgeon, Royal London Homœopathic Hospital, London.

CHAPTER 12

LYMPHATICS AND LYMPH NODES

Acute lymphangitis is caused by infection spreading from a wound in the drainage area of the involved lymphatic vessels. The infection is usually limited by the lymph nodes immediately proximal to the site of infection, where an abscess may form, but occasionally infection by-passes a group of lymph nodes and affects those at a higher level. Thus, in the case of an infected foot, the abscess may form in the external iliac lymph nodes instead of the groin, and because the wound itself may have healed and been forgotten by the time the mass appears, it may be mistaken for an appendix abscess.

Acute lymphangitis is characterised by the appearance of red blushes and streaks in the skin, corresponding to the inflamed lymphatics (fig. 179). The streptococcus is the common infecting organism. Toxæmia is often severe, and is greater when the more proximally the lesion has extended. Permanent lymphatic occlusion may follow acute lymphangitis, leading to persistent œdema.

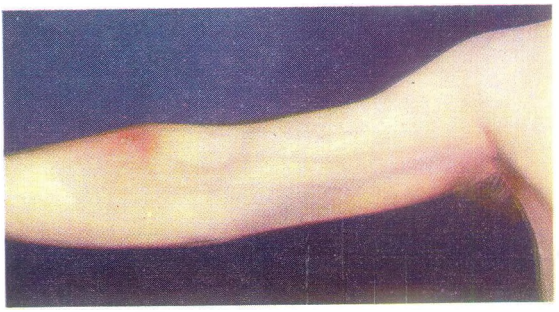


FIG. 179.—Acute lymphangitis of the arm. Red streaks extend from the infection on the forearm up to the enlarged and tender axillary lymph nodes.

Treatment consists of bed rest, with the affected limb comfortably elevated, and giving cloxacillin, which usually causes rapid resolution. *Only* when there are definite signs of pus should an incision be made.

Chronic lymphangitis may follow repeated attacks of acute lymphangitis (see acquired, secondary, lymphœdema p. 186).

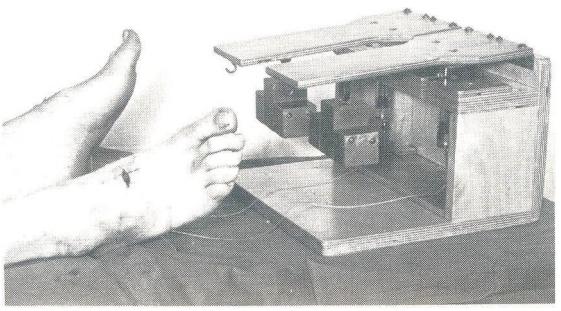


FIG. 180.—Pedal lymphangiogram: Blue dye is injected subcutaneously between the toes to outline the lymphatic vessels. A small incision is made on the dorsum of each foot and a 30G needle is passed into a vessel. Using an infusion pump, such as this (McIvor–Charing Cross), oily contrast medium (ultrafluid lipoidol) is injected slowly and subsequently lodges in the sinuses of the pelvic and abdominal lymph nodes. *(Dr. James McIvor F.D.S., F.R.C.R., London.)*

Lymphangiography

Lymphangiography (fig. 180) has solved many of the problems concerning lymphatic œdema and the assessment of the spread of malignant disease (e.g. malignant melanoma, testicular tumours, reticulosis) (fig. 181).

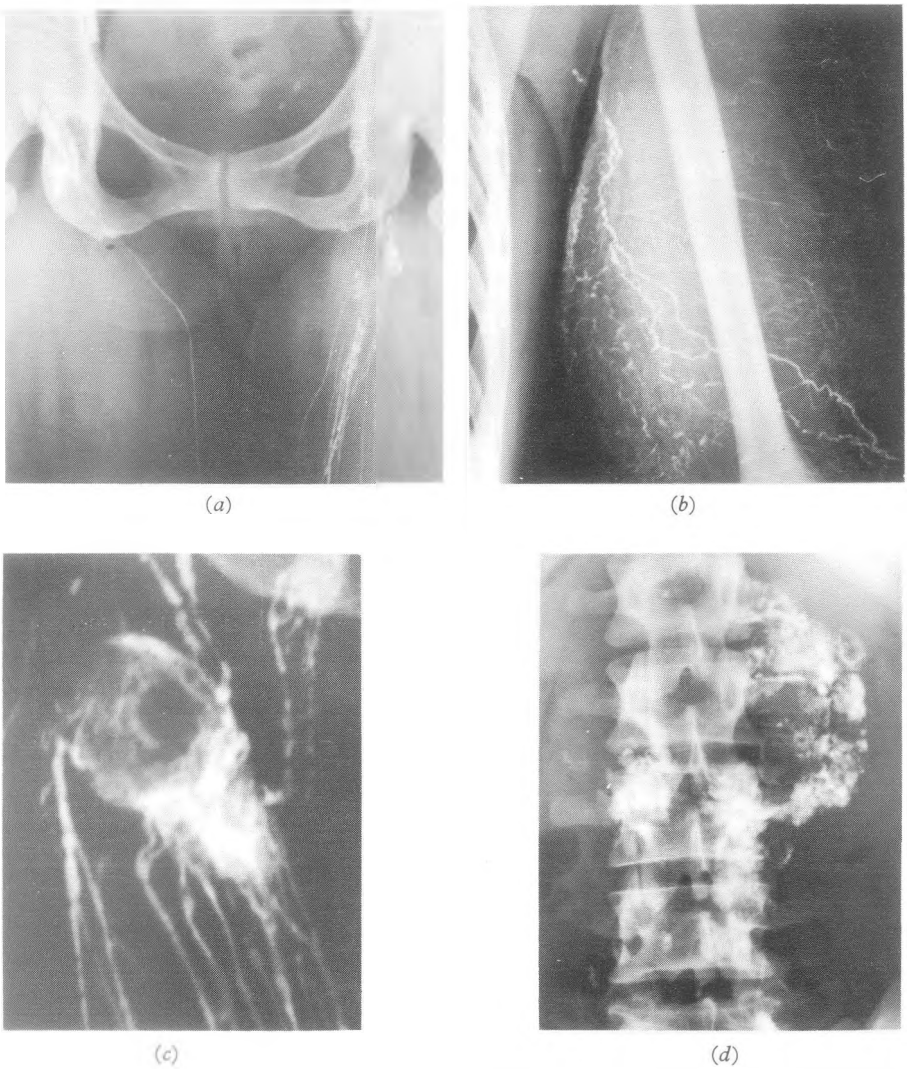


FIG. 181.—(*a*) Congenital lymphoedema of right leg due to lymphatic hypoplasia. (*b*) Obstructive lymphoedema of the left arm secondary to radical mastectomy plus radiotherapy. (*c*) Filling defect in an inguinal node caused by metastatic melanoma. (*d*) Enlarged para-aortic nodes due to metastases from a testicular teratoma.

LYMPHŒDEMA (and ELEPHANTIASIS)

Lymphœdema is caused by the accumulation of fluid in the lymphatics. In the limbs it affects mainly the subcutaneous tissues where there is an in-

crease both in the stagnant lymphatic channels and in the supporting fat. Lymphœdema is either congenital or acquired.

(*a*) **Congenital malformations** (**primary lymphœdema,** fig. 182), include:

(i) Aplasia—the main subcutaneous trunks fail to develop. The dermal lymphatic plexus is dilated.

(ii) Hypoplasia—the main subcutaneous trunks are few and underdeveloped.

(iii) Dilated and varicose lymphatic trunks—this may be associated with congenital arterio-venous fistula, and also with ' chylous reflux ' (p. 188).

Congenital lymphœdema is either hereditary or familial (Milroy's disease). The œdema may be in one or both legs. Starting at birth (Lymphœdema congenita), or at puberty (L. præcox), or in adult life (L. tarda) continues throughout life and does not of itself produce any constitutional disturbance.

FIG. 182. — Primary lymphœdema of the left leg.

(*b*) **Acquired obstruction** (**secondary lymphœdema**) is due to:

(i) Trauma, e.g. the removal of axillary lymphatics in radical mastectomy.

(ii) Repeated acute infection, as in those who go about barefoot.

(iii) Chronic infection, e.g. tuberculosis, filariasis[1] (p. 49) (fig. 183), and fungus infection.

(iv) Obstruction by malignant disease (fig. 184).

Whatever the cause, the stagnation in the lymph stream is usually followed by recurrent infection. Each such attack makes the obstruction worse until the limb becomes grotesquely swollen, the skin folded and wrinkled and, particularly in the leg, coarsened (like that of an elephant) with brown areas of dry undesquamated keratin. Ulceration eventually follows.

Differential Diagnosis.—*Bilateral* œdema of the legs always requires an examination to exclude cardiac, renal and metabolic (hypoproteinæmia) causes. *Unilateral* lymphœdema may be confused with that caused by deep vein thrombosis or compression, particularly of the iliac veins on the left side:—

Iliac Vein Compression (Cockett).—Venograms of the iliac and pelvic veins, and the cava, may be obtained by injection of diatrizoate into the femoral vein, or into the bone marrow via the greater trochanter (per-trochanteric venogram). The left common iliac vein or the cava may be compressed by the over-riding aortic bifurcation and thrombosis in, or a stricture of, the vein may be present. Operations involving the mobilisation of the aortic bifurcation, or direct venous disobliteration, are still under trial.

Treatment.—1. *Palliative.*—For the attacks of inflammation, prolonged bed-rest, elevation, and the appropriate antibiotic are important. Even in

[1] Sir Patrick Manson, 1844–1922, discovered elephantiasis was caused by filaria in 1877 while working in China. The organisms may be discovered microscopically in a nocturnal blood smear.

William Forsyth Milroy, 1855–1942. Professor of Clinical Medicine, University of Nebraska, Omaha, Nebraska, U.S.A. Described hereditary œdema of the legs in 1892.
Frank Bernard Cockett, Contemporary. Surgeon, St. Thomas's Hospital, London.

FIG. 183. — Lymphœdema due to filariasis.
(R. S. Naik, Durg, India).

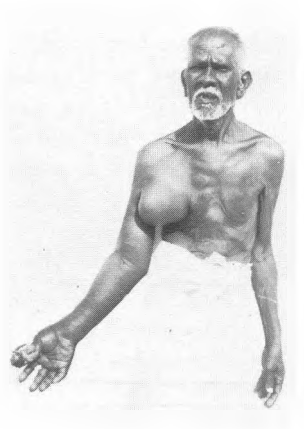


FIG. 184.—Secondary lymphœdema, due to malignant melanoma of the right thumb with massive involvement of axillary lymph nodes.
(V. Radhakrishnan, Tamilnadu, India).

between attacks, the patient should sleep with the foot of the bed raised and may require to use efficient elastic stocking pressure (e.g. Sigvaris®). Intermittent diuresis, induced by modern diuretic drugs, also is helpful.

2. *Surgery* is reserved for those with severe disability. One of many surgical procedures is the removal of all the abnormal subcutaneous tissues and the covering of the exposed deeper tissues with a split skin graft (flaying operation). Another method is to bury the dermal part of the excess skin like a ' swiss-roll cake ' along the whole length of the leg, so that the subdermal lymphatics may orientate themselves lengthwise and thereby assist drainage (fig. 185). Other operations seeking to divert the lymph flow through the deep fascia by removing strips of it (Kondoleon's operation) or across the obstructed zone by a skin pedicle bridge, have failed, though a bridge of mesentry and ileum, bereft of mucosa, is being tried (Kinmonth) in early cases, otherwise it is difficult to reduce the great mass of subcutaneous tissue.

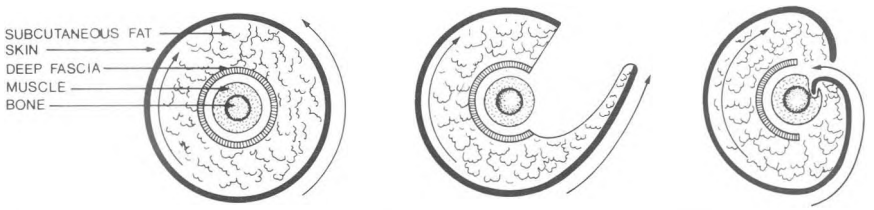


FIG. 185.—Swiss Roll operation (buried dermis) see text. *(N. Thompson, F.R.C.S., London).*

3. *Microsurgery*.—It is becoming possible to anastomose dilated lymphatics to veins (lymphovenous anastomosis), for example in the antecubital fossa in cases of œdema of the arm (O'Brien).

Chylous Reflux.—Lymphangiography may reveal a backflow of milky chyle from the cisterna chyli, with dilated lymphatics, resulting in cutaneous chylous vesicles and fistulæ on the limbs. Ligation and excision of the lymphatics on the posterior abdominal wall may cure the reflux, though lymphœdema persists.

Emmanuel Kondoleon, 1879–1936. Surgeon in Athens.
John Bernard Kinmonth. Contemporary. Professor of Surgery, St. Thomas's Hospital, London.
B. McC. O'Brien, Contemporary. Director, Microsurgery Research Unit, and Assistant Surgeon, St. Vincent's Hospital, Melbourne, Australia.

LYMPHANGIOMA

Capillary Lymphangioma.—Localised congenital anomalies may be composed of capillary-like lesions in the skin. They are brownish papules or wart-like excrescences. On examination with a lens, small vesicles can be seen, which are lymphatic nævi.

Cavernous lymphangioma is often associated with the preceding variety, and consists of masses of lymphatic cysts, particularly in the neck or axilla, the condition being termed a *cystic hygroma* (p. 599). A similar lymphangiectatic condition may affect the lips (macrocheilia) or the tongue (macroglossia) producing sometimes gross soft tissue enlargement.

LYMPH NODES

Acute Inflammation.—Lymph nodes, draining any area where there is acute infection, will also become inflamed (p. 185).

Chronic inflammation is either simple (pyogenic) or specific.

1. *Simple Lymphadenitis* is due to persistence of infection, such as occurs in recurrent tonsillitis or pediculosis capitis, and the cause should be treated.

2. *Specific Lymphadenitis*

(*a*) **Tuberculous lymphadenitis** is common in children and young adults, particularly those who have been in contact with open human tuberculosis or who drink infected milk. It also occurs in the aged. The cervical lymph nodes are most often seen to be enlarged, but the remnants of disease may be observed in the mediastinal and mesenteric lymph nodes as speckled calcification on routine X-rays. Axillary lymph nodes may be involved by spread from the mediastinum.

Tubercle bacilli most commonly reach a lymph node by lymphatics, when tubercles first form in the cortex. Microscopically, endothelial cells and lymphocytes are in evidence. Giant cells are seen with many nuclei arranged round the periphery like a horseshoe.

The stages of infection show most clearly in the neck. From the tonsillar portal of entry the infection spreads by the lymphatics to the nearest lymph node. If the disease spreads, several lymph nodes are involved. They coalesce and break down to form caseous tuberculous pus, which may perforate the deep fascia and present as a fluctuant swelling on the surface

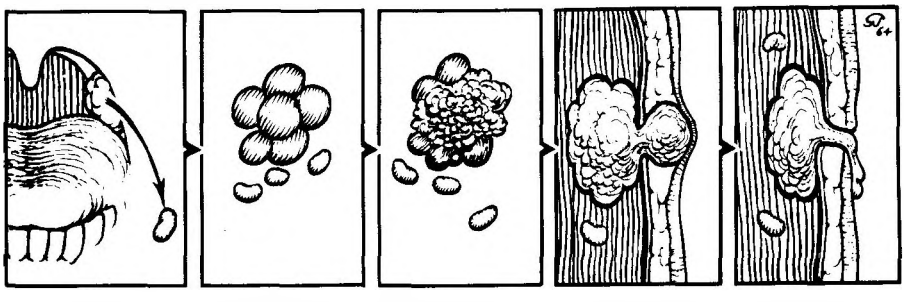


FIG. 186.—A summary of the natural history of tuberculous lymphadenitis.

(collar-stud abscess) (figs. 186 and 187). The skin gradually becomes indurated, breaks down, and forms a sinus which if ignored will remain unhealed for years. From each of these stages resolution may take place with calcification (if caseation has occurred), and with much scarring (if sinuses have formed).

FIG. 187.—A cold abscess of the neck.

Treatment.—(i) Attention to nutrition and general health.

(ii) Tuberculous material is aspirated for culture and drug sensitivity tests. A specimen must be obtained before the anti-tuberculous drugs are started.

(iii) Anti-tuberculous drugs are given immediately after the aspiration (p. 37).

(iv) When the patient's condition begins to improve, breaking-down tuberculous lymph nodes must be removed, because the drugs will not reach the organisms in the avascular caseous material.

(*b*) **Syphilitic adenitis.**—'Shotty' lymph nodes in the groin associated with a genital chancre are characteristic. During the secondary stage a generalised enlargement of lymph nodes occurs. Especially noticeable are those above the internal epicondyles and along the posterior border of the sternomastoid.

3. *Other Infections*

Glandular Fever (*syn.* infective mononucleosis) is an acute virus infection. After an incubation period of five to fourteen days, enlarged, elastic and slightly tender lymph nodes appear, associated with an irregular fever and often a sore throat, a rash, and splenic enlargement. The blood examination is diagnostic, revealing an absolute and relative lymphocytosis, and an unusual concentration of sheep-cell agglutins (the heterophile antibody of the Paul-Bunnell reaction). The condition may be mistaken for acute leukæmia or lymphadenoma. Treatment is symptomatic, and recovery is the rule, although it may be some months before the lymph nodes return to normal. The patient is isolated in the early stages.

Toxoplasmosis.—*T. Gondii*, a small intracellular protozoan is liable to be transmitted from mammals to humans who eat raw or underdone meat which has not been previously frozen. The most tragic manifestation is neonatal jaundice and encephalomyelitis followed by hydrocephalus or microcephaly, blindness and intracerebral calcification. Occasionally the disease manifests itself in children and adults with enlarged lymph nodes and fever. A complement fixation test is necessary.

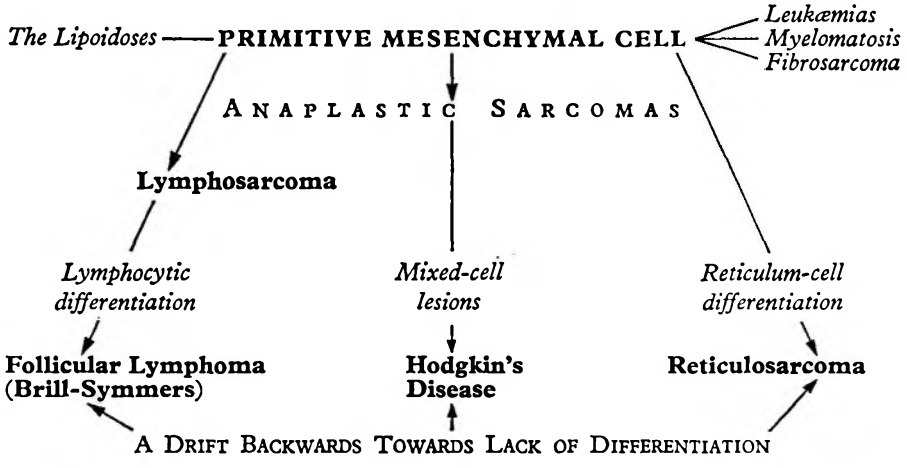
Cat-Scratch Fever is due to a virus of the lymphogranuloma-psittacosis group. Localised inflammation occurs at the site of the lesion, associated with fever, malaise, and anorexia. This subsides in a few days, but from two to several weeks later the regional lymph nodes become enlarged. Suppuration often occurs, but the pus is sterile, and after evacuation the abscess subsides. Diagnosis is usually suggested by unilateral involvement of lymph nodes and the history of cat scratches. It is confirmed by a skin test with antigen prepared from human lymph-node pus. This distinguishes the condition from chronic pyogenic or tuberculous adenitis with which it is often confused. Broad-spectrum antibiotics help only in reducing the fever.

THE RETICULOSES

Numerous progressive diseases arise from neoplastic change in the various elements of the reticulo-endothelial system. In general, the less they are histologically differentiated, the more rapidly do they grow. In addition,

John Rodman Paul, Contemporary. Emeritus Professor of Preventive Medicine, Yale University School of Medicine, New Haven, Connecticut, U.S.A. Described this test in a joint paper with Bunnell in 1932.
Walls Willard Bunnell, 1902–1965. Physician, Hertford Hospital, Connecticut, U.S.A.

they may start as slowly growing well-differentiated lesions and then become rapidly growing and more primitive as time goes on (' drift-back '). There is a great deal of variation in the histological types and no rigid classification is entirely satisfactory. The following diagram gives a possible classification, and summarises this paragraph.



These diseases may either present as lymph node enlargement, or in the systemic form when virtually any structure can be affected. The lymph nodes are usually enlarged as one group, though other groups (e.g. Hodgkin's disease) may be affected. The nodes are discrete and elastic, being more rubbery in Hodgkin's disease and follicular lymphoma, but larger and softer in lymphosarcoma and reticulosarcoma. Tuberculous nodes classically are matted together by periadenitis, but may be discrete in early cases and in the senile form.

Diagnosis.—The clinical examination includes palpation of other groups of lymph nodes, and the abdomen for the presence of an enlarged spleen or liver. Investigations include X-ray of the chest (? mediastinal lymph nodes) and a full cytological examination of the blood (? leukæmia). A biopsy of an easily accessible lymph node should not be delayed. It is best to perform this under a general anæsthetic. The groin is avoided, if possible, as histology of the nodes is often complicated by inflammatory changes.

Hodgkin's disease is now described as an example:

HODGKIN'S DISEASE (LYMPHADENOMA)

Pathology.—Hodgkin's disease is a malignant neoplasm of the reticulum cells of lymphoid tissue and is the commonest of the malignant lymphomas. Wherever there is lymphoid tissue Hodgkin's disease may occur. On macroscopical section the involved nodes are pink-grey in colour and of rubbery consistency. Except in the more anaplastic forms the nodes are discrete with no periadenitis. The spleen, when involved, does not at first have the

Nathan Edwin Brill, 1860–1925. Physician, Mount Sinai Hospital, New York, U.S.A. Described this disease in 1925.

Douglas Symmers, 1879–1952. Professor of Pathology, New York Hospital Medical School, New York, U.S.A. Published his description of this disease in 1927.

Thomas Hodgkin, 1798–1866. Curator of the museum and demonstrator of morbid anatomy, Guy's Hospital, London. In 1832, read a paper to the Medico-chirurgical Society of London entitled 'On Some Morbid Appearances of the Absorbent Glands and the Spleen,' in which he described a number of cases of lymphadenoma. In 1865, Sir Samuel Wilks (1824–1911), Physician, Guy's Hospital, London, suggested that the condition be called Hodgkin's disease.

same homogeneous appearance as the lymph nodes, but as the disease advances the infiltration becomes more diffuse. Bone deposits occur in the vertebral column and pelvis. Rarely, Hodgkin's disease may be confined to a single organ such as the stomach.

The histological appearance may vary with the stage of the disease but a striking feature is the cellular pleomorphism. To a variable extent the normal nodal architecture is replaced by a cellular infiltration with lymphocytes, reticulum cells, eosinophils and fibrous tissue and the characteristic Reed-Sternberg cells will be seen—a giant cell containing two large pale mirror image nuclei which may overlap (like pennies on a plate).

The nature of Hodgkin's disease is still questioned. The Reed-Sternberg cell is an unusual cell in that it undergoes nuclear but not cytoplasmic division. Studies with tritiated thymidine suggest that the reticulum cell is really the basic malignant cell and that the binucleate giant cell is its product. Evidence against the malignant nature of Hodgkin's disease is based upon the fact that Reed-Sternberg cells have occasionally been found in the blood of patients with other diseases—e.g. glandular fever. In addition the similarity between the end stage of Hodgkin's disease and the graft-versus-host reaction suggests an immunological basis. The lymphocytic and acute inflammatory cellular response seen in involved nodes are thought to represent the host's immunological reaction.

Histological Classification.—The current histopathological classification of Hodgkin's disease is based predominantly on the lymphocytic infiltration, and the fibrous and reticular elements. The reason for the change in classification from the original of granuloma, paragranuloma and Hodgkin's sarcoma is that this classification was found to be prognostically insignificant in a large proportion of cases, especially those showing the features of the so-called granuloma. Fig. 188 shows the comparison

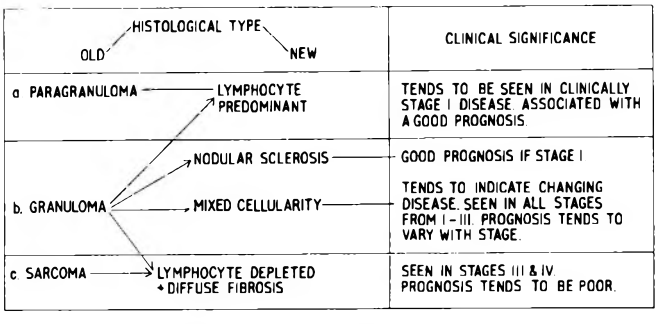

HISTOLOGICAL TYPE
OLD
NEW
CLINICAL SIGNIFICANCE
a PARAGRANULOMA
LYMPHOCYTE PREDOMINANT
TENDS TO BE SEEN IN CLINICALLY STAGE I DISEASE ASSOCIATED WITH A GOOD PROGNOSIS
NODULAR SCLEROSIS
GOOD PROGNOSIS IF STAGE I
b. GRANULOMA
MIXED CELLULARITY
TENDS TO INDICATE CHANGING DISEASE. SEEN IN ALL STAGES FROM I-III. PROGNOSIS TENDS TO VARY WITH STAGE.
c SARCOMA
LYMPHOCYTE DEPLETED + DIFFUSE FIBROSIS
SEEN IN STAGES III & IV. PROGNOSIS TENDS TO BE POOR.

FIG. 188.—Histological classification of Hodgkin's disease (see fig. 193).

between the original and present classification and their prognostic significance and clinical correlations. The accompanying figs. 189, 190 and 191 illustrate the histological features seen in biopsy specimens of lymph nodes.

Clinical Features (fig. 192).—The disease is more common in males, and usually it affects young adults (25–40 age group).

The most common presentation is painless progressive lymph node enlargement in the cervical or supraclavicular region which may or may not be associated with generalised symptoms such as malaise, fever, weight loss or pruritus. Pressure effects such as superior vena-caval obstruction from enlarged mediastinal lymph nodes may follow or even be the presenting feature. Bone pain, particularly spinal may indicate vertebral collapse

Dorothy Reed, 1874–? Pathologist, Johns Hopkins Hospital, Baltimore, U.S.A. Described the giant cells in 1902.
Karl Sternberg, 1872–1935. Pathologist, Vienna. Described the giant cell of Hodgkin's disease in 1898.

secondary to bony metastases. A recognised but unexplained symptom is pain in the sites of disease induced by drinking alcohol.

On examination, the involved nodes tend to be discrete, non-tender and

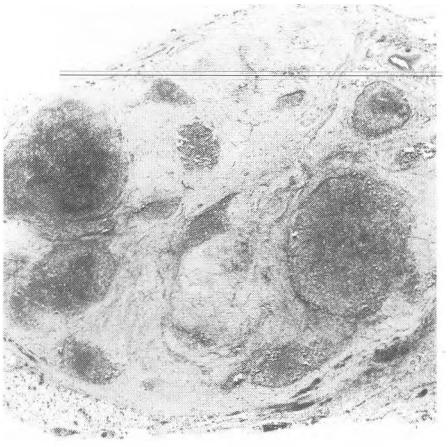


FIG. 189.—Section of whole lymph node (nodular-sclerotic).

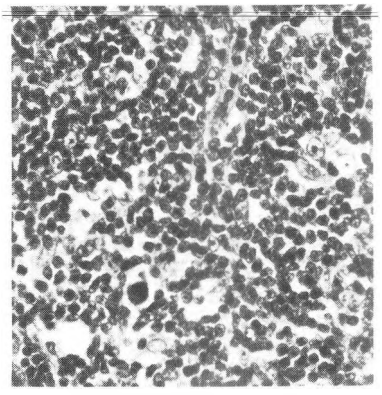


FIG. 190.—Lymphocytic predominance.

rubbery in consistency as opposed to tuberculosis where the nodes are matted together. In late or anaplastic cases however the nodes may become matted. Splenomegaly and hepatomegaly is a variable accompaniment (see below). As the disease advances irregular elevations of the temperature may occur.[1] With dissemination bony metastases, anæmia and pancytopænia may develop. There may also be skin deposits in advanced cases (Mycosis fungoides). Jaundice due to excessive hæmolysis of red cells or diffuse liver involvement is also seen.

The course of the disease is very variable: death may follow in weeks but long term survivals are not uncommon and with modern methods of treatment the disease may be cured.

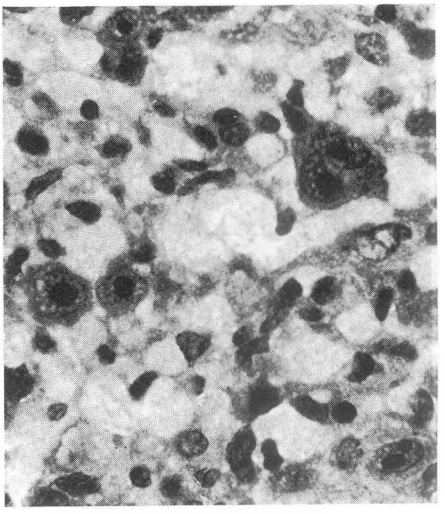


FIG. 191.—Mixed cellularity. Reed-Sternberg cells.

The Clinical Staging of Hodgkin's Disease (fig. 193).—The clinical staging of Hodgkin's disease, which includes localised, generalised and intermediate varieties, is necessary to determine accurately the extent of the disease, the subsequent treatment and the prognosis.

[1] The periodic Pel-Ebstein fever, originally described as a feature of Hodgkin's disease is a mistake. This Pel-Ebstein fever was due to Brucellosis.

Pieter Klazes Pel, 1852–1919. Professor of Medicine, Amsterdam, Holland. Described a relapsing fever in Hodgkin's disease in 1885.

Wilhelm Ebstein, 1836–1912. Professor of Medicine, Göttingen, Germany. Also described the relapsing fever of Hodgkin's disease in 1887.

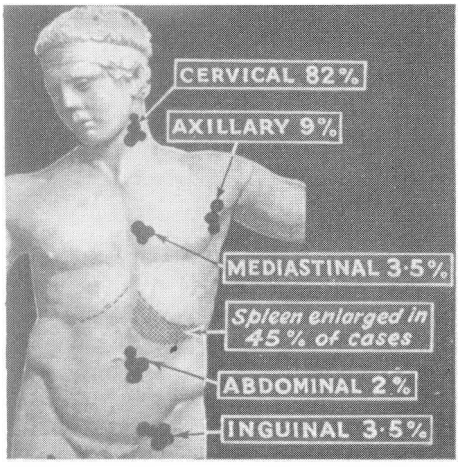


FIG. 192.—Showing the position of Hodgkin's disease when the patient was seen first. In 14% of cases more than one region was involved. (*Baker and Mann's statistics.*)

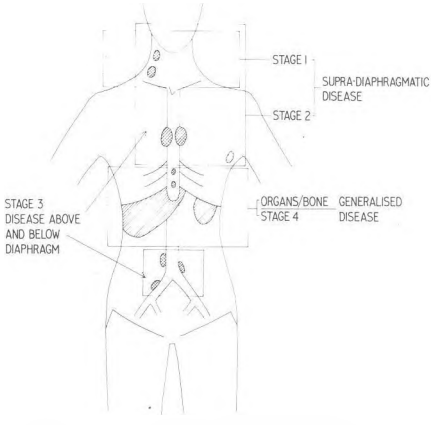


FIG. 193.—The clinical staging of Hodgkin's disease.

The disease is subdivided into four stages. Each stage is further subdivided into groups *a* or *b* according to the absence or presence of associated generalised symptoms such as weight loss, fever, pruritus, anæmia and bone pain.

Special Investigations Leading to Clinical Staging.—The following procedures are currently in use:—

A. *Node Excision Biopsy* to establish the diagnosis and accurate histological grading.

B. *Chest X-Ray* to demonstrate enlarged mediastinal nodes and tomography when indicated.

C. *Mediastinal Scanning* with Gallium 67 has been found to demonstrate involved mediastinal nodes.

D. *Intravenous Urography* may show distortion or compression of the renal calyces by retroperitoneal nodes.

E. *Lower Limb Lymphangiograms* will demonstrate both pelvic and retroperitoneal nodes. Unfortunately this investigation is associated with both false positive and false negative results.

F. *Laparotomy/Splenectomy/Liver and node biopsy* are being used more frequently to stage Hodgkin's disease.

Clinical splenomegaly is unreliable in assessing splenic involvement. In one series 50% of patients with splenomegaly were found not to have splenic Hodgkin's at laparotomy. Moreover 25% of those cases without clinical splenomegaly were found to have deposits in the spleen at laparotomy. In addition this procedure may aid in decreasing the problem of hypersplenism and obviate splenic irradiation and its complications. Enlarged aortic, iliac and mesenteric nodes are sought and biopsied. While the value of this procedure must be weighed against its morbidity and mortality, its value is obvious in staging and treatment.

The Relationship between Histological Grade, Clinical Stage and Survival.—There are two clinical varieties of Hodgkin's disease; a localised

predominantly non-aggressive form, and an aggressive, progressive and fatal variety. Between these two extremes there are intermediate or changing types of disease.

Histologically inactive disease is lymphocyte predominant, clinically stage I and potentially curable. Progressive disease is characterised histologically by diffuse fibrosis and lymphocyte depletion or anaplastic reticulum cells. Clinically it is seen in stage IIIb and IV and is associated with systemic symptoms. Changing disease is characterised by mixed cellularity or nodular sclerosis in stage II or III disease. Nodular sclerosis in stage I disease however may be associated with a good prognosis.

Treatment Policy for Hodgkin's Disease and Reticuloses.—There are two forms of treatment available according to the accurate staging of the disease, radiotherapy and combination chemotherapy. Both treatments are associated with bone marrow depression and toxic side-effects and must be carefully planned and controlled with regular blood counts. Radiotherapy is the treatment of choice in stage I, II and IIIa disease, whilst combination chemotherapy is used in stage IIIb and IV. Megavoltage radiotherapy allows wide areas to be treated. Supradiaphragmatic disease is treated by radiotherapy to cervical, axillary and mediastinal nodes. Normal tissue is protected by lead shields. Radiotherapy is given in divided doses over several weeks and the dose is in the region of 3,500–4,000 rad. Infradiaphragmatic disease is treated by fields covering the para-aortic and iliac nodes (inverted Y fields). Combination chemotherapy involves repeated courses of cytotoxic drugs and steroids as it has been shown that the drugs produce better remission when used in combination. A combination commonly used is Nitrogen mustard, Vinblastine, Procarbazine and Prednisone.

FIG. 194.—Reticulosarcoma of the parotid 'lymph gland', which responded to radiotherapy.

Results and Prognosis of the Reticuloses.—50% five-year cures can be obtained in Hodgkin's disease and reticulosarcoma (fig. 194). Lymphosarcoma is often very radiosensitive, the five-year cure rate for localised disease in the neck being 70%.

Malignant Lymphoma of Africa (Burkitt Tumour) (fig. 195).—*The ætiology* is of special interest. This rapidly growing tumour is the commonest malignant disease of children living in the equatorial regions of Africa. The age distribution is characteristic with over 80% of cases occurring between the ages of 3 and 12 years inclusive. Although this tumour is now known to occur occasionally anywhere in the world, it has only been reported in endemic proportions in tropical Africa and New Guinea. The observation that endemic distribution is limited to warm, moist climates strongly suggests the implication of some biological agent. A

Denis Parsons Burkitt, F.R.S., Contemporary. Medical Research Council Scientific Staff. Described this type of sarcoma in 1958.

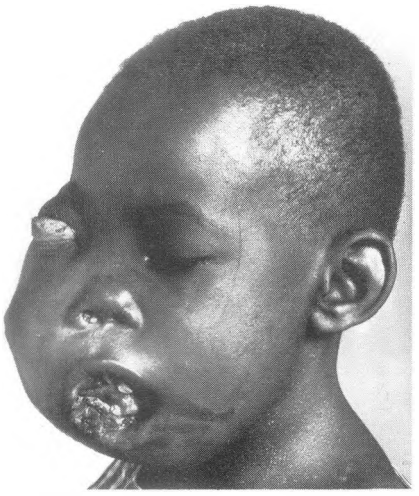


FIG. 195.—Burkitt tumour. (*Dr. Denis Burkitt, London.*)

vectored virus was consequently considered the most likely ætiological factor. The realisation that many regions are apparently tumour-free, whereas the distribution coincides with that of hyperendemic and holoendemic malaria, suggests that variations in incidence may be dependent on malarial changes in the reticulo-endothelial system, with an added virus infection inducing malignant change. Evidence of E.B. virus infection which is now believed to be the cause of infectious mononucleosis has been almost invariably found in Burkitt's lymphoma and the two conditions may be associated.

Clinical and Pathological Features.—The tumour is multifocal, grows rapidly and is soft and relatively painless. In Africa, approximately 50% of patients present with jaw tumours which may affect one or more of the jaw quadrants. Maxillary lesions may present as oral or orbital tumours. The second commonest clinical presentation is an abdominal tumour. Renal involvement, usually bilateral, is present in 75% of all patients. In girls, ovarian involvement is particularly characteristic and paraplegia without vertebral collapse is also common. Peripheral or mediastinal lymphadenopathy or lung involvement is exceptional, but tumours are frequently observed in the thyroid, testicles, skeleton and breasts. Radiologically the tumours first appear as multiple, small osteolytic deposits which coalesce to form larger areas of bone destruction. On microscopic section dense masses of darkly staining primitive lymphoid cells interspersed with large clear histiocytes give an appearance under low power magnification of a 'starry night'.

Treatment.—The tumour is unusually sensitive to both radiotherapy and a wide range of cytotoxic drugs such as cyclophosphamide, methotrexate and orthomelphalan. A much higher proportion of long-term survivors have been reported than in any other form of childhood cancer. Several of these have followed single doses of therapy suggesting a strong host defence mechanism, possibly immunological. Surgery is not indicated with the possible exception of removing large ovarian tumours. A number of spontaneous remissions have been recorded.

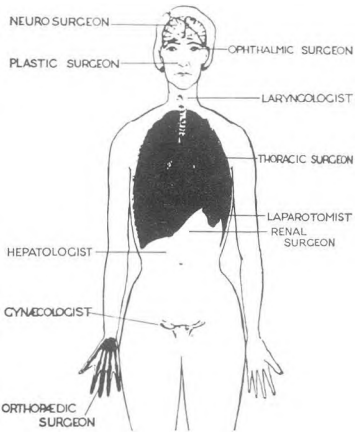


FIG. 196.—Presentations of sarcoid tissue to various surgical specialists.

Boeck's Sarcoidosis is a multisystem granulomatous disorder of unknown ætiology most commonly affecting young adults and presenting most frequently with bilateral hilar lymphadenopathy, pulmonary infiltration, skin or eye lesions. The diagnosis is established most securely when clinicoradiographic findings are supported by histological evidence of widespread non-caseating epithelioid cell granulomas in more than one organ or a positive Kveim-Siltzbach skin test. Immunological features are depression of delayed-type hypersensitivity suggesting impaired cell-mediated immunity and raised or abnormal immunoglobulins. There may also be hypercalciuria with or without hypercalcæmia. The course and prognosis may correlate with the mode of onset: an acute onset with erythema nodosum heralds a self-limiting course and spontaneous resolution while an insidious onset may be followed by relentless progressive fibrosis.

Cæsar Peter Moeller Boeck, 1845–1917. Professor of Dermatology, Oslo, Norway. Described sarcoidosis in 1899.
Morten Ansgar Kveim, 1892–1967. Dermatologist, Oslo, Norway.
Louis Elliot Siltzbach, 1906–1980. Clinical Professor of Medicine, Mount Sinai Hospital, New York, U.S.A.

DIFFERENCES BETWEEN SARCOIDOSIS, AND A NON-SPECIFIC LOCAL SARCOID-TISSUE REACTION

	Sarcoidosis	*Sarcoid-tissue Reaction*
No. of systems involved	Several	Usually one
Age group	20–50 years	Any
Chest radiograph	Abnormal in 84%	Normal
Slit lamp examination of eyes	Abnormal in 25%	Normal
Tuberculin test	Negative in 66%	Variable
Siltzbach-Kveim test	Positive in 84%	Always negative
Calcium metabolism	Abnormal in 20%	Normal
Response to corticosteroids	Good	Variable
Other treatments	Oxyphenbutazone Chloroquine p-Aminobenzoate	Depends on cause —Anti-Infective —Immunosuppressive —Cytotoxic

COMPARISON BETWEEN SARCOIDOSIS AND HODGKIN'S DISEASE

Feature	*Sarcoidosis*	*Hodgkin's disease*
Age	20–50	Any
Fever	Rare	Common
Weight loss	Infrequent	Frequent
Splenomegaly	In 12%	Very common
Submental lymph nodes	No	Yes
Erythema nodosum	Common	Very rare
Pruritus	Absent	Present
Bone lesions	'Punched out' phalangeal cysts	Sclerotic lesions of spine and pelvis
Leukocytosis; lymphopenia; eosinophilia	Absent	Present (sometimes)
Hilar lymphadenopathy	Bilateral	Bilateral or unilateral
Pulmonary infiltration	Common	Uncommon
Pulmonary infiltration followed by hilar adenopathy	Never	Frequent
Secondary infections	Rare	Common
Pleural effusion	No	Yes
Immediate-type hypersensitivity	Normal	Normal
Delayed-type hypersensitivity	Depressed	Depressed
Circulating antibodies	Normal	Normal
Serum immunoglobulins	Normal or nonspecific increase	Normal
Homograft rejection	?	Delayed
Histology	Distinctive	Distinctive
Radiotherapy	Unhelpful	Helpful
Corticosteroids	Therapy of choice	Helpful
Immunosuppressive regimes	Not indicated	Indicated
Prognosis	Good	Poor

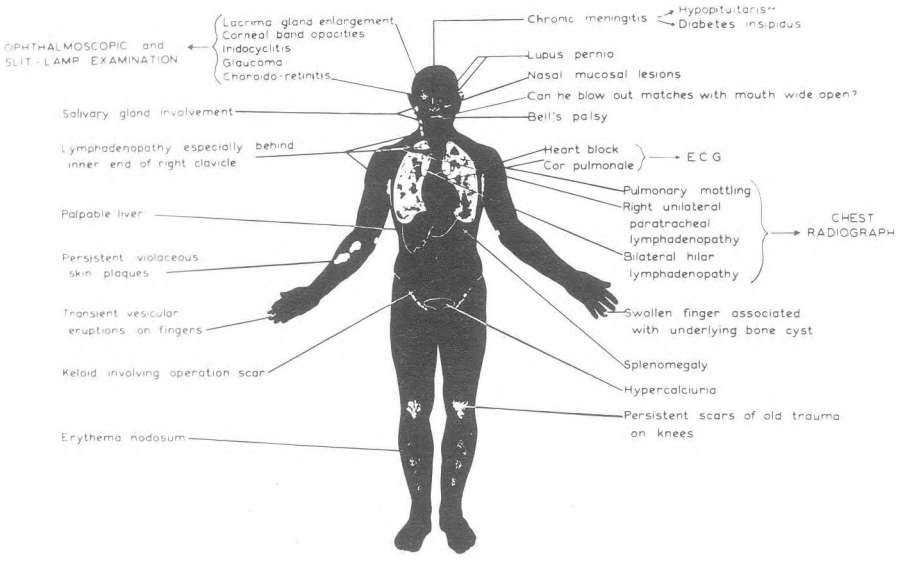


FIG. 197.—Clinical examination of a patient with suspected sarcoidosis.

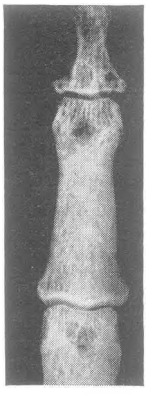


FIG. 198.—Bone cysts in sarcoidosis.

Clinical Manifestations.—It affects lungs, eyes, skin, spleen, lymph nodes, lacrimal and parotid glands, central nervous system so it presents to surgeons of different disciplines (fig. 196). Examination of a patient with suspected sarcoidosis comprises full physical examination (fig. 197), slit lamp examination of the eyes and chest radiography.

Diagnosis is based on the twin criteria of a compatible clinical picture and histological evidence of sarcoid tissue; the latter is obtained by biopsy of skin, lymph node or liver or by scalene node biopsy, mediastinoscopy or fibreoptic bronchoscopy. The Kveim-Siltzbach skin test is a simple specific outpatient technique which is positive in 80% of patients with sarcoidosis. It is an index of activity of the disease. The sarcoid granuloma in the pulmonary capillary bed produces angiotensin-converting enzyme, which can be measured in the serum (SACE). Elevated SACE levels are noted in about one-half of patients with sarcoidosis. Like the Kveim-Siltzbach test, it reflects activity of the disease. Its production is suppressed by corticosteroid therapy, so SACE is a useful monitor of the response to treatment and of relapse when treatment has been discontinued.

Differential Diagnosis.—Sarcoidosis must be distinguished from a non-specific sarcoid-tissue reaction and from Hodgkin's disease (see tables).

Treatment.—Corticosteroids relieve symptoms, suppress inflammation and granuloma formation, and are used for uveitis, pulmonary infiltration, hypercalciuria and renal calculi, disfiguring skin lesions, neurosarcoidosis, myocardial sarcoidosis, and involvement of spleen, lymph nodes, parotid and lacrimal glands. Alternative treatments comprise oxyphenbutazone for acute exudative disease and chloroquine or potassium-p-amino-benzoate (Potaba) for chronic fibrotic sarcoidosis.

CHAPTER 13

THE HAND. HAND INJURIES. THE FOOT

INFECTIONS OF THE HAND

INFECTIONS of the hand are encountered most commonly in manual workers and housewives who frequently suffer small abrasions or pricks in the course of their work. The infective organism is the *Staph. aureus* in 80% of cases, *Strep. pyogenes* and gram-negative bacilli. Unless the infection can be aborted suppuration will follow. The early detection of pus and its accurate localisation are of cardinal importance. Œdema is a common feature in hand infections, being most evident on the dorsum of the hand (fig. 199) irrespective of the site of the lesion, and is a potent cause of subsequent stiffness of the digits.

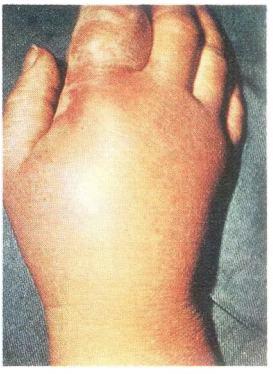


FIG. 199.—Pitting œdema of the dorsum is usually due to infection on the palmar side of the hand.
(T. J. McNair, F.R.C.S., Edinburgh.)

Types of Infection

Acute Paronychia is the most common infection of the hand. The infection, which is subcuticular and under the eponychium, arises from a hangnail (fig. 200), careless nail paring, or an unsterile manicure instrument. Suppuration usually follows, and confined by the adherence of the eponychium to the base of the nail, the pus tends to track around[1] the cutaneous margin and, in 40% of cases, under the nail.

FIG. 200.—Paronychia. Often organisms enter through a 'hang-nail', as in this case.

Treatment.—Cloxacillin or tetracycline given early may abort the infection. If pus has formed it must be let out (see principles of treatment p. 206).

At operation only the eponychium is stripped gently and completely away from the base of the nail. All pus is wiped away; loose cuticle is cut away, and there should be a pocket under the corner of the nail fold, a wedge of overlying skin is removed to ensure healing from the bottom. The loose and ragged eponychium is then excised with delicate, sharp-pointed scissors. When pus has extended beneath the nail, the undermined portion must be excised with fine-pointed, but strong, scissors. 'Floating' nail is dead nail; consequently all unattached nail must be excised. Only when pus has extended beneath half or more of the width of the nail is excision of the proximal third of the nail required.

[1] The colloquial American term for paronychia is 'run around'.

Chronic Paronychia[1].—The history is measured by months, rather than days, and the onset is insidious[2]; seldom does it follow acute paronychia. Today the housewife who does not wear rubber gloves when 'washing-up' is the usual sufferer. Antibiotic therapy has little or no effect, but operative treatment following the lines detailed above is often successful if the infection is a bacterial one. In many cases, however, the infection is due to a monilia or yeast (p. 36). This can be settled by microscopical examination of scrapings and/or special cultures for fungi.

A useful method of treating these indolent infections is by 1:500 Bradosol (domiphen bromide) in spirit, or Penotrane tincture (hydraphen) dropped into the nail-fold twice daily. Nystatin ointment (Squibb) can also be tried. When the pockets become filled with granulations the treatment is discontinued, and the hands must be kept as dry as possible until epithelialisation occurs.

Subcutaneous infections and abscesses can occur at various levels (fig. 201). The volar surface of the hands of manual workers is often covered

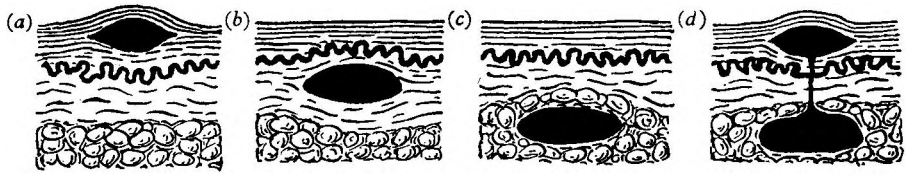


FIG. 201.—Superficial abscesses at various levels. (*a*) Intraepidermal (purulent blister), (*b*) Intradermal, (*c*) Subcutaneous, (*d*) The superficial loculus of a collar-stud abscess.
(*After R. S. Pilcher, F.R.C.S., London.*)

with greatly thickened epithelium. Especially in such individuals, a subcutaneous abscess may burst through the dermis and extend in the layers of the epidermis (fig. 201 (*d*)), in which event it is impossible to differentiate it from a purulent blister until the deeper loculus has been discovered at operation.

Treatment.—The abscess is opened by transfixion. The resulting undermined flaps are cut away with scissors. The unroofed cavity is swabbed free from pus which is examined by culture for the identification of the bacteria and for tests of their sensitivity to various antibiotics. Then it is explored for a sinus leading to a deeper loculus. If such is found, the communicating channel is stretched by inserting and opening the jaws of a small hæmostat.

Dorsal infection is usually a furuncle or boil of the overlying skin. Incision is only necessary if pus is present (see principles of treatment p. 206).

Infection of the terminal pulp space (*syn.* Felon[3]) is the second most frequent infection of the hand (about 25 per cent. of all cases). The index finger and the thumb are affected most often. The origin of the infection is usually a prick.

Surgical Anatomy.—The deep fascia, which is attached to the thin skin of the distal flexion crease, fuses with the periosteum just distal to the insertion of the deep

[1] Called by some dermatologists (quite descriptively) chronic *peri*onychia.
[2] A constant look out must be kept for the occasional case resulting from neuropathy, e.g. syringomyelia.
[3] Felon (Latin, *fel* = gall). An abscess near the nail; also, one who has committed a felony.

Robin Sturtevant Pilcher, Contemporary. Emeritus Professor of Surgery, University College Hospital, London.

flexor tendon, thereby closing the terminal pulp compartment at its proximal end. Through the space, which is filled with compact fat, feebly partitioned by fibrous septa, run the terminal branches of the digital artery. Thrombo-arteritis of these vessels accounts for the frequency with which osteomyelitis complicates infection of this closed space. The basal plate of the epiphysis is rarely involved.

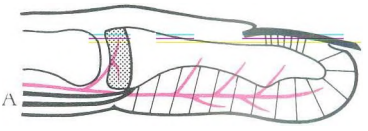


FIG. 202.—Distal pulp space to show how its proximal end is closed by attachment of the deep fascia and by the fibrous septa. The blood supply to the epiphysis does not traverse this area. A = tendon sheath and deep fascia.

Clinical Features.—Dull pain, worse when the hand is dependent, and swelling are the first symptoms. Forty-eight hours later there are severe nocturnal exacerbations of throbbing pain, interfering with sleep. Light pressure over the affected pulp increases the pain. Frequently the corresponding regional lymph node is enlarged and tender. If the pulp is indurated and has lost its normal resilience, pus is present (fig. 203). Untreated, the abscess tends to point towards the centre of the pulp beneath a patch of devitalised skin. A collar-stud abscess then occurs; if still untreated, the abscess bursts.

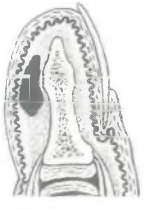


FIG. 203.—The location of pus in terminal pulp-space infection.

Treatment.—In the early stages when there is no localisation large doses of cloxacillin or tetracycline may bring about resolution. Once pus is present operation without delay is the rule (see General Principles, p. 206).

FIG. 204.—Incision for draining a pulp abscess. It is essential to drain the deep loculus of a collar-stud abscess, if such is present.

Operation.—A short incision as indicated in fig. 204 is made through the skin at the point of greatest tenderness. The beginner is warned not to be beguiled by entering only the superficial loculus of a collar-stud abscess. Removal of slough, which is frequently present, is most desirable, but great care must be taken not to traumatise the periosteum.

Osteomyelitis of the terminal phalanx may be a sequel of terminal pulp-space infection (fig. 201). That part of the bone bereft of its blood-supply will become a sequestrum and separates some weeks after the abscess has been opened, in which event the wound continues to discharge. Repeated radiographs and probing (revealing rough bone) will indicate when the sequestrum has separated. Only then must it be removed, after which healing will proceed apace. In case of a child, regeneration of the diaphysis is is possible, provided the periosteum is relatively undamaged. In the adult no regeneration occurs, and the patient is left with a shortened terminal phalanx covered by an ugly curved nail.

Apical subungual infection arises from a prick (including a splinter) beneath the nail, causing infection of the space between the subungual epithelium and the periosteum (fig. 205 (*a*)). The lesion (fig. 205 (*b*)), which is exquisitely painful, gives rise to comparatively little swelling. Tenderness is greatest just beneath the free edge of the nail, and pus comes to the surface here or beneath the nail.

Operation.—A small V is removed from the centre of the free edge of the nail, and a little wedge of the full thickness of the skin overlying the abscess is excised also (fig. 205 (*c*)). The amount of pus and debris evacuated is surprisingly small; com-

monly the abscess cavity extends down to the bone, but osteitis is unusual. Following the operation, relief of symptoms is immediate and the wound heals in about a week.

Infection of the Middle Volar Space of a Digit.—The fibro-fatty tissue occupying this space is more loosely packed than that of the terminal pulp space. The

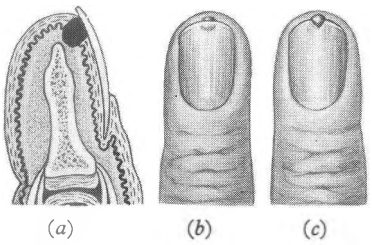


FIG. 205.—(*a*) Showing the anatomical relations of an apical abscess. (*b*) Apical abscess; clinical presentation. (*c*) V-exposure for evacuating the contents of the abscess.

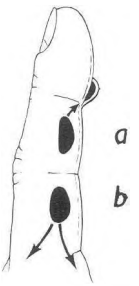


FIG. 206.—Abscess of (*a*) the middle and (*b*) the proximal volar spaces. Showing direction of spread.

middle volar space is separated above and below by fibrous partitions while, like the proximal space, it is shut off from the dorsal cellular tissue by fibrous septa extending from the skin to the periosteum.

Clinical Features.—Infection of this space is much less common than that of the terminal pulp space. The finger is held in semi-flexion. In about one-third of cases attempts to straighten it are painful. There is tender induration over the space, while the soft tissues of the terminal and proximal segments, although swollen, are neither tender nor indurated.

Frequently a purulent blister appears in the distal flexion crease (fig. 206). In early cases it is difficult to distinguish infection of the middle volar space from infection of the underlying flexor tendon sheath; however, in the former, extreme tenderness over the proximal end of the tendon sheath is completely lacking.

Infection of the Proximal Volar Space.—This space is well partitioned from the middle volar space but it communicates freely with the corresponding web spaces (fig. 206). Once localisation has occured infection of the proximal volar space is comparatively easy to diagnose, and frequently the swelling is asymmetrical because of concomitant involvement of a web space (see below).

Operation.—After pus has become localised in either of the above spaces, it should be evacuated through a transverse incision made at the site of greatest tenderness. When the diagnosis is uncertain (localised tenosynovitis cannot be excluded) the space should be explored through a lateral longitudinal incision.

Infection of a Web Space

Surgical Anatomy.—The web spaces are the three triangular regions between the dorsal and volar skin filled with loose fat that bulges between the divisions of the palmar fascia (fig. 207). The spaces, when filled with pus, straddle the deep transverse ligament; consequently, although most of the pus is volar, the abscess points dorsally.

Ætiology.—The infection arises (1) from a skin crack, (2) from a purulent blister or from beneath a callosity on the forepart of the hand, or (3) via a lumbrical canal from an abscess in a proximal volar space.

Clinical Features.—As the constitutional symptoms are severe, patients with this condition are often seen before localisation of the infection has occurred. At this stage there is œdema of the back of the hand. Although the condition is strongly suspected from the location of the tenderness, a precise diagnosis cannot yet be made. The patient should be in bed with

the arm splinted and elevated by suspension. Cloxacillin or tetracycline are given. Once localisation has occurred, the signs of infection of a web space become manifest.

FIG. 207.—The three web spaces between the divisions of the palmar fascia are floored by the transverse ligaments.

Localising Signs.—The base of one finger is swollen, and in severe cases the fingers immediately adjacent to the space are separated. Often there is a fan-shaped blush extending from the web on to the dorsum and a small area of purplish discoloration of the skin over the affected space (fig. 208). The maximum tenderness is found in the web and on the anterior surface of the base of one of the fingers extending a short way into the palm. Untreated, pus can track across the base of the finger into an adjacent web space, and also along the sides of the proximal segments of the digits related to the infected web.

FIG. 208.—Web-space abscess. Note the separation of the related digits.

Operation.—A transverse incision is made on the palmar surface over the affected web space (fig. 207). The incision must be deepened very cautiously until the subcutaneous fat is reached. Only a few strands of palmar fascia need to be divided, and if pus does not flow it is sought with a probe or director. If the diagnosis is correct, probing is soon rewarded by a gush of pus. The opening is enlarged to reveal an abscess cavity, often the size of a thimble. The edges of the wound are cut away, so as to leave a diamond-shaped opening (fig. 209). When the abscess communicates with a dorsal pocket, a counter-incision is advisable. If, as sometimes happens, there is a twin web abscess (one on each side of the digit), each space must be opened, but there is no need to slit the communicating channel.

Deep Palmar Abscess

An abscess beneath the palmar fascia is a serious, but rare (about 1 per cent.), infection of the hand.

Ætiology.—The infection can arise as (1) a penetrating wound; (2) infection via the blood-stream of a hæmatoma in this situation; (3) as a complication of suppurative tenosynovitis (p. 204).

Clinical Features.—As a rule the patient is a manual worker. At an early stage there is intense throbbing pain in, and deep tenderness of, the palm of the hand. Almost from the commencement this is accompanied by obvious œdematous swelling of the dorsum of the hand, which increases rapidly to become greater than that seen in any other infected lesion of the hand—so great as to give rise to what is known as 'the frog hand'. The fingers are held in a flexed position because the palmar fascia is more relaxed in this posture. Extension of the metacarpophalangeal joints is very painful, but extension of the interphalangeal joints is both painless and free. This is a most valuable observation in distinguishing the condition from suppurative tenosynovitis. The temperature is raised, and regional lymphadenitis is commonly present.

FIG. 209.—Incision for draining a web-space abscess.

As tension mounts, the normal concavity of the palm becomes flattened; a time is reached when the imprisoned pus erodes and bursts through the palmar fascia, then suddenly the intense pain passes off, but the palm becomes slightly convex. If the patient is seen for the first time after this happening, there is no means of

distinguishing a deep palmar abscess with a collar-stud extension from an abscess of a subaponeurotic space, except by operation.

Operation.—Under general anæsthesia (or regional block of the median and ulnar nerves at the wrist), a central transverse incision is made in the line of the flexion crease passing across the middle of the palm. Should pus be encountered beneath the aponeurosis, the floor of the abscess (the palmar fascia) must be probed systematically for a sinus leading to the deeper plane. In other circumstances the palmar fascia is divided *in a longitudinal direction* (to avoid digital nerves and blood-vessels). Pus is mopped up. Continued free drainage is ensured by trimming, with scissors, the skin edges as well as those of the incision in the palmar fascia. This obviates premature healing, which otherwise is prone to occur.

Lymphangitis.—Organisms, nearly always streptococci, gain entrance through an abrasion that may be microscopic. Within a few hours the adjacent portion of the hand becomes swollen and painful, and there is often considerable elevation of the temperature. Because superficial lymphatic vessels pursue the shortest course to the dorsum, œdema, which comes on early, is most in evidence on the back of the hand. Later, red streaks, so characteristic of lymphangitis (fig. 179), can be seen coursing up the arm. Especially in lesions of the ulnar half of the hand, the first lymph node to become enlarged and tender is the epitrochlear. In a few instances of infection entering the middle finger, the first lymph node to become swollen is *above* the clavicle (fig. 210), in which case infection is liable to enter the general circulation and give rise to severe constitutional symptoms. The lymphatics of the thumb and index finger pass straight to the axillary nodes. Lymphangitis can occur (*a*) without any other demonstrable manifestation of inflammation, or (*b*) as an accompaniment of the entities to be described, particularly terminal pulp-space infection and fulminating tenosynovitis. Lymphangitis responds well to the conservative measures detailed on p. 185.

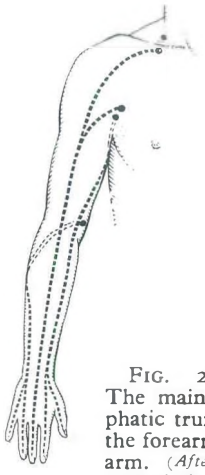


FIG. 210.—The main lymphatic trunks of the forearm and arm. (*After Kanavel and Mason.*)

ACUTE SUPPURATIVE TENOSYNOVITIS

Usually infection of the sheath of a flexor tendon is due to bacteria introduced by the point of a needle or other sharp object penetrating the tendon sheath. Exceptionally the sheath is infected by extension from its terminal pulp space, in some cases from the scalpel transgressing the hallowed ground of the septum that closes the proximal end of the space (fig. 202).

Acute fulminating tenosynovitis involves the whole sheath rapidly, and nearly always the

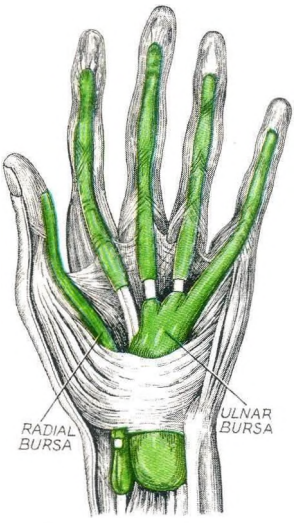


FIG. 211.—The flexor tendon sheaths. This typical arrangement is present in 75 per cent. of cases.

infecting organism is the *staphylococcus aureus* or the *streptococcus hæmolyticus*. The classical local signs are:

1. Symmetrical swelling of the entire finger.
2. Flexion of the finger (the 'hook' sign) with exquisite pain on extension.
3. Tenderness over the infected sheath, especially over its proximal *cul-de-sac* (fig. 211).

Localised suppurative tenosynovitis.—Swelling and tenderness are limited to one portion of the digit, rendering confident diagnosis prior to exploration difficult.

Infection of the ulnar bursa (fig. 211) is characterised by:

1. Œdema of the whole hand, especially the dorsum, due to lymphatic spread.
2. Moderate swelling of the palm.
3. Sometimes a fullness immediately above the flexor retinaculum.
4. The flexed fingers resist extension, the maximum difficulty being experienced in the little, and the least in the index finger.
5. Especially valuable is Kanavel's sign: the area of greatest tenderness is over that part of the ulnar bursa lying between the transverse palmar creases (fig. 212).

FIG. 212.—Kanavel's sign.

It should be noted that the ulnar and radial bursæ intercommunicate in 80 per cent. of cases, and often when an untreated infection of one has persisted for more than forty-eight hours, the other becomes involved also. In no less than 25 per cent. of cases the tendon sheath of the index, or the middle, or the ring finger communicates with the ulnar bursa, which is a fact of great surgical importance.

Infection of the radial bursa (fig. 211) is distinguished by:

1. The distal phalanx of the thumb is held in flexion, with rigidity and inextensibility of the interphalangeal joint. The other digits can be extended fully.
2. Tenderness over the sheath of the flexor pollicis longus.
3. Sometimes swelling just above the flexor retinaculum.

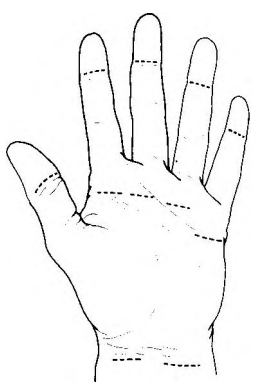


Fig. 213.—Sites of incision for treating acute suppurative tenosynovitis. The sheath is also exposed at the site of the initial trauma when such is discernible.

Treatment of Acute Infected Tenosynovitis.—Full doses of cloxacillin are given. The forearm and hand, the latter being placed in the position of rest (fig. 215), are splinted and elevated. Clinical re-examination is made every six hours. Non-operative treatment is continued only so long as there is a good local and general response. Delay in decompressing an unresponding infection may result in sloughing of the tendon.

Operation.—When the site of puncture of the integument is visible, or has been indicated without hesitation by the patient, the first incision is made in a transverse direction directly over the tendon sheath. When there is no certain information as to where the sheath was punctured, the more distal of the two relevant incisions shown in fig. 213 is made and deepened until the fibrous portion of the sheath is displayed. This is divided and the thin, bulging theca comes into view. Some of the fluid within it is aspirated and sent for bacteriological examination; the theca is then incised in a transverse direction and its edges are pared so as to form a diamond-shaped opening. Pressure is now exerted over that portion of the sheath proximal to the incision, and if this results in a gush of hazy exudate, a second relevant incision (over

Allen Bucknell Kanavel, 1874–1938. Professor of Surgery, North Western University, Chicago, Illinois, U.S.A. The fascial spaces were described in his book 'Infections of the Hand' published in 1933.

the cul-de-sac) is made and similar steps to prevent premature closure of the wound are taken. Employing a fine catheter, the length of the sheath is irrigated with a solution of antibiotic.

Complications of Suppurative Tenosynovitis

Involvement of the Forearm from the Hand.—When a radial or ulnar bursa, distended with pus bursts, pus travels up the forearm between the flexor profundus ventrally and the pronator quadratus and interosseous membrane dorsally. It is here, in the space of Parona, that a quantity of pus can collect without giving rise to much swelling. There is, however, brawny induration above the wrist, unless the original lesion has been incised and continues to discharge pus. Therefore, in cases of infection of the radial or ulnar bursa, if pus can be expressed by pressure over the wrist at the time of operation or subsequently, it is essential that the forearm be drained by making the incisions shown in fig. 214 and deepening them until the periosteum is reached. A hæmostat is then thrust beneath the flexor tendons, and the jaws of the forceps are opened, as a result of which the proximal extremity of the infected bursa (or bursæ) is ruptured thoroughly into the space beneath the flexor tendons.

FIG. 214.—Incisions for draining the space of Parona.

Continuation of Suppuration.—Provided the principles set out above concerning early decompression of infected tendon sheaths have been followed, continued suppuration is rare. If it occurs, the first point to consider in most situations is the possibility of extension of the infection to a fascial space or another tendon sheath. Should suppuration continue for fourteen days, the hand should be radiographed for evidence of bone necrosis. In relevant cases, the possibility of a non-opaque retained foreign body should also be borne in mind. Sloughing tendon is a potent source of prolonged suppuration and much time will be saved by excising the diseased portion, care being taken to anchor its proximal end by sutures to prevent the cut end being carried into the forearm by muscular contraction, and thereby spreading infection.

Suppurative arthritis in a related joint occurs occasionally as a complication of suppurative tenosynovitis. In these circumstances timely amputation of any digit except the thumb will reduce the period of disability (S. Bunnell).

A Stiff Digit Results.—In the case of a finger, it should be remembered that in many walks of life total amputation of a digit is less of a handicap than a stiff finger, but amputation should seldom be undertaken until the infection has subsided completely. In the case of a thumb, the surgeon's watchword for infection as well as trauma is always ' Save all possible '.

Paralysis of the Median Nerve.—When signs of median nerve palsy develop in a case of infection of the hand, early decompression of the carpal tunnel by severing the flexor retinaculum is recommended (D. Bailey). In these circumstances, involvement of the median nerve is due to compression of the nerve by the distended radial or the ulnar bursa, or (more frequently) by both bursæ, which of course must be drained thoroughly at the same time.

GENERAL PRINCIPLES OF TREATMENT IN ALL CASES OF INFECTIONS OF THE HAND

The five principles in the treatment of infections of the hand can be summarised as follows :

(i) Antibiotic therapy.
(ii) Provision of rest and elevation to the affected limb.
(iii) Early recognition of the presence of pus and its accurate localisation.
(iv) Evacuation of pus and, in the case of fascial spaces, debridement of the walls of the abscess cavity.
(v) Adequate after-treatment.

To consider these principles in more detail:

Francesco Parona, 1861–1910. Surgeon, Ospedale Maggiore Novara and at Milan, Italy.
Sterling Bunnell, 1882–1957. Consultant in Hand Surgery to the Surgeon-General of the United States Army and to the United States Navy.
David Alan Bailey, Contemporary. Surgeon, University College Hospital, London.

Antibiotic Therapy.—Antibiotic therapy is given without delay. Because in over 90 per cent. of cases of infections of the hand the original infection is caused by staphylococci, streptococci and gram-negative organisms, cloxacillin or tetracycline are currently the antibiotics of choice. The final choice of antibiotic depends upon the sensitivity tests. It is futile, damaging, and often disastrous to rely on antibiotics when suppuration has occurred. *If there is pus in any part of the hand it must be evacuated.*

Rest and Elevation of the Hand.—If it is considered possible that resolution will occur, and also following operation, the hand must be placed in the position of rest (fig. 215). When it is anticipated that in all probability some portion of the hand will become stiff, as soon as the ultra-acute

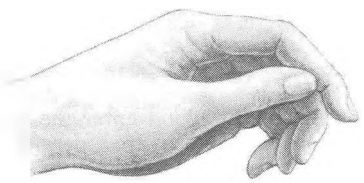


FIG. 215.—The position of rest taken up by an acutely inflamed hand. The index finger is not flexed as much as the others.

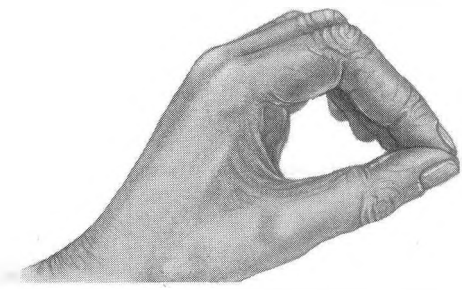


FIG. 216.—The position of function.

stage has passed the digits should be arranged in the position of function (fig. 216). For ambulatory patients a light plaster-of-Paris slab, moulded to fit the volar surface of the hand and forearm, cannot be bettered. In addition, the forearm is supported in a sling as high as possible towards the opposite shoulder, in order to lessen œdema. For in-patients a Cramer wire splint, which is readily suspended, is both efficient and comfortable. Full elevation in the manner shown (fig. 217) decreases œdema and lessens the throbbing pain.

These remarks concerning elevation do not apply to cases of uncomplicated paronychia and minor superficial abscesses; for these an ordinary sling is all that is required.

In all cases rest for an inflamed hand should be insisted upon. When the acute phase has abated, gentle voluntary movements are encouraged.

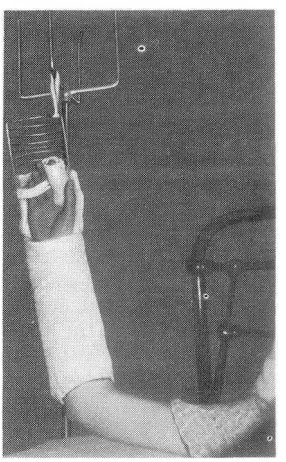


FIG. 217. — Suppurative tenosynovitis being treated by immobilisation and elevation.

Anæsthesia.—For the distal part of the finger regional anæsthesia is employed, using 2 per cent. procaine or xylocaine (*without adrenaline*, as vaso-constriction is harmful). After raising a weal, the hypodermic needle is introduced at the relevant dual points shown in fig. 218. While injecting the anæsthetic solution the needle is advanced distally and forward until it is judged that the digital nerve has been reached; 0·75 ml. of the anæsthetic solution is deposited here. The procedure is repeated on the contralateral aspect of the affected finger. In the case of an abscess of the hand itself, a general anæsthetic is administered, or a regional block of the median and ulnar nerves at the wrist is under-

Freidrich Cramer, 1847–1903. Surgeon, St. Joseph's Hospital, Würzburg, Germany.

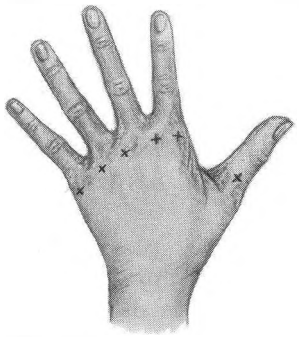


FIG. 218.—Points of puncture for anæsthetising a digit.

taken. On no account should a short general anæsthetic, e.g. nitrous oxide gas, be employed. Complete muscular relaxation and ample time are most desirable when operating in this area.

A bloodless field is essential. Only in the absence of bleeding can the exact site and extent of the lesion be determined and damage to tendon sheaths and nerves be avoided. The cuff of a sphygmomanometer is applied to the upper arm. The limb is then elevated for two minutes, after which the bag is inflated to a pressure of 40 mmHg (5·3 kPa) above the systolic blood-pressure.

Operation is undertaken at a time when there is a high antibiotic level in the blood. With the exception of tendon-sheath infection, it is insufficient merely to evacuate the pus. The operation must be meticulous. Slough is removed unless it is densely adherent and, what is extremely important, granulations are abraded by gauze or scooped away with a curette, avoiding the latter in situations where it might damage the periosteum or a tendon sheath. Only after granulation tissue has been removed, leaving the walls clean and oozing blood, will the injected antibiotic from the blood enter the cavity freely. Provided every nook and cranny has been attended to in this manner, no drainage material is employed, for no further pus is expected to form; merely a little serum, at first containing blood and dead bacteria is all that oozes from a cavity thus treated. This lessens in amount about the third day, when quick healing is to be expected.

After-treatment of Serious Infections of the Hand.—In all cases dry dressings are employed. The dressings are changed at the end of twenty-four hours after operation. Thereafter often an interval of two days can elapse between re-dressings. The patient must be instructed not to get the dressings wet. These instructions differ only in the case of paronychia; in this instance the patient is instructed to wash the hands frequently, dry them thoroughly on a towel kept for the purpose, and re-apply the dressing himself. Physiotherapy and exercises form an important part of the late after-treatment. In rare cases with persisting deformity, rehabilitation for a new job may be necessary.

OTHER INFECTIONS

Human Bites.—Because the wound becomes contaminated with so many types of bacteria (including Vincent's organisms from the mouth), a human bite can prove very dangerous to life or limb. Although not strictly a bite, a common type of injury of this kind is an incised wound over the knuckles resulting from a clenched fist of one combatant striking the front teeth of his opponent. The joint is usually penetrated, but the track closes when the fingers are extended. In such a case the wound must be excised and, if the capsule has been penetrated, a portion of the capsule must be included in the debridement. Because of heavy contamination, primary closure of the wound resulting from excision of a human bite is inadvisable (see Chapter 1).

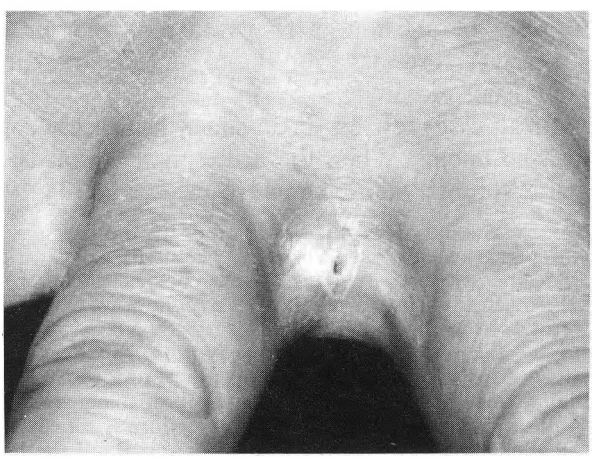


FIG. 219.—Interdigital pilonidal sinus in a barber.

Orf (Contagious Pustu-

lar Dermatitis of Sheep).—A virus disease of sheep transmitted to humans who handle infected animals and their skins or carcases. It forms granulomas on the hands.

Barber's Pilonidal Sinus.—Hair clippings have a bevelled extremity like the point of a hypodermic needle. Barber's pilonidal sinus is due to the customers' clippings penetrating the skin, most frequently the web between digits 3 and 4 of the left hand. In the uninflamed state the lesion is marked by a small black dot with a collarette of epithelial scales around it; a cyst-like nodule can be palpated beneath the visible lesion. Recurrent attacks of subacute or acute inflammation (fig. 219) in the sinus cause the patient to seek relief by excision. (For the common variety of pilonidal sinus see Chapter 52.)

HAND INJURIES

Classification (Based on Rank and Wakefield)

1. *Tidy Injuries.*—These are due to sharp agents, e.g. glass, knives, etc. The cuts are clean and incised. Tendons, nerves and blood-vessels (bone seldom) may be involved. Common in children, these injuries must receive the correct primary treatment.

2. *Untidy Injuries.*—These injuries are common in industry. The skin is often ragged and there may be multiple fractures. It is essential to get correct skin closure after excision of all the devitalised tissues, by whichever method is applicable—suture, grafting or flap (p. 133). Primary repair of nerves and tendons in these injuries is *not* advisable.

3. *Indeterminable Injuries*, for example the severe crush or burn (fig. 220). It is impossible to know early on the full extent of the injury. One must wait to see what remains viable. During the observation stage, elevation of the arm and antibiotic cover are vital. It may be necessary to wait up to 3 weeks before proceeding with excision of dead tissue and repair.

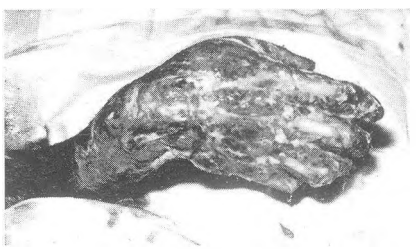


FIG. 220.—Indeterminable injury of the hand. (*E. Melmed, F.R.C.S., Dallas.*)

Evaluation of the Injury.—Careful examination of the injured hand is essential in order to ascertain the extent of injury of the following:—

1. *Nerves.*—The patient may be in too much pain and may be too apprehensive for accurate sensory testing. Motor testing may be impossible because of associated injuries to tendons or bones.

2. *Arteries.*—Arterial damage is suspected by profuse hæmorrhage. The state of the blood-supply to a finger distal to a laceration should always be carefully noted.

3. *Tendons and their Sheaths.*—Tendon sheaths may be lacerated without the tendon that they contain being involved. This injury can only be detected when the laceration is explored. Division of a tendon will result in lack of active movement whilst the appropriate passive movement remains possible although painful. The tendon may be visible in the wound. Isolated division of the superficial flexor tendons may be difficult to detect clinically,

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Allan Ross Wakefield, Contemporary. Plastic Surgeon, Royal Children's Hospital, Melbourne, Australia.

since flexion of the involved fingers will appear to be normal on casual examination.

4. *Bones.*—The presence of a bony injury can usually be detected clinically and can always be detected radiologically.

5. *Joints.*—Injuries to joints may be clinically obvious, or may only be detected when the wound is explored.

Treatment, in order of importance at this time, is as follows:—

1. *Repair of Skin.*—Skin cover is *vital* as raw areas predispose to infection and fibrosis. Fibrosis is the big enemy of hand surgery.

2. *Treatment of bone and joint injuries.*

3. *Repair of nerves.*

4. *Repair of tendons.*

Principles of Technique

Tourniquet.—The use of a tourniquet in hand surgery is essential.[1] The maximum safe time for a tourniquet is 1·5 hours. Strict recording of the time of application, plus constant reminders to the surgeon of the progress of time (15 min. intervals) will avoid the risk of too long application. In finger surgery, rubber tubing can be tied around the base of the finger to give an adequate bloodless field.

Anæsthetic.—The type of anæsthesia varies with the type of injury and the general condition of the patient. If a tourniquet is used, it is often more comfortable for the patient to be under a general anæsthetic. Adequate sedation is usually necessary for a good brachial plexus or local block. Local blocks, or intravenous anæsthesia, with a tourniquet are very useful. Adrenaline in a local anæsthetic in finger surgery is strictly contraindicated.

Careful Wound Toilet.—The skin, the fingernails and the wound must be thoroughly cleansed. Any of the accepted antiseptics is adequate, e.g. Hibitane. It is very important to get rid of all the debris.

Excision (p. 4).—All devitalised tissue must be excised in untidy injuries.

Hæmostasis is essential. In an emergency, direct pressure applied over the bleeding area will stop hæmorrhage (p. 74). *Not* in any circumstances should the operator grab at bleeding points with artery forceps. After release of the tourniquet, all bleeding points are ligated.

Antibiotics and Antitetanus Toxoid.—These should be administered routinely (pp. 7 and 31).

REPARATIVE SURGERY

Skin Loss

Skin Loss without Involvement or Exposure of the Deeper Structures.—On the *dorsum* of the hand a split thickness (Thiersch) graft is the method of choice, due to the simplicity and speed. These grafts are unlikely to contract as the pull of the strong flexors of the fingers overcomes this tendency. On the *palm*, a split thickness graft will contract, and thus it is necessary to use a thicker skin, viz. a full thickness (Wolfe) graft (p. 132). This type of graft is more difficult in that its vascularity is more precarious and thus hæmostasis is essential, and adequate fixation and immobilisation vital to success.

Finger Tips—see p. 211.

Skin Loss with Exposure of Deeper Tissues.—Split thickness grafts will not take on the following—bare bone (devoid of periosteum), bare cartilage, or bare tendon (devoid of paratenon), and necessitates the choice between the following:—(*a*) *Local Flaps*: e.g. cross finger flaps. These are extremely valuable in covering bare flexor tendon in the finger. Flaps of the dorsum and the palm are very difficult as the blood supply can easily be misjudged and necrosis will follow.

(*b*) *Distant Pedicle Flaps and transplanted flaps*, see p. 133. These must be reserved for severe injuries.

[1] Bunnell (p. 206) said, 'Can a jeweller repair a watch in a pool of ink?'

Fractures (p. 353).

Nerve Injury (Chap. 28).—In tidy injuries primary suture is preferable. Digital nerves, easily identified at the initial operation, may be impossible to find at a secondary procedure.

Tendon Injuries

Timing of Tendon Surgery.—Tidy injuries may be dealt with immediately. In untidy and indeterminable injuries repair is delayed until the wound has healed.

Extensor Tendons.—Primary suture usually gives good results as these tendons are highly elastic and vascular and have a good paratenon. Moreover, the tendons act on the metacarpo-phalangeal joint and have a short movement. (The inter-phalangeal joints are extended by the interossei and lumbricals.)

Flexor Tendons. (*a*) *Palm and Wrist*: Simple suture of lacerated tendons give good results as there is abundant areolar tissue which prevents adhesions. When multiple tendons are severed, it is advisable to suture only the profundus tendons.

(*b*) *Divided profundus distal to the proximal interphalangeal joint*: Simple suture is the method of choice.

(*c*) *Tendon injury between the distal palmar crease and the proximal interphalangeal joint* (the ' danger area ' for tendon division): The correct procedure to be adopted is the subject of diverse surgical opinion and only an outline is given here.

(i) When both tendons are divided, most surgeons advocate excision of the sublimis tendon. Simple suture of the profundus is not favoured due to adhesion formation at the injury site with poor functional results. Most authorities advocate tendon grafting as a primary procedure.

(ii) When profundus only is divided, it may be advisable to do nothing (depending on the demand on the digit). Excision of the sublimis and primary grafting has been disappointing and it does not appear to warrant sacrifice of sublimis action. However, excision of half of the sublimis tunnel allowing primary grafting through the other half of the tunnel (Harrison) may prove to be the procedure of choice.

Thumb.—The tendon can either be sutured or grafted (if primary suture is not possible). Either way, the results are found to be adequate by most patients.

Post-operative Care

Elevation and Prevention of Stiffness.—Œdema occurs immediately following injury (and operation). Elevation is thus essential. During the phase of elevation, immobilisation is important as movement may lead to further hyperæmia, etc., with further œdema. This phase lasts 2–4 days. Immobilisation should continue until the wound is healed, i.e. up to 21 days with untidy or indeterminable injuries. *The aim is for early healing, not for early movement.* When bandages are removed some residual swelling is usually present which causes pain on movement. Active movement must be encouraged, however. After severe injuries the hand will need additional intensive physiotherapy, i.e. active exercises, combined with warmth and elevation.

Position of Splinting.—This is very important. The hand must be splinted with the metacarpo-phalangeal joints in 80° flexion, and the inter-phalangeal joints fully extended. This is the position in which the collateral ligaments are fully stretched and thus, should fibrosis and stiffness occur, it will be maximum in that position and on restarting active movement, it should then be possible to regain a range of full movement.

Finger-tip Injuries.—On the thumb and index finger, a tender scar is a nuisance and good sensation is essential for fine movements. In judging what operation is best suited to his finger-tip loss, much will depend on the use to which a patient will put

Stewart Hamilton Harrison, Contemporary. Plastic Surgeon, Mount Vernon Hospital, Northwood, Middlesex, England.

his hands, e.g. a miner whose bread winning depends on early return to work which does not require exquisite finger-tip sensation, will not appreciate the most successful delicate grafting which may take him off work for several weeks.

(1) If skin only is lost, then a free split-thickness graft is all that is needed. With time this may contract and hardly be noticeable.

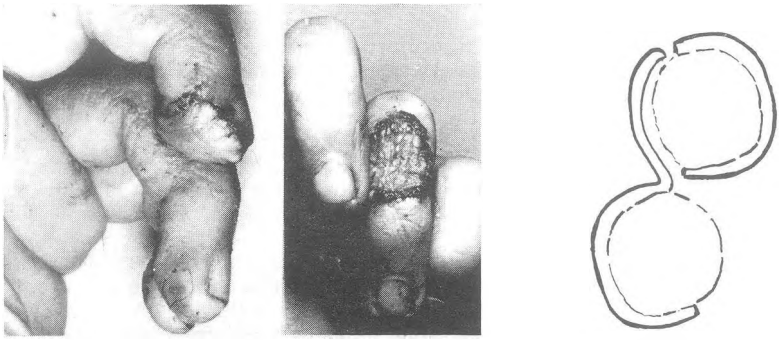


FIG. 221.—Guillotine injury of finger tip covered by a flap from the adjacent finger (see diagram), the bared extensor donor area being covered by a free split-thickness graft. (*E. Melmed, F.R.C.S., Dallas.*)

(2) When the pulp is lost, as well as the skin, then a Wolfe graft may be needed. The donor site is usually skin from the forearm.

(3) In ' Guillotine ' injuries, with loss of pulp and bone, one must take into account the type of work of the patient:

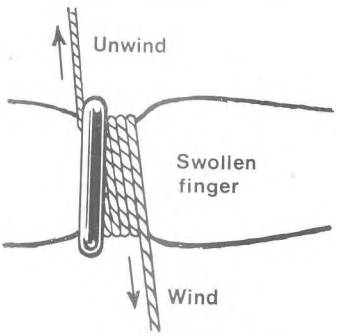


FIG. 222.—The string method for removing a ring. (*F. Exner and the 'Medical Tribune'.*)

(*a*) Free grafting will preserve length. This is usually the most advisable procedure. However, the graft may adhere to bone and may remain tender. In children the graft may contract with time and not need any further treatment. If not suitable, a flap graft can always be applied at a later date.

(*b*) Repair by flap may give a better result, but is time-consuming and may be very difficult. It gives the best results especially for clean cut thumb and index losses. It cannot be used in older people due to stiffness, and in children it may result in an unsightly donor area. Fig. 221 depicts one of the different flap techniques. Another is the Kutler repair, which involves the advancement over the cut end of two lateral triangles (bases distal) of skin and subcutaneous tissues retaining an intact neurovascular connection, a **V—Y** plasty.

(*c*) The principle of sacrificing length for primary closure, by cutting back until the flaps will close, is only employed when it is imperative to get the patient back to work early.

Stuck Finger.—This emergency is common in children who poke fingers into small holes, but it is also encountered when wedding and other rings require to be removed (e.g. for operations). Soap or some fat may ease the ring. The string method (fig. 222) is nearly always successful and should be tried before resorting to saws (Exner).

Microsurgery in Hand, and Foot, Injuries.— The value of the operating microscope and microsurgical technique in the restoration of severed digits, hands and feet speaks for itself in fig. 223. Microsurgery is a rapidly expanding multidisciplinary area of modern surgery.

Frederick Blythe Exner, Contemporary. Radiologist, Seattle, Washington, U.S.A.
William Kutler, 1903–1968. Surgeon, Polyclinic, Cleveland, Ohio, U.S.A. Described this method of repairing amputated fingers in 1944.

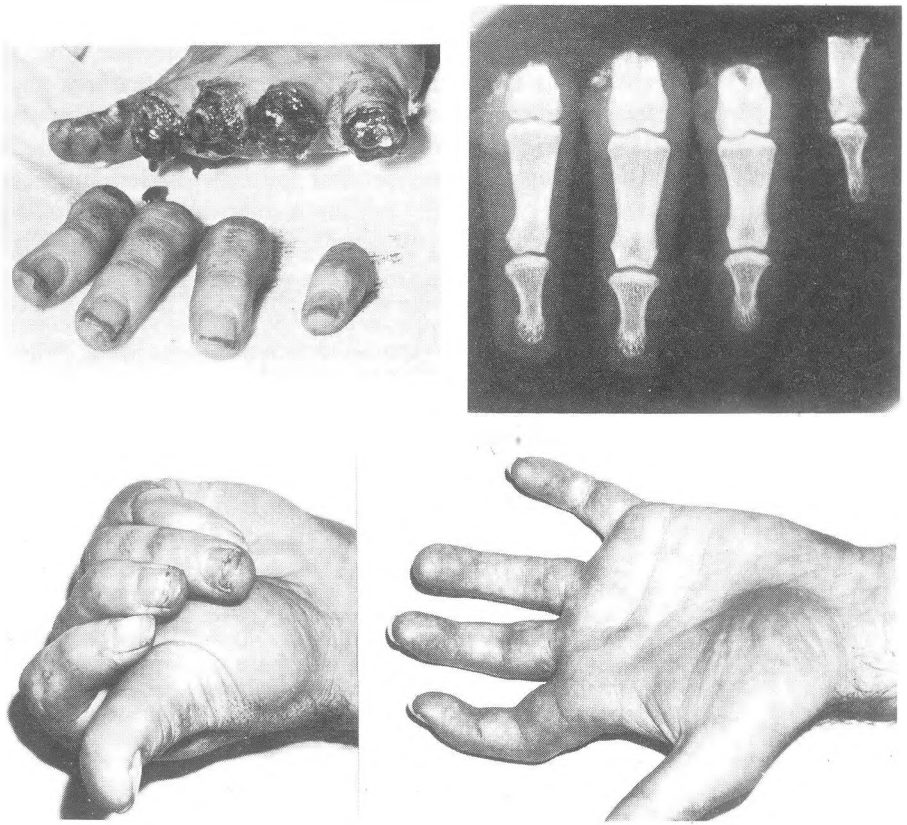


FIG. 223.—(*a*) Saw-cut amputations of 4 fingers in a young man. (*b*) x-ray of digits amputated through proximal phalanges. (*c*) Successful replantation of fingers with primary reconstruction after 1 year. (*d*) Flexion of replanted fingers of same patient at 3 years.
The severed fingers require continued cooling—*not freezing*—until vascular continuity is restored. Two operating teams are employed. (*B. McC. O'Brien, F.R.C.S., Melbourne, Australia.*)

INFECTIONS OF THE FOOT

Especially in countries where some of the inhabitants go barefooted, infections of the foot are commonplace; they also occur in the shod. In each and all of the infections of the foot about to be described it will be assumed that the reader, remembering the lymphatic drainage of the foot, will examine the lymph nodes of the groin and, in relevant cases, those of the popliteal space. He should also examine the urine for sugar. Furthermore, he must remember that arterial disease may present as an infection of the foot.

The prelude to the treatment of any of these infections is thorough washing of the foot with soap and water or, preferably, a detergent, applied with sterile gauze or cottonwool. Except in trivial infections, bed-rest with elevation of the foot must be insisted upon until the inflammation has subsided.

Infected blister is one of the most common infections of the foot. When the patient's temperature is normal and the content of the blister appears doubtfully purulent, the blister can be aspirated. If the fluid is opalescent, the patient should be given tetracycline and the aspirated fluid sent for bacteriological examination. When a blister is frankly purulent it should be incised.

Paronychia of the great toe is also common. It often occurs as a complication of an ingrowing toe-nail. An abrasion of the eponychium with contaminated scissors is also a frequent cause. The clinical features and treatment do not differ from those of a paronychia in a finger or thumb, except that when an ingrowing toe-nail is present it is necessary to deal with it radically (p. 216). Washing, as described above, is carried out each time the wound is dressed.

Infected adventitious bursa beneath a corn is usually the result of improper chiropody. There are signs of inflammation around the corn, the slightest pressure on which evokes excruciating pain. Drainage is accomplished by paring the corn with a sterile scalpel until pus exudes.

Terminal pulp space infection is rare as compared with that of the hand. When it occurs in the foot, usually it is the great toe that is affected.

Infected Bursa over a Hallux Valgus (p. 327).

Infection of a Web Space.— The web spaces of the foot are four in number, the space between the great toe and the second toe being the largest. The clinical features of infection of this space are similar to those of the hand, but extension from the plantar to the dorsal aspect of the web occurs earlier, and with great regularity. The treatment also is similar. In a diabetic, orthodox drainage is so disappointing that disarticulation of the relative toe, leaving the flaps unsutured, is recommended (fig. 158).

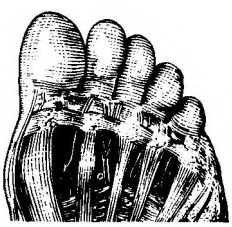


FIG. 224.—The four interdigital subcutaneous spaces, which are separated from the four corresponding web spaces by the transverse plantar ligament.

Infection of a Plantar Interdigital Subcutaneous Space.— There are four interdigital subcutaneous spaces that lie between the five digital slips of the central aponeurosis (fig. 224). Infection of one of these spaces is very common among coolies who work barefooted, especially in urban areas. As a rule the patient states that a sharp stone, a nail, or a thorn penetrated the sole. The patient complains of increasing pain between the shafts of the two metatarsals that bound the infected space. Soon he is unable to walk, and the constitutional symptoms are moderately severe. Exquisite tenderness located over the infected space proclaims the diagnosis. When pus decompresses itself between the two bones into the dorsal subcutaneous space, localisation is more difficult. Drainage must be placed well away from the weight-bearing area, and consequently an incision similar to that used for drainage of a web space is advised, after which a hæmostat is directed into the cavity filled with pus, and its jaws are opened. If the dorsal subcutaneous space is involved, a counter-incision should be made.

Infection of the Heel Space.—The infection is intradermal in one-third of cases, a few of these being a collar-stud extension from a deeper plane. Two-thirds of cases are due to infection of the fat-pad of the heel, which is situated in the subcutaneous portion of the posterior third of the sole. The portal of entry usually is a crack in the overlying calloused skin, and seldom, as one would think, from treading on a thorn or similar object.

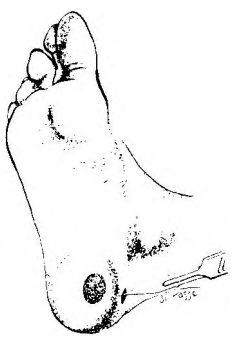


FIG. 225.—The fibrous septa of an abscess of the fat-pad of the heel are so tough as to require division with a scalpel.

Steadily increasing throbbing pain, severe enough to interfere with sleep, is the leading symptom. The patient dare not put his heel to the ground. Swelling

of the soft tissues that cover one or both sides of the calcaneus is present, and in severe cases œdema of the ankle becomes manifest. Tenderness over the space leaves no doubt as to the diagnosis. As a rule, by the time the patient presents, the abscess is ready for incision, which is made through the medial or lateral side of the heel, so that the scar does not come to lie on a weight-bearing area. Fibrous septa within the abscess of the fat-pad need division with a scalpel (fig. 225). The lips of the cutaneous incision should be trimmed elliptically to prevent premature closing of the skin.

Deep Plantar Abscess.—The central plantar space situated deep to the plantar fascia is arranged like an apartment house of four stories, each of which is occupied by the muscles that constitute the flexors of the toes. Infection of the various floors becomes increasingly less common as one proceeds from the ground floor, upwards. For drainage of the central plantar space an incision parallel to, and just above, the medial border of the foot in the neighbourhood of the instep, is made (fig. 226).

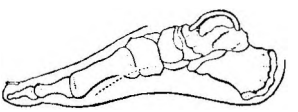


FIG. 226.—Incision for draining the central deep plantar space.

Infections of the Dorsum of the Foot

The dorsal subcutaneous space usually is infected by extension from a subcutaneous interdigital space or a web space, while the *dorsal subaponeurotic space* is infected either from a direct puncture or from involvement by extension from the deep plantar space. To drain the former space the incision should be placed distal to the dorsal venous arch, but in the line of the digital vessels and nerves, in order to avoid them. To drain the latter space it is best to confirm the presence of pus by attempting aspiration, and if diagnostic aspiration is positive, to make a longitudinal incision alongside the needle.

Leprosy not infrequently attacks the toes, and the initial manifestation may simulate a chronically infected corn. Leprosy is discussed on p. 40.

Madura foot (*syn.* mycetoma pedis) is a chronic granulomatous disease encountered especially in tropical countries, notably in certain parts of India or Africa, but with increasing frequency in territories where hitherto it has been unrecognised, such as the Southern United States, South America, and Cuba. Most cases are caused by a filamentous organism (*Nocardia maduræ*) resembling actinomyces and abounding in road dust. In nine out of ten cases the organism gains entrance through a prick in those who go about barefooted. The first manifestation is a firm, painless, rather pale nodule. Soon other nodules appear. Later, the nodules become surmounted by vesicles which burst to form discharging sinuses. In the watery discharge granules can be discovered, sometimes only with perseverance. The granules may be yellow, red, or black. In 'black' madura foot, as it is called, spread is mainly in the subcutaneous plane; in the yellow and red varieties the infection burrows deeply, and bone necrosis ensues. As in actinomycosis, there is no lymphadenitis, but unlike actinomycosis, dissemination to other parts of the body does not occur. Gross swelling of the foot with flattening or convexity of the instep is characteristic. Sooner or later secondary infection supervenes, with rapid deterioration of the local condition.

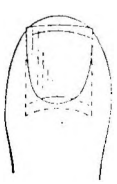


FIG. 227.—The relation of the nail to the nail fold and nail walls. Note the corners of the germinal envelope. (*Courtesy British Journal of Surgery.*)

Treatment.—A wide-spectrum antibiotic to deal with secondary infection, followed by a prolonged course of dapsone (100 mg. b.d.) improves many patients, and may obviate or postpone the necessity for amputation (Cockshott).

Guinea Worm.—This infestation is described on p. 68.

William Peter Cockshott, Contemporary. Professor of Radiology, McMaster University, Hamilton, Ontario, Canada.

INGROWING TOE-NAIL

Ingrowing toe-nail (*syn.* embedded toe-nail) of the big toe, usually results from encasing sweaty feet in tight shoes, and is encouraged by cutting the nail short and convexly. The side of the nail curls inwards and grows to form a lateral spike, which causes a painful infection of the overhanging nail fold (fig. 228).

FIG. 228.—Typical ingrowing toenail with infection.

Treatment.—Conservative measures can be successful, but much depends upon the patient's willingness to help himself. The overhanging nail fold is pushed away and reduced in size by daily packing of the lateral groove with a wisp of gauze soaked in a mild antiseptic. The centre plate of the nail is thinned slightly by filing; this encourages the nail to become flat. The end of the nail is cut straight across or concavely. The corners must not be cut back; it is necessary for them to extend over the pulp.

Operation.[1]—The nail is like a letter protruding from a shallow envelope. The envelope is the germinal matrix and this must be included in radical removal of the affected side or the whole of the nail. Failure to remove the

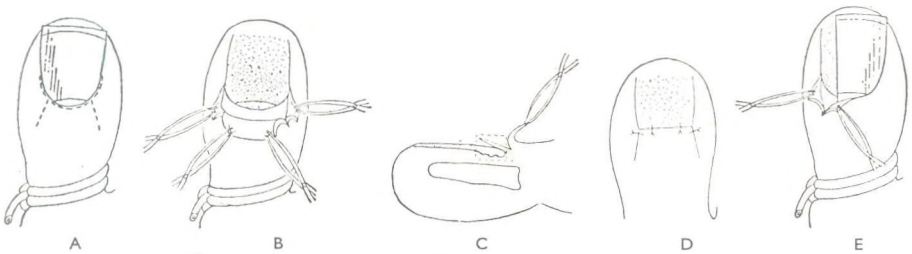


FIG. 229.—Technique of total excision of the germinal matrix. A, Skin incisions. B, Skin-flaps dissected and retracted. The epithelial layer on the deep surface of the flaps is left attached to the matrix. C, Longitudinal section showing the block of tissue to be removed. D, Skin-flaps sutured to cover the raw area. E, Segmental excision of the germinal matrix. The skin-flaps have been raised preparatory to excision of a segment of the germinal matrix. (*Courtesy British Journal of Surgery.*)

corners of the germinal envelope (fig. 227) will result in the recurrence of a nail spike. The technique (Fowler) is portrayed in fig. 229. A tourniquet is essential in order that the germinal envelope may be seen and dissected out in its entirety.

Onychogryphosis is a thickened and crooked overgrowth of a toe-nail, usually that of the big toe. Trauma and fungus infection are implicated as causes. It occurs in elderly people, especially if bed-ridden, and it may become so curled as to resemble a ram's horn. A Gigli saw will cut through the base and the remainder can be controlled by filing. If necessary the nail-bed and nail are removed as above.

[1] Described originally by Quenu in 1887, and again by Zadik in 1950.

Edouard Quenu, 1852–1933. Surgeon, Paris.
Frank Raphael Zadik, Contemporary. Orthopædic Surgeon, Sheffield.
Alan William Fowler, Contemporary. Orthopædic Surgeon, Bridgend General Hospital, Glamorgan, Wales.
Leonardo Gigli, 1863–1908. (*See p. 415.*)

CHAPTER 14

INFECTIONS OF BONES AND JOINTS

ACUTE OSTEOMYELITIS

ACUTE osteomyelitis used to be a common and serious, indeed often a fatal, disease in children. Over recent years there has been a fall in the incidence of the disease, probably due to an improvement in the general health of children. At the same time antibiotics have made the disease less serious: it need never now be fatal and should be curable.

Ætiology.—The bacteria reach the bone by the blood-stream. A primary focus may be obvious in the form of a boil or an infected graze, but not uncommonly no obvious source of infection is evident. Rarely the disease may be secondary to a frank septicæmia or pyæmia. More commonly the blood-borne infection takes the form of a bacteræmia.

It has been suggested that a lowered general resistance on the part of the patient, and local trauma, may predispose to this disease, but the evidence in support of these suggestions is unconvincing.

The usual causative organism is the *staphylococcus aureus*. Other organisms which may be responsible include the streptococcus, pneumococcus, staphylococcus albus and a number of other organisms, no one of which is present commonly.

Pathology.—The disease always, or nearly always, begins in the metaphysis. The infective process progresses through the thickness of the cortex via the Haversian canals and as it does so causes thrombosis of the vessels in the bone. As a consequence, by the time the infection reaches the sub-periosteal region of the bone a variable amount of the cortex may have been infarcted. In the first 24 or 48 hours after the onset of the infection, an inflammatory exudate forms deep to the periosteum, elevating the membrane from the bone. Periosteal elevation is painful and, since the periosteum is inelastic, the inflammatory exudate deep to it is under tension. As a consequence the patient rapidly develops marked toxic signs. Approximately 48 hours after the first symptom, frank pus develops sub-periosteally. Partly as a consequence of the resistance of cartilage to invasion by the septic process, and partly because of the very firm attachment of the periosteum (more accurately the perichondrium) to the epiphyseal plate, transgression of the plate itself and consequent interference with growth is rare. The inflammatory process progresses along the length of the medulla causing venous and arterial thrombosis as it does so. Sub-periosteally, pus tracks both longitudinally and circumferentially around the bone, stripping the periosteum and interrupting the periosteal vessels. Thus progressively larger areas of the cortex become infarcted and involved in the inflammatory process.

In the absence of treatment pus finally bursts through the periosteum, and tracks through the muscles to present subcutaneously. Eventually the skin breaks down and pus discharges from a sinus which connects the bone with the skin surface.

The bone infarct in acute osteomyelitis is known as a *Sequestrum.* Surrounding the sequestrum, the elevated periosteum lays down new bone which entombs the dead bone within. This ensheathing mass of new bone is known as the *Involucrum.* In the places where pus has broken through the periosteum to form a defect in it, sinuses develop which are represented in the involucrum by holes known as *Cloacæ*[1] (fig. 230). The development of such advanced pathology is now rarely seen since modern treatment if adequate, aborts the disease before pus has formed, and certainly before a significant amount of bone has died.

FIG. 230.— Sequestrum enveloped by an involucrum which is pierced by cloacæ.

Two factors are responsible for the chronicity of this disease: the presence of dead, infected bone which cannot be resorbed; and the fact that the intraosseous abscess cavity cannot be obliterated because it has rigid bony walls. As a consequence of these factors the body's normal defence mechanisms (leucocytes and antibodies) together with any antibiotics that may be given therapeutically are unable to reach all the bacteria in the bone. Accordingly, although the disease process may be sterilised in the living bone, recurrence is always likely.

Clinical Features—Pain is the presenting symptom.

It is essential that an accurate history is taken so that the onset of the first complaint of local pain can be timed exactly. The significance of this feature of the history is discussed further under *Treatment.* The pain gradually increases in severity, and the child becomes increasingly febrile and toxic, at a rate dependent upon the toxicity and virulence of the infective organism. It is usual for the mother to seek medical advice within 48 hours of the onset of the first symptom.

Physical Signs.—The essential physical sign is localised tenderness. When the doctor first examines the child, the child is likely to be irritable and to resent examination. It is imperative that the physician should be patient, and gently palpate the child's limbs until the exact area of maximum tenderness has been identified. If this tenderness lies over the metaphysis of a long bone, the diagnosis of acute osteomyelitis should be presumed until it can be proved otherwise. The adjacent joint may contain an effusion, raising the differential diagnosis of suppurative arthritis. The joint itself however is not tender and although the child resists movement of the limb, with patience it is possible to demonstrate that some movement of the joint is allowed. This contrasts with acute suppurative arthritis in which absolutely

[1] Cloaca (Latin) = A drain.

no movement is permitted. The temperature is raised, often markedly so, and an associated increase in the pulse rate occurs. Some days after the onset of the first symptom noticeable swelling and heat may be detected in addition to tenderness. Finally the area of the abscess (for such it is by this time) is fluctuant.

It is absolutely essential that blood cultures should be undertaken before antibiotic treatment is commenced. In order to provide the maximum possible chance of a positive culture, three separate venipunctures should be made and from each venipuncture three aliquots[1] should be cultured separately. The child's body surface should be searched minutely for possible primary foci of infection and if these are found they should be cultured.

Special Investigations.—Other investigations are of no diagnostic value early in the disease. The E.S.R. and white cell count are usually raised but this is entirely non-specific.

X-ray.—There are no abnormal radiological features in the first few days of the infection. As time goes by, new bone can be seen deposited by the elevated periosteum, but this sign does not appear until 2 or 3 weeks after the onset of the disease and will then be demonstrable whether or not the disease has been sterilised: it depends entirely upon the presence or absence of periosteal elevation. Some rarefaction in the bone due to local hyperæmia will also occur after 2 or 3 weeks but again does not distinguish continuing osteomyelitis from the sterilised disease. The radiological appearances of chronic osteomyelitis are dealt with elsewhere (p. 221).

Treatment.—The child is admitted to hospital and the limb is splinted, but in such a way that easy access to the tender area is retained. The outline of the tender area is marked on the skin.

If the patient is first seen within 48 hours of the appearance of the first symptoms, antibiotic treatment is begun immediately after appropriate samples have been taken for blood culture. Acute osteomyelitis is one of the few diseases in which it is justifiable to begin antibiotic treatment without waiting for bacterial sensitivity, a peculiarity which stems from the fact that if the disease can be sterilised within the first 48 hours, complete resolution can be guaranteed. If sterilisation fails or is not attempted in this time, the disease may become chronic, so generating life-long disability and a possible cause of death. The great majority (about 90%) of the isolates from osteomyelitis are *Staphylococcus aureus* and cloxacillin should be administered at a dosage of 500 mg 6-hourly intramuscularly. For penicillin-hypersensitive patients clindamycin 150 mg b.d. may be given intramuscularly. In children under three *Hæmophilus influenzæ* may be a responsible organism and especially affects the small bones of the hands and feet. At the present time ampicillin 250 mg q.d.s. intramuscularly is recommended. Unfortunately antibiotic resistance amongst organisms causing osteomyelitis creates problems. The staphylococci are usually resistant to benzyl penicillin and ampicillin and therefore require a penicillinase-stable penicillin. Most strains of *Hæmophilus influenzæ* are currently susceptible to ampicillin but if failure to respond is thought to be due to a resistant organism, chloramphenicol should be sub-

[1] Aliquot (L). Alius = Other. Quot = How many. In this case 3 other samples.

stituted. Other antibiotics may be substituted if they are dictated by the sensitivity tests.

If the patient is first seen 48 hours or more after the onset of the first symptom, the possibility arises that pus is present. If pus is present, it may be sterilised by antibiotics, but the general surgical principle applies to bone as to other tissues that *an abscess requires surgical evacuation.* The presence of pus may be difficult or impossible to detect with certainty since fluctuation is late to develop. Fluctuation cannot be demonstrated in the early stage of abscess formation because the periosteal membrane is tense, the involved bone is often deep to muscle, and the area is too tender to palpate firmly. Therefore the surgeon has to rely upon his general impression as to the severity of the disease and his knowledge of its duration in deciding either to treat the patient initially with antibiotics, or to combine this therapy with incision of the tender area.

If it is decided to rely on antibiotic therapy alone in the belief that no pus is present, antibiotics should be given and the effect of this treatment upon the toxic signs and upon local tenderness should be watched very closely. If the antibiotic is controlling the disease, and if no pus is present, the temperature will subside to become normal within 2 or 3 days and tenderness will progressively disappear. If, on the other hand, the antibiotics are inappropriate to the sensitivities of the organism or pus is present, the temperature is likely to settle but not to normality: spikes up to 38°C. will continue. If this occurs, the tender area must be explored surgically with a view to evacuating pus if any is present and to obtaining the organism for culture and sensitivity.

Operation.—Operation is carried out under general anæsthesia and is preceded by exsanguination of the limb by elevation and the use of an inflatable tourniquet. An incision is made over the tender area and carried down to the bone where pus is usually found deep to the periosteum. The abscess cavity is fully opened and the pus evacuated. A swab is taken for culture and sensitivity at this stage. There is controversy as to whether or not this procedure should be followed by drilling the cortex in order to enable any pus that may be present in the medullary cavity to drain to the surface. It is the author's opinion that a drill hole should always be made. If no pus is found, no harm is done in practice although in theory a previously sterile medulla might be contaminated. On the other hand if pus wells up from the drill hole, the hole should be enlarged until free drainage appears to have been obtained. The wound is then closed with interrupted sutures over a closed sterile suction drain. Antibiotics and local splintage are continued post-operatively.

Differential Diagnosis

Acute Suppurative Arthritis.—Here the sepsis is intra-articular, and therefore the patient allows no movement of the joint. In the 'sympathetic' effusion associated with acute osteomyelitis, a certain range of painless movement can usually be obtained if the patient is examined gently. The maximum tenderness is near the end of the bone in osteomyelitis rather than over the joint in suppurative arthritis.

Acute Rheumatic Arthritis is usually polyarticular and fleeting in any one joint. There is a history of a sore throat and cardiovascular signs are often present.

Hæmarthrosis may occur in hæmophilia. The patient is usually a known hæmophiliac and aspiration, if necessary, reveals blood.

Scurvy.—Sub-periosteal hæmatomata are sometimes very tender, and if near an epiphysis may be confused with acute osteomyelitis.

Acute Exanthemas and Typhoid Fever.—These conditions may be suspected

on account of the profoundly toxic and even comatose condition of the patient. If careful palpation over a localised area of the end of a long bone induces resentful movements or moaning, the possibility of osteomyelitis should be considered.

Ewing's Tumour.—See p. 264.

Complications.—These may be divided into two types, general and local. The general complications are septicæmia and pyæmia which may give rise to metastatic abscesses. Either complication, if uncontrolled, may prove fatal. Amyloid disease may develop as a complication of chronic osteomyelitis (below).

The local complications include: (1) secondary involvement of the joint if the epiphyseal line is intra-articular, (2) spontaneous fracture which is rare provided the limb is splinted and the disease adequately treated, (3) deformity which, surprisingly, is rare, and (4) chronic osteomyelitis.

Acute Traumatic Osteomyelitis

This condition arises as a result of infected wounds, for example compound fractures, and operations on bones. The constitutional disturbances are less severe than in acute (infective) osteomyelitis, as the causative wound provides some measure of drainage. Treatment consists of more extensive opening of the wound, removal of dead bone, and antibiotics. The prevention of this condition depends upon the adequate initial treatment of compound fractures (p. 347) and upon sterile operating conditions.

CHRONIC OSTEOMYELITIS

Pathology.—Acute hæmatogenous osteomyelitis may pass into chronic osteomyelitis if early treatment is not available, or is inadequate so that infected bone dies to form a sequestrum. The disease may take two forms. The pathology of the more common variety in which a large volume of bone is involved has been described under acute osteomyelitis (p. 217). The incidence of this condition has been greatly reduced by the modern treatment of the acute infection, but some cases remain as a legacy of the pre-antibiotic era, and more will probably occur in the future if the acute infection is inadequately treated. The second variety is known as Brodie's abscess. The infection in this form of the disease is closely contained so as to create a chronic abscess within the bone composed of pus or jelly-like granulation tissue surrounded by sclerotic bone. The lesion may be the sequel to a pyogenic septicæmia from which the patient has recovered, leaving a bone abscess which may remain dormant for years. On the other hand, it may be found in a patient who is known to have had osteomyelitis (but not septicæmia) affecting a bone other than the one in which the Brodie's abscess is discovered.

Clinical Features.—Chronic osteomyelitis may remain quiescent for months or years, but from time to time acute or sub-acute exacerbations occur. An exacerbation is ushered in with constitutional upset and local evidence of inflammation, which may culminate in a discharge of pus, often from a pre-existing sinus. An X-ray sometimes reveals a sequestrum which has separated from the surface of the bone or which lies in a cavity (fig. 231).

Sir Benjamin Brodie, 1783–1862. Surgeon, St. George's Hospital, London.

Tomographs may help to demonstrate a sequestrum and a sinogram may delineate an abscess cavity in the bone.

A Brodie's abscess causes intermittent local pain and occasionally transitory effusions in the adjacent joint during an exacerbation. Examination may reveal tenderness and thickening of the bone. A radiograph is diagnostic and reveals a band of sclerosis surrounding a central lucent area. The mount of sclerosis is variable, ranging from dense sclerosis extending a considerable distance round the cavity (fig. 232) to, more commonly, a faint line of sclerosis at the junction of the abscess with the cancellous bone.

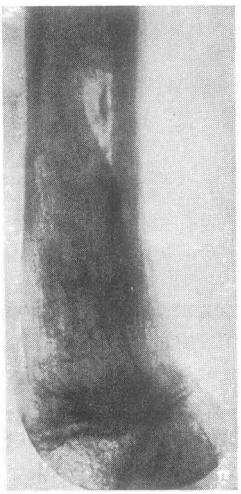


FIG. 231. — Chronic osteomyelitis of the femur with a cavity containing a sequestrum.

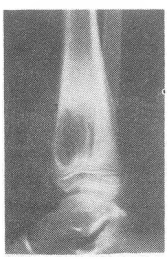


FIG. 232.— Brodie's abscess of lower end of the tibia.

The chronicity of a Brodie's abscess is the result of the physical characteristics of bone, because the abscess can never close by collapse of the walls as happens in soft tissues. Moreover, the infection kills the hard, bony walls of the abscess and provokes new bone deposition, thus preventing leucocytes, antibodies and antibiotics from reaching the contents of the cavity.

Treatment of exacerbations in chronic osteomyelitis consists of immobilisation of the limb and the administration of antibiotics. On this regime the exacerbation often subsides, but only to recur again later in life.

Surgical intervention in chronic osteomyelitis has as its objective the removal of dead bone and the elimination of dead space. Dead bone in the form of a sequestrum may be detected by probing a sinus or by X-ray. Seams of dead bone dispersed within living bone cannot be detected with certainty but may be suspected if an X-ray shows an area of sclerosis. An appropriate antibiotic (which is chosen in the light of the sensitivity of the causative organism) is administered for some days prior to operation. Access to the bone is usually gained through a previous scar. The soft tissues are stripped from the bone, and the involucrum is removed to reach the sequestrum. If a cavity is present, the over-hanging walls are removed with an osteotome, until it is ' saucerised '. Sclerotic bone is removed *en bloc* if this is practicable. The wound is drained and closed in such a way as to eliminate dead space as far as possible.

So difficult is it to guarantee that an operation will cure chronic osteomyelitis affecting a large volume of bone, that operative intervention is not to be considered lightly unless a sequestrum is known to be present. If, however, a sequestrum is present and is removed, sinuses will often close and the disease may be cured. If only a cavity or sclerosis is present in the bone without a sequestrum, the attempt to ' saucerise ' may fail and still leave a sinus. There are many patients for whom, if the discharge is slight

and easily controlled by a dressing, it is preferable to retain the sinus and dressings permanently. Amyloid disease need be feared only when a copious discharge of pus has persisted for some years.

Amputation may be advisable if exacerbations are frequent or prolonged, in order to rid the patient of recurring periods of painful disability, and to forestall the onset of amyloid disease.

A Brodie's abscess should be treated by surgical evacuation and curettage of the cavity under antibiotic cover followed, if the cavity is of moderate size, by packing with cancellous bone chips.

ACUTE SUPPURATIVE ARTHRITIS

Like acute osteomyelitis, this used to be a common disease especially in children, but is now rare. Acute infection of a joint occurs as a result of:

1. Direct infection, as by a penetrating wound or a compound fracture which involves the joint.

2. Local extension, from some neighbouring focus, such as acute arthritis of the hip joint from osteomyelitis of the femoral neck.

3. Blood-borne infection, the usual organisms being the streptococcus, staphylococcus, and pneumococcus, and less commonly the gonococcus and *B. typhosus*.

The knee joint, owing to its large size and exposed position, is the commonest joint to be involved by penetrating wounds, while suppurative arthritis from blood-borne infections is the more common cause in other joints.

Clinical Features.—The patient complains of steadily increasing pain, inability to move the joint and malaise. On examination the patient is often severely toxic with a raised temperature and pulse rate. The joint is held in the position of its greatest capacity (the 'position of ease') and, if subcutaneous, it can be seen to be swollen (see Table below). Palpation reveals increased heat, tenderness and an effusion. Movements are prevented absolutely by muscular spasm, and attempts at either active or passive movement cause severe pain.

Treatment.—After all these lines of treatment the joint must be immobolised until the infection has been cured. As any case of suppurative arthritis may be followed by ankylosis, it is the duty of the surgeon to anticipate this possibility by immobilising the joint in the best position for ankylosis (i.e. the position of optimum function), as indicated in the table on p. 224. The limb is supported and fixed by a suitable splint or appliance in the correct position, an anæsthetic being administered if necessary. Traction, which was commonly used in the past with the idea of holding the joint surfaces apart and preventing damage to the cartilage, is no longer used.

Antibiotics are administered systemically as in acute osteomyelitis (p. 219).

The following lines of treatment are to be considered.

1. *Aspiration*, which is useful for both diagnostic and therapeutic reasons. The nature of the fluid can be ascertained, and the organism cultured to obtain its antibiotic sensitivity. Aspiration reduces the tension within the

Joint	Position of ease	Site of maximum swelling	Position for ankylosis
Shoulder	Adducted	Under the deltoid along the tendon of the biceps and in the axilla	40° to 50° of abduction, with elbow joint just anterior to the coronal plane and hand in front of the mouth.
Elbow	Flexed at a right angle and pronated	On either side of the triceps tendon	90° of extension semi-pronated. If both sides, one elbow at 75° of extension, the other at 135°. These positions enable the patient to reach the external orifices.
Wrist	Slight flexion	Under extensor and flexor tendons	Slightly dorsi-flexed to allow a firm grasp.
Hip	Flexed (fig. 244), adducted, and externally rotated	Upper part of Scarpa's triangle	20° to 30° of flexion to allow sitting, and in neutral position as regards abduction and rotation.
Knee	Flexed	Suprapatellar bursa, and either side of patellar tendon	5° to 10° of flexion to allow foot to clear ground in walking.
Ankle	Slightly plantar-flexed (and inverted at the subtalar joint)	Anteriorly and on either side of the Achilles tendon	At a right angle.

joint, thereby relieving pain, and limiting the stretching of ligaments and capsule. It has the disadvantage that a previously uninfected ' sympathetic ' effusion may be infected if the needle traverses a septic focus on its way into the joint. On balance, the advantages outweigh this disadvantage. If frank pus is aspirated, the joint is opened (see below).

2. *Aspiration and Injection.*—After fluid has been aspirated, antibiotics may be injected into the joint. Repeated injections of antibiotic into a joint are unnecessary, since systemic administration is adequate.

3. *Arthrotomy and Drainage.*—In neglected cases with radiological destruction of bone the joint is opened, washed out, and closed suction drains are placed down to the synovial cavity. This is only done if the joint is found on aspiration to contain frank pus, or if bone destruction has involved the articular surfaces so that some degree of ankylosis is all that can be expected when healing has occurred. Though commonly used 30 years ago, this technique is now less often needed, because the disease, if diagnosed early, can be treated by antibiotics and aspiration. In all cases adequate immobilisation is essential.

Extra-articular abscesses sometimes require to be opened and drained.

In the case of the knee joint, pus is particularly liable to track upwards beneath the quadriceps, where its presence may be overlooked.

4. *Excision.*—In modern medicine this is rarely if ever required but if the condition of the patient deteriorates in spite of treatment, or if suppuration is prolonged, drastic surgical ablation of the diseased bone is necessary.

TUBERCULOUS ARTHRITIS AND OSTEOMYELITIS

Historical and Social Background.—At the end of the 19th century tuberculosis was widespread—especially in towns—because patients with open human pulmonary tubercle remained in the dense urban populations, because milk was infected with bovine tubercle, and because the general health of the population suffered from overcrowding, poor food and air pollution. Since no specific treatment was available until the advent of antibiotics, the disease usually ran a slowly progressive course to death or crippledom. The social consequences surrounded the disease with an aura of dread and even shame which still persists. All medicine could offer was improvement of the general health of the patient by reversing his social circumstances —substituting rural sunlight for urban smoke, rest for work, and good food for poor—in the hope that his own body mechanisms would arrest the disease. 'Arrest' in this context rarely amounted to cure, but rather to an unsteady equilibrium between the patient and his quiescent disease. Against this backgound, sanatoria were constructed and a variety of physical signs and stages in the disease were described. With the advent of antibiotics and early surgery where necessary, the nature of the disease and of its social consequences changed entirely: sanatoria and sunlight became irrelevant; cure became the usual outcome and persistent disability uncommon.

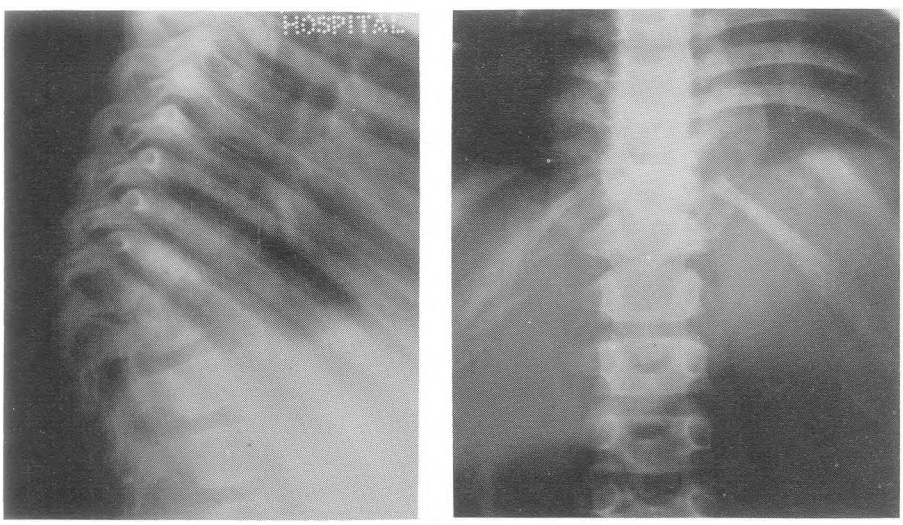
Pathology.—Bone and joint tuberculosis is hæmatogenous in origin. The primary focus is related either to the gastro-intestinal tract if the disease has been acquired by the ingestion of bovine tubercle, or to the lungs if the disease has been acquired by the inhalation of the human strain. Fifty years ago, when cow's milk was often infected, bone and joint tuberculosis in the United Kingdom was usually bovine. With the eradication of bovine tuberculosis in dairy herds and of human pulmonary tuberculosis, bone and joint tuberculosis has become a rarity in the United Kingdom. However, in underdeveloped countries, where bone and joint tuberculosis is still common, it is usually due to the human strain of the organism since little milk is drunk.

The disease starts either in the *synovial membrane* or in *intra-articular bone.* The disease may develop in any synovial joint (especially those with extensive synovial membranes such as the hip and knee), in tendon synovial sheaths (especially those of the finger flexors), or in bursæ (such as that over-lying the greater trochanter). The spine is also commonly involved and tuberculosis here carries the eponymous description of ' Pott's Disease '. The vertebral bodies—almost always those of two neighbouring vertebræ—are involved first.

Percival Pott, 1714–1788. Surgeon, St. Bartholomew's Hospital, London.

Typical tubercles develop in the synovial membrane which becomes bulky and inflamed, and an infected effusion collects in the synovial cavity. If the infection can be diagnosed and cured at this stage, full function may be restored to the joint. If, on the other hand, the pathological process progresses, articular cartilage is destroyed and the adjacent bone is involved. At this stage some loss of function is certain since healing leaves a fibrous ankylosis, not two healthy surfaces of articular cartilage separated by the synovial cavity. If the disease starts in intra-articular bone, the synovial membrane rapidly becomes involved. For practical purposes involvement of both the synovial membrane and of the bone must be assumed when a diagnosis is made of tuberculous arthritis.

In the spine, the diagnosis is rarely made until the bodies of two neighbouring vertebræ are significantly involved so that the end result, at best, is the replacement of an intervertebral disc and of the diseased bone by fibrous tissue. Should treatment for spinal disease be delayed, abscess formation occurs and the vertebral bodies collapse (fig. 233). The pus tracks along tissue planes to present superficially in places often distant from the involved



(*a*) (*b*)

FIG. 233.—Tuberculosis of the 11th and 12th dorsal vertebræ. (*a*) Collapse of two vertebral bodies into a wedge. (*b*) Perispinal abscess shadow.

vertebræ; for example, pus arising from D12/L1 may track along the psoas muscle to present in the groin (fig. 237). Vertebral collapse produces forward angulation of the spine (a 'Kyphus': fig. 234) and the combination of pus formation and spinal angulation compresses and may damage the spinal cord. The cord may also be prejudiced by interference with its blood supply from the anterior spinal arteries. As a consequence paraplegia (Pott's paraplegia) may develop.

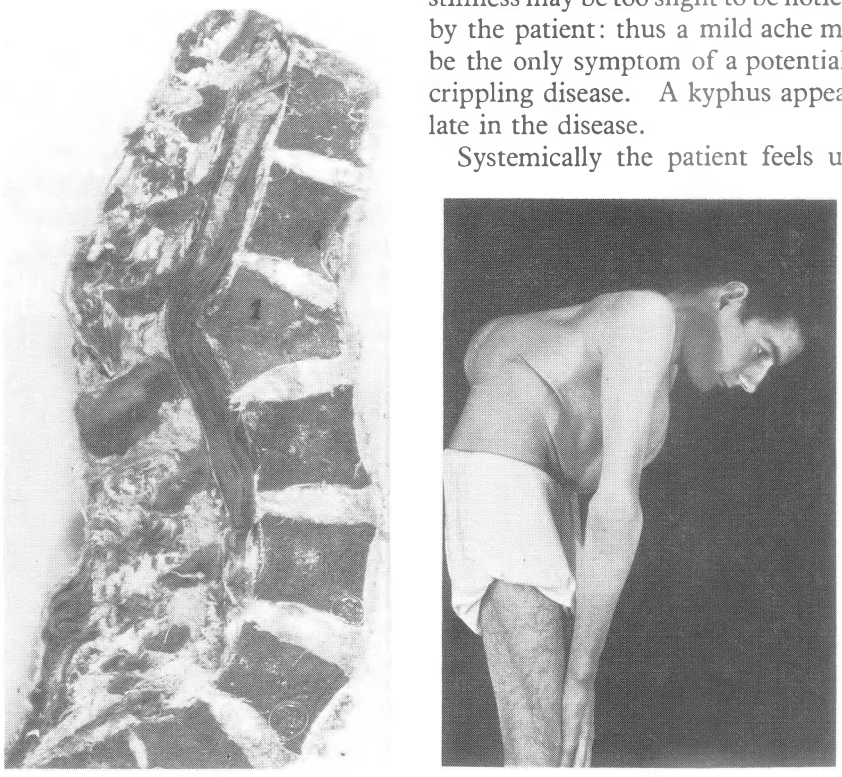
Tuberculosis of the shaft of a long bone occurs in miliary tuberculosis, but is rare.

Clinical Features

Symptoms.—These may arise from the diseased joint, from the primary focus, and from the systemic effects of the disease.

Locally the patient complains of an ache in the joint, at first mild in nature, which is worse on exertion or at night. If the joint is subcutaneous, it may be noticed to be swollen, a feature made more obvious by the wasting of the associated muscles. As the disease progresses the joint becomes increasingly stiff, partly because movement is painful and partly because movement is limited by adhesion formation, muscle spasm and bone destruction. As the bone is destroyed, the joint may dislocate until local deformity is obvious. In the spine swelling is not visible until a considerable quantity of tuberculous pus has collected and stiffness may be too slight to be noticed by the patient: thus a mild ache may be the only symptom of a potentially crippling disease. A kyphus appears late in the disease.

Systemically the patient feels un-



(*a*) (*b*)

FIG. 234.—(*a*) Tuberculosis of the spine. L1 and L2 (1) have collapsed to produce wedging (and hence kyphus). A further tuberculous lesion is present in D12 (2). (*b*) The clinical appearance of a kyphosis due to tuberculosis of the spine.
(*from London Hospital Museum.*)

well in himself, listless and febrile—the latter especially at night when he may have ' night sweats '.

Physical Signs.—If the joint is superficial, the synovial thickening and effusion may be visible. The muscles acting on the joint are markedly

wasted. The joint is held in its position of ease (figs. 235 and 236 and Table, p. 224).

On palpation, the synovial thickening and effusion can again be made out and the joint will be found to be moderately tender. The skin overlying the joint, even if abscess formation has occurred, is not red and is only slightly warm: a feature which is characteristic of tuberculous inflammation and abscess formation, so that such abscesses are known as 'cold abscesses'. Active and passive movement of the joint will be limited and painful.

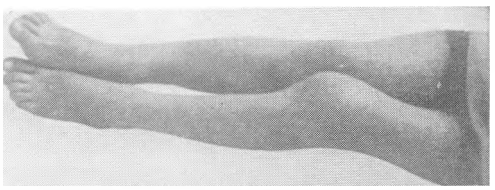


FIG. 235.—Tuberculosis of the left knee joint showing flexion, swelling, and muscle wasting.

In the spine the only physical signs of the disease in its early stages are tenderness on percussion of the spinous processes of the involved vertebræ and minimal limitation of movement. Later, a kyphus may be seen (fig. 234 (*b*)) and abscesses may be visible in the groins (fig. 237) or posteriorly in the triangles of Petit. A kyphus in the lumbar spine may be masked by the normal lumbar lordosis.

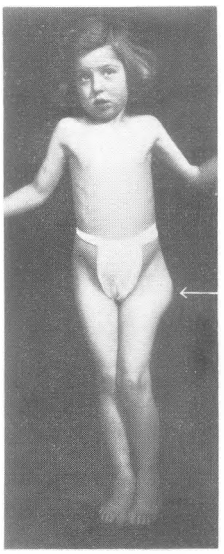


FIG. 236.—Tuberculosis of the left hip. The arrow indicates a periarticular abscess.

FIG. 237.—A psoas abscess pointing in the right groin.

FIG. 238.—Tuberculosis of the wrist joint. Note the loss of density in the carpus (especially in the capitate) compared with the radius and metacarpals.

Special Investigations.—*Hæmatology and Immunology.*—The ESR and white cell count are raised, the latter with a lymphocytosis. The Mantoux test is positive—sometimes violently so. The hæmoglobin concentration should be measured since anæmia is common and requires correction.

Radiology.—The early radiological signs are not dramatic: the bone adjacent to the joint is a little less dense than normal (fig. 238) and it may be possible to make out a soft tissue swelling. As the disease advances, the joint space or disc space narrows and bone destruction becomes visible as an

Jean Louis Petit, 1674–1750. French Surgeon.
Charles Petit Mantoux, 1877–1947. Physician, Le Cannet, France.

area of osteolysis. Thus in the spine a characteristic appearance now develops: the disc space narrows, and lytic lesions, typically anterior, appear in the bone of the adjacent vertebral bodies. Further bony destruction is accompanied by abscess formation so that diseased bone is seen to lie in and around a soft tissue shadow containing loose fragments of bone and calcified soft tissue (figs. 233 (*b*) and 239). At this stage deformity may be radiologically obvious (fig. 233 (*a*)).

A chest X-ray should always be taken and may reveal active tuberculosis.

Histology.—Early accurate diagnosis is imperative, since tuberculous arthritis can be cured (to leave the patient with no loss of function) provided it is adequateiy treated before bone and cartilage are destroyed. Since early in the disease the clinical, hæmatological, immune and radiological features are not diagnostic, histological examination of biopsy material (revealing acid-fast bacilli and typical tubercles) is essential.

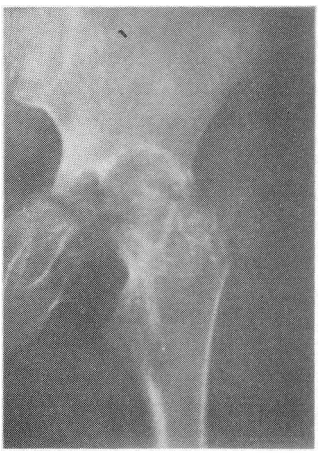


FIG. 239.—X-ray appearances in active tuberculosis of the hip joint in a child.

Methods of Surgical Biopsy

1. *Removal of Lymph Nodes*.—An involved node draining the diseased joint may be removed. The disadvantage of this method is that a negative result does not exclude the presence of the disease.

2. *Arthrotomy and Biopsy of the Synovial Membrane*.—This method allows the appearance of the joint to be noted and provides certain histological diagnosis. The disadvantage is the risk of sinus formation through the operative wound. Provided the operation is carried out under antibiotic cover (see p. 230) this risk is negligible. This is therefore the method of choice.

3. *Needle biopsy* of radiologically involved tissue. This is the method of choice in the spine because direct surgical access may be difficult.

Bacteriology.—Joint aspirate, biopsy material, sputum and urine should be cultured for tubercle bacilli and inoculated into the guinea-pig. A positive culture may take some weeks to obtain. A diagnosis should have been made by this time on the basis of the histology, and treatment started. Bacteriology is therefore confirmatory only.

Differential Diagnosis.—Tuberculous arthritis may be confused with rheumatoid arthritis in a single joint, infective arthritis, and hæmarthroses occurring in hæmophilia. In the spine the differential diagnosis is from osteomyelitis due to other organisms (especially staphylococcus, typhoid bacillus), ankylosing spondylitis, back pain due to disc prolapse and degeneration, and neoplasm.

Treatment.—With the advent of antibiotics, the treatment of bone and joint tuberculosis has become similar in principle to the treatment of pyogenic infections. Early in the disease the pathological process can be arrested and cured by the use of antibiotics. Should treatment be deferred until pus has

formed, surgery will be required to evacuate the tuberculous abscess in exactly the same way as surgery is required for the evacuation of abscesses in pyogenic arthritis and osteomyelitis.

If abscesses form and require evacuation, the joint never regains its normal function: it is stiff, movement is painful, and weight-bearing in the spine and the legs is painful or impossible. Accordingly further surgical treatment is required in order to make the joint permanently comfortable. Such treatment always takes the form of arthrodesis, so that the final result is a permanently stiff, but painless and stable joint. Occasionally the disease leaves a joint which is replaced by short firm fibrous tissue (a 'sound fibrous ankylosis'). Tissue of this kind may be sufficiently stable to allow the joint to function as if it were arthrodesed, especially in the upper limb, so that no further surgery may be necessary.

Antibiotics.—A minimum of two antibiotics are used for the treatment of tuberculosis: streptomycin, para-amino-salicylic acid (PAS), isoniazid (INAH), ethambutol and rifampicin are all effective. Treatment with two antibiotics is begun immediately the diagnosis is made. If the diagnosis depends upon synovial biopsy, antibiotics are started immediately postoperatively, without awaiting the histological result. The drugs are given in the following (adult) doses: streptomycin 1 g daily, PAS 12–15 g daily, INAH 300 mg. daily, ethambutol 15 mg/kg body-weight, rifampicin 600 mg daily for 3 months and thereafter 450 mg daily (p. 37). Treatment is continued for a minimum of 18 months (but streptomycin should not be used for more than 3 months in patients under 50, and not at all in patients over 50).

Immobilisation.—The diseased joint is immobilised in the position of function (see Table, p. 224) until local symptoms have settled and the ESR has returned to normal. In the case of the spine, immobilisation requires the use of a plaster bed. Recently, especially in Africa where hospitalisation may be difficult, tuberculosis of the lumbar spine has been treated by antibiotics alone, the patient remaining ambulant throughout the period of treatment.

General Management.—There is no need for special diet nor for special atmospheric conditions such as sunlight. Sanatoria are unnecessary. The general health of the patient should of course be improved as far as possible by providing an adequate normal diet and by giving an iron supplement or even blood transfusion if there is significant anæmia. Should there be coincidental pulmonary tuberculosis with tubercle bacilli in the sputum, it will of course be necessary to isolate the patient until this aspect of the disease is brought under control.

Surgery.—Surgery is not required at the synovial stage of the disease since the disease can then be arrested by antibiotics alone. If, however, the synovial membrane is very markedly inflamed and thickened, synovectomy and joint toilet may be helpful.

If abscess formation has occurred, the abscess is incised and evacuated 3 or 4 weeks after the commencement of antibiotic treatment. When the

abscess has been evacuated, the originally articulating bones terminate in the abscess cavity. Some form of arthrodesis will then be required in order to provide the patient with a stable limb. Before antibiotics became available, arthrodesis of a tuberculous joint was carried out avoiding, if possible, the infected tissue (fig. 240). Such operations ('extra-articular arthrodeses') were technically difficult. Since the advent of antibiotics, the bone ends can be brought into apposition across the evacuated abscess cavity and sound bony fusion can commonly be obtained in this way.

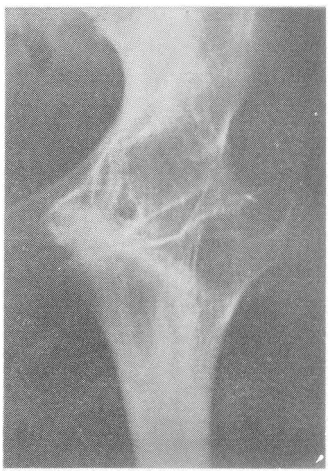


FIG. 240. — Brittain's ischio-femoral, extra-articular arthrodesis of the hip (see text).

In the spine, exactly the same surgical principles apply. The disease, however, is rarely diagnosed before bone involvement has taken place so that some form of arthrodesis is nearly always required. If no significant pus formation has occurred when antibiotics are given, it is usually possible to sterilise the lesion in the vertebral bodies with little or no vertebral collapse. The lesion itself can then be ignored surgically and the spine stabilised by a posterior fusion. Should pus formation have occurred, the abscess requires evacuation through an antero-lateral approach. At the same time as the abscess is evacuated bone grafts are packed between the involved vertebral bodies so that healing results in an anterior body-to-body fusion.

Prognosis.—The prognosis for this disease is now excellent. Death either from the tuberculous process itself or from secondary amyloidosis is now rare. Disability of any kind can often be prevented by early, adequate antibiotic treatment combined with appropriate immobilisation. At the worst the patient may spend 3 to 6 months in hospital and although finally cured of the tuberculous process, have a permanently stiffened joint.

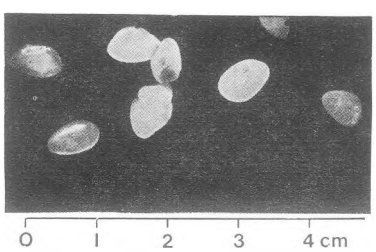


FIG. 241.—'Melon-seed' bodies from tuberculosis synovitis at the wrist.

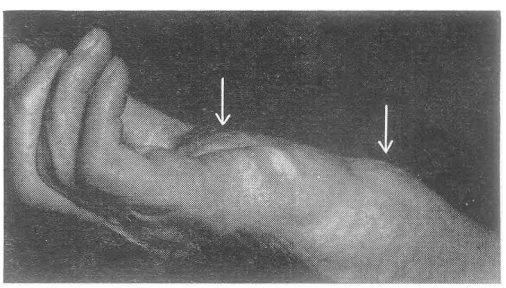


FIG. 242.—A compound palmar ganglion (tuberculous flexor synovitis at the wrist). The swelling in the palm communicates with the swelling above the wrist.

H. A. Brittain, 1904–1954. Orthopædic Surgeon, Norfolk and Norwich Hospital, England.

Tuberculous Tenosynovitis.—This may take two forms:

(*a*) The endothelial lining of the sheath is replaced by œdematous granulation tissue containing miliary tubercles. Very little free fluid is present. A soft elastic swelling appears and if the disease progresses, pus may form and track into neighbouring sheaths or joints.

(*b*) An effusion occurs in the tendon sheaths and 'melon-seed' bodies are usually present in large numbers, so that a soft, coarse crepitus is detected on pressing fluid from one part of the sheath to another. Melon-seed bodies resemble grains of boiled sago. They are composed of collections of fibrin, cellular debris and occasional tubercle bacilli (fig. 241). The term 'compound palmar ganglion' is applied to this condition when it occurs in connection with the flexor tendons of the fingers. A soft, painless swelling appears (fig. 242), and fluctuation may be transmitted above and below the anterior carpal ligament. As with all forms of tuberculous disease of bone, joint or tendon, obvious wasting of adjacent muscles is present. Treatment consists of general measures, the use of antibiotics and the application of an appropriate plaster cast to immobilise the involved tendon sheath. If the condition progresses, careful dissection and removal of the tendon sheath is indicated.

CHAPTER 15

THE RHEUMATIC DISEASES

OSTEOARTHROSIS (OA)[1]

Ætiology.—Normal synovial joints may be regarded as being in a state of equilibrium between the mechanical properties of the articular cartilage and the mechanical stresses imposed upon this tissue. If the loads applied to the joint rise or the same loads are applied through unusually small areas (i.e. the joint becomes abnormally incongruous), this equilibrium will be disturbed because the applied stresses rise. The equilibrium will also be disturbed if disease damages the cartilage so as to prejudice its mechanical properties. OA can perhaps be viewed as the outcome of any such dis-equilibrium and, as such, as the final common path travelled by any joint whose mechanics have been disturbed. Thus OA is perhaps best thought of, not as a specific disease entity, but rather as a constellation of clinical and anatomical features, analogous to heart failure: indeed it might with advantage be renamed ' joint failure '.

There is some evidence that OA is not initiated by abrasive wear caused by a breakdown in lubrication. Thus, if OA be loosely regarded as ' wear ' in a joint, it is probably wear in the sense that linoleum ' wears ' when walked over by stiletto heels, not wear in the sense in which it occurs in a poorly lubricated bearing.

OA developing after some recognised predisposing event is known as Secondary OA. All such predisposing causes either damage cartilage or reduce the contact area in a joint. Thus injuries to articular cartilage, especially if they interrupt the normal contour of the subchondral bone to produce ' high points ' in the articular surface, render the joint liable to develop osteoarthrosic changes over the ensuing years. Diseases which make the joint surface irregular (such as Perthes' Disease (p. 292) or Slipped Femoral Epiphysis (p. 290) predispose to OA by reducing the contact area in the joint and hence raising the contact pressure on the cartilage so as to damage the tissue mechanically. Articular cartilage may also be damaged enzymatically, for example, in septic arthritis, tuberculous arthritis, or rheumatoid arthritis and all these conditions may cause Secondary OA. (Since the rheumatoid process (see Rheumatoid Arthritis) can ' burn itself out ' after it has irrevocably damaged the articular surfaces, the late disability

[1] The Rome Conference of the International League Against Rheumatism in 1961 proposed that this condition should be named alternatively *osteoarthritis* or *osteoarthrosis*. These alternatives were accepted by the American Rheumatology Association in 1963. The term *osteoarthrosis* is now in almost universal use amongst British rheumatologists and is to be preferred to *osteoarthritis* since the latter implies, misleadingly, that the condition is an inflammatory disease of joints.

in this disease is not always strictly attributable to the rheumatoid process but rather to secondary osteoarthrosic changes superimposed upon a burnt-out rheumatoid joint.) Recurrent hæmarthroses (as in hæmophilia) and chemical infiltration of cartilage, for example with pigment (in ochronosis) or calcium pyrophosphate (in chondrocalcinosis), also damage articular cartilage and may result in OA.

Most cases of OA at the hip in Caucasians are idiopathic and are sometimes (misleadingly) called ' Primary '. Although the ætiology is unknown, there is mounting evidence that (1) a number of anatomical variants, not easily detectable in life, may exist at the hip and produce high local contact pressures on the cartilage and (2) that the repeated application of these pressures may cause fatigue failure in the collagen network in cartilage thus initiating the destruction of the tissue. In Caucasians radiological evidence of the disease is most frequent at the knees but disability severe enough to require surgery is most common at the hip.

Pathology.—The first visible abnormality in primary OA affects articular cartilage, the surface of which becomes roughened (a change known as ' fibrillation '). This surface roughening signifies fragmentation of the collagen network in the tissue and is accompanied by a loss of matrix proteoglycan and by softening. Fibrillation extends progressively deeper into the tissue until bone is exposed, a condition described pathologically as ulceration.

Fragments of cartilage become detached from the subchondral bone as ulceration occurs. They may form loose bodies in the joint, but most eventually become attached to the synovial membrane. Here they set up a low-grade inflammatory response which spreads to involve the capsule. The resulting gradual fibrosis of the synovial membrane and capsule shortens the capsule and thus limits joint movement. Endochondral new bone formation occurs around the periphery of the joint surface (where the articular cartilage is usually preserved because it is not exposed to significant load carriage) and leads to typical excrescences around the joint known as ' osteophytes '. In the bone adjacent to the joint there is an increase in vascularity very early in the disease, indeed it is suggested by some authorities that the alteration in bone hæmodynamics represents the initial change in the disease process. Later, as the cartilage becomes destroyed, the subchondral bone and trabeculæ become thickened, a change known as ' sclerosis ', and areas of soft tissue, (' cysts ') often liquefied in the centre, form in the cancellous bone adjacent to the joint. The bone separating such cysts from the synovial cavity may collapse into the cyst leading to progressive deformation of the bony articular surfaces and the separation of loose fragments of bone.

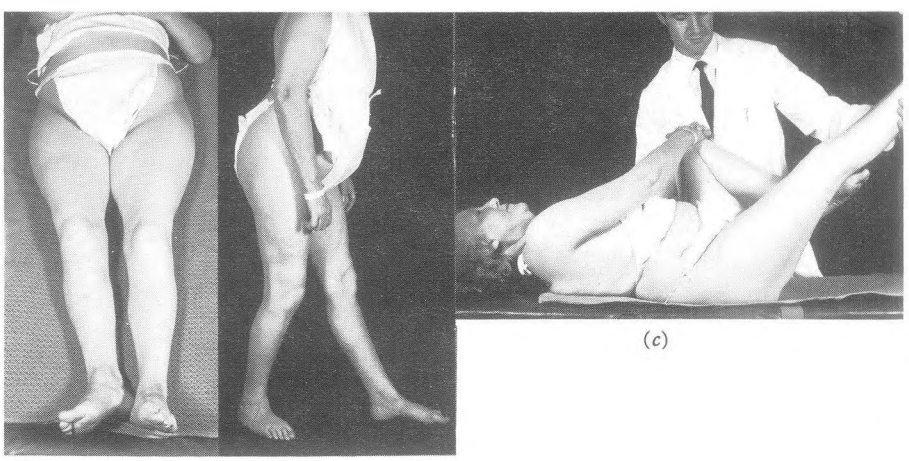
Clinical Features

The sequence of events by which the pathological process in OA causes the clinical features is discussed in relation to surgical treatment on p. 240.

Symptoms.—The initial complaint is always of pain felt on exercise and/or at night. The pain on exercise gets gradually more severe as the joint is used, a symptom particularly noticeable in the weight-bearing joints such as

the hip and knee. Pain is also felt when the joint is first moved after a period of immobility. Pain at night may be so troublesome as to be a predominant symptom: it is not uncommon for a patient with OA of the hip to be woken several times every night by pain. As the pain increases, the joint gradually loses movement because of muscle spasm, reluctance to move the joint, fibrosis of the capsule, and the bony changes described above. In the joints of the lower limb pain and stiffness combine to produce a limp. As the disease progresses the stiffness becomes increasingly severe until all movement at the joint is lost. By this time the joint usually lies in the position of 'ease' (Table p. 224) which may well be a position which is functionally useless, for example the hip typically becomes fixed in flexion, adduction and external rotation (fig. 243). This development of complete stiffness in a functionally inappropriate position constitutes deformity. Thus the patient, especially if more than one joint is involved, may be completely crippled and either bed-ridden or forced to use a wheelchair.

Physical Signs.—On inspection the abnormal position of the joint may be evident and a swelling attributable to a small synovial effusion may be



(*a*) (*b*) (*c*)

FIG. 243.—OA right hip. (*a*) Note external rotation, adduction and shortening. (*b*) Fixed flexion causing lumbar lordosis and protruding buttocks when the patient tries to stand erect. (*c*) Thomas's test to reveal fixed flexion (here 45°) at the right hip. The patient flexes her left hip fully and in so doing straightens her lumbar spine until it touches the couch. The right leg rises from the couch to reveal fixed flexion at the hip. The surgeon places his right hand under the spine to ensure that the lumbar lordosis is fully flattened and with his left hand supports the right leg to prevent pain but does not attempt to flex the hip.

visible in subcutaneous joints such as the knee. On palpation the joint may be mildly tender and fluctuation produced by an effusion may be detectable. When the patient is asked to move his joint, the loss of movement in certain directions will be evident and in the subcutaneous joints a sensation of grating can be appreciated if the hand is placed over the joint as it moves. This sensation is known as 'crepitus'. At the hip the tendency for the joint to become stiff in adduction and flexion leads to apparent shortening of the

Hugh Owen Thomas, 1834–1891. Surgeon, Liverpool, England. *See footnote p. 402.*

leg (fig. 243). True shortening may also occur as the femoral head and acetabular roof collapse.

Radiological Appearances.—These are characteristic (figs. 244 and 245). In the first instance they consist merely of narrowing of the joint space: a space which is of course occupied by radiolucent articular cartilage. As this cartilage thins and ulcerates the radiologically apparent joint space diminishes. Shortly thereafter bone changes become evident: sclerosis in the subchondral bone is the first abnormal feature to appear followed by cyst and osteophyte formation. Finally the bone collapses. The very marked new bone formation (manifest as sclerosis and the presence of osteophytes) distinguishes this disease radiologically from rheumatoid arthritis, in which the predominant radiological feature is one of bone loss.

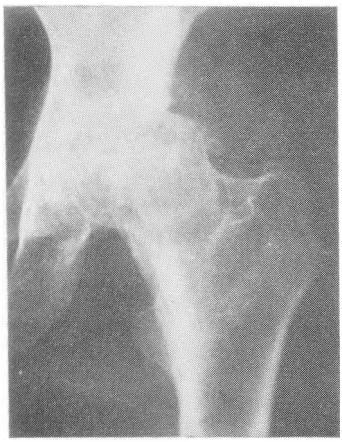


FIG. 244.—Osteoarthrosis of the hip.

Treatment.—There is no specific conservative nor pharmacological treatment for OA. In the early stages of the disease, the patient can usually be made comfortable by advice as to how to keep his joints mobile whilst loading them as little as possible, and by simple analgesics. The advice concerning the use of the joint may be supplemented by physiotherapy, by which the muscles acting across the involved joint are built up in strength and in which an attempt is made to regain a full range of active movement. In OA affecting the joints of the leg, weight loss, when possible, is desirable and the use of a walking stick and of a raise to the heel of the shoe (if the leg is short) may well make the patient more comfortable. Should these measures fail operative treatment has to be considered (p. 240).

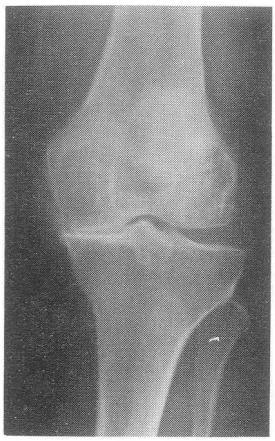


FIG. 245.—OA of the knee affecting the medial compartment to cause a varus deformity (genu varum). This is a weight-bearing X-Ray: films taken with the patient supine may grossly underestimate the deformity. (See also figs. 251, 252, 253.)

RHEUMATOID ARTHRITIS (RA)

In this text-book, the pathology, clinical features and conservative treatment of rheumatoid arthritis can only be touched on in outline. For a more detailed treatment of these aspects of the disease the reader is referred to a textbook of rheumatology. The disease is systemic in nature having its major, but by no means its only, manifestations in the joints.

Ætiology.—The ætiology of RA is unknown, but a number of theories

have been advanced of which probably the most widely credited today rests upon the belief that the disease is immune in nature.

Pathology.—In the joints, the tissue first affected in rheumatoid arthritis is the synovial membrane, which becomes inflamed and bulky. A striking histological feature in the synovial membrane is the presence of numerous lymphocytes and plasma cells which can be shown to be actively synthesizing antibodies. In addition an effusion accumulates so that the volume of material (synovial fluid and membrane) within the capsule of the joint becomes very considerably increased. As the synovial tissue enlarges, it gradually encroaches upon the articular cartilage where it is known as *pannus*[1]. Beneath the synovial pannus, the articular cartilage is destroyed and never subsequently regenerates. Intra-articular structures, such as the cruciate ligaments in the knee, may also be destroyed. The bone adjacent to the joint is eroded by the intrusion of exuberant synovial membrane. It also becomes osteoporotic, partly as a consequence of the hyperæmia associated with the disease, but sometimes partly as a consequence of its treatment with steroids. The combination of a loss of cartilage and osteoporosis may lead to rapidly progressive, severe bone collapse and hence to instability and deformity at the involved joint.

Clinical Features

Symptoms.—The disease may commence in childhood, when it is known as Still's disease, or at any time in adult life. RA developing in the elderly seems to run a more benign course than the disease developing in the young adult. Women are more commonly affected than men. The disease is typically poly-articular and often starts in the small joints of the hands or feet. Thereafter all the synovial joints of the body may become involved simultaneously or consecutively. Each joint as it is involved is painful, stiff and swollen. The activity of the disease tends to wax and wane both in the patient as a whole and locally in the involved joint, so that a joint may be painful, stiff and swollen for a period of 2 to 3 months and then gradually settle to be followed after an interval by a similar episode in another joint. The patient often feels unwell in himself and is mildly febrile.

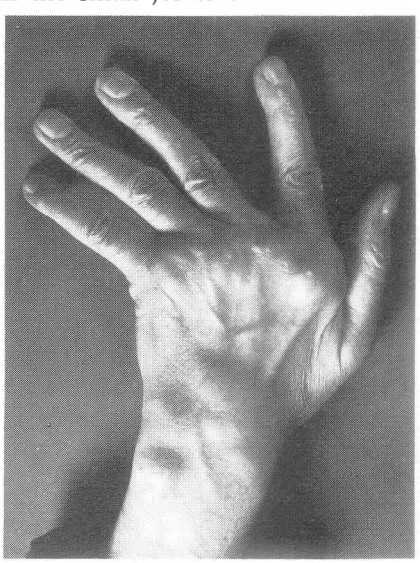


FIG. 246.—The hand in rheumatoid arthritis.

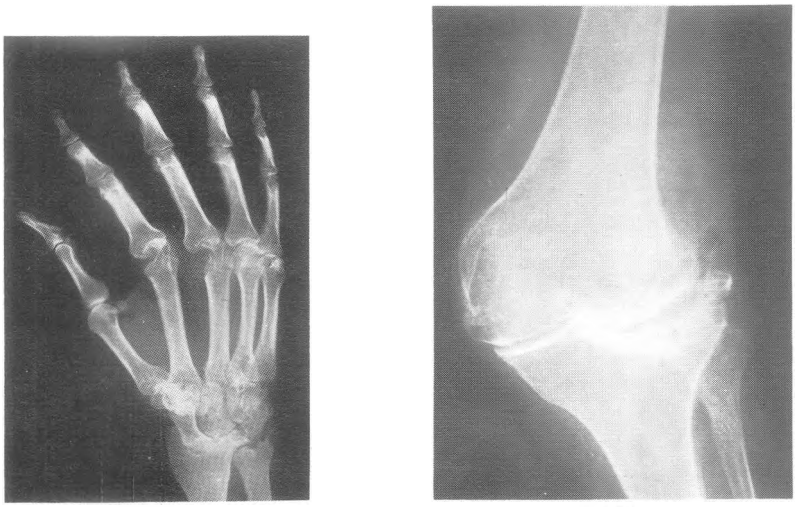
Physical Signs. — The involved joints, especially those of the hand, show marked synovial thickening and as the disease advances typical deformities develop (fig. 246). On pal-

[1] Pannus (Latin) = cloth.

Sir George Frederic Still, 1868–1941. Professor of Diseases of Children, King's College Hospital, London.

pation the synovial thickening, together with the presence of a synovial effusion, may be obvious. The joint can often be felt to be mechanically unstable and movement is painful and limited. As the disease progresses, limitation of movement, muscle wasting and deformity gradually develop at involved joints so as to cripple the patient.

Radiological Features.—The joint space is initially narrowed as in OA (fig. 247 (*a*)) but the intrusion of affected synovium into the bone produces



(*a*) (*b*)

FIG. 247.—(*a*) The hand in rheumatoid arthritis: note loss of joint space, erosion of the metacarpal heads and disorganisation of the wrist. (*b*) The knee in rheumatoid arthritis. Gross collapse of the lateral tibial and femoral condyles.

typical lytic lesions in the latter. In contrast to OA, osteophytes are small or absent and the bone adjacent to the joint becomes increasingly porotic so that it collapses under the compressive loads to which it is subject (fig. 247 (*b*)). As the disease advances, a combination of bone destruction and capsular stretching allows the joint to subluxate and finally to dislocate.

The active rheumatoid process may remit in a particular joint, leaving its articular cartilage destroyed. Thereafter the joint may progressively develop osteoarthrosic changes so that the final X-ray appearances are to some extent those of OA.

The sequence of events by which the pathological process in RA causes the clinical features is discussed in relation to surgical treatment on pp. 240 and 248.

Special Investigations.—The ESR is raised and an abnormal immune globulin known as the *rheumatoid factor* may be demonstrated in the blood by use of the Latex or Rose Waaler tests. The demonstration of rheumatoid factor is not, however, diagnostic of RA since it is found in healthy individuals and in those suffering from various chronic infections, neither is rheumatoid factor always present in RA. These abnormal hæmatological findings are in

Harry M. Rose, Contemporary. Professor of Medical and Surgical Research Chemistry, Department of Microbiology, Columbia-Presbyterian Medical Centre, New York.
Erik Waaler, Pathologist, Ullevaal Hospital, Oslo, Norway.

sharp contrast to OA in which the blood picture is entirely normal and in which there is no systemic disturbance of any kind.

Treatment.—The conservative treatment of RA consists of exercises and splintage to maintain joint function and to prevent deformity, combined with drug therapy. Surgery (p. 240) may be used to remove involved synovial tissue, to relieve the disability of a destroyed joint, or to treat isolated soft tissue lesions such as compression of the median nerve in the carpal tunnel (p. 310).

ANKYLOSING SPONDYLITIS

Pathology.—This is a crippling disease which, although it has some features in common with RA, forms a distinct clinical entity and is regarded as such in the U.K. On the other hand the initial histological appearances of this disease are so similar to those of RA that in the U.S.A. ankylosing spondylitis is regarded as a variant of RA.

FIG. 248.—Ankylosing spondylitis.

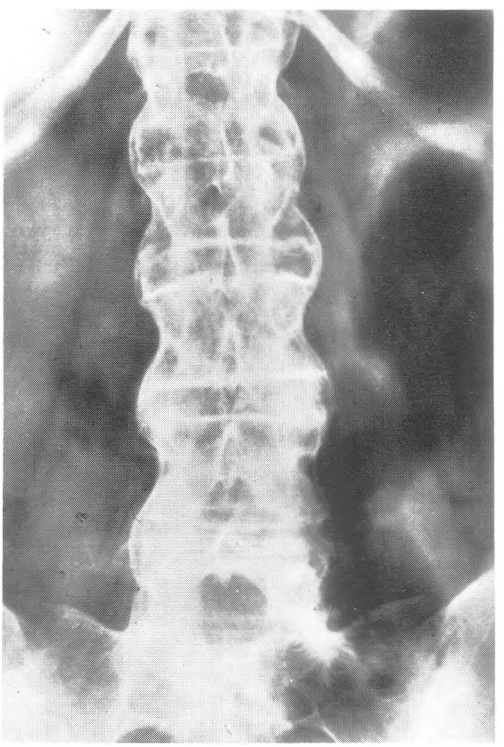


FIG. 249.—Ossification of the lumbar spine ('bamboo' spine).

The disease is essentially a process of calcification and then ossification of the ligaments and capsules of joints, which results in complete bony ankylosis of the central articulations of the body. (This contrasts with rheumatoid arthritis which is not predominantly an ankylosing disease and which affects the small, distal joints of the extremities first). Whereas RA affects mainly females, ankylosing spondylitis is more common in men, starting in early adult life.

It has recently been shown that the tissue antigen HLA-B27 occurs in 96% of patients with ankylosing spondylitis, while present in only about 6% of general Caucasian populations. Tissue typing may thus help in diagnosing the condition, but the mechanism by which this genetic factor operates in predisposing to the disease is not yet established.

The joints most affected, in order of frequency, are the sacro-iliac joints, the joints of the spine (ascending from the lumbar level), the hips, the costal joints, and the shoulders. In addition to developing bony ankylosis, the spine becomes flexed so that eventually the patient is bent double and can no longer see in front of herself (fig. 248).

Special Investigations.—The diagnosis is confirmed by the finding of a raised ESR (an investigation which should never be omitted in the examination of young adults complaining of low back pain), by the radiological demonstration of bony obliteration of the sacro-iliac joints, and finally by the presence of ossification in the spinal ligaments ('bamboo spine') and ankylosis of other involved joints (figs. 249 and 250).

Treatment.—There is no specific treatment for this condition. Fortunately the disease sometimes arrests spontaneously before the full picture of spinal ankylosis in gross deformity is reached. Patients in whom the thoracic cage

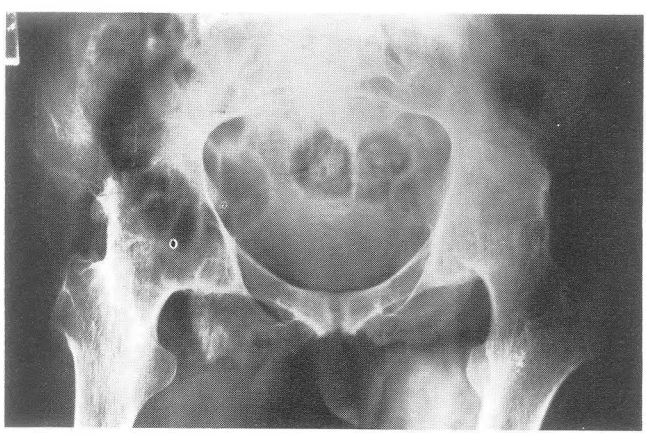


FIG. 250.—Ankylosing spondylitis: ankylosis of the hips.

is ankylosed and the spine is flexed, may die of pulmonary infection because of the reduction in their vital capacity. Simple analgesics help to relieve pain and postural exercises may help prevent increasing deformity. Osteotomy of the spine (p. 253) is occasionally indicated to correct severe deformity. Pain and stiffness in the hips may be relieved by arthroplasty (p. 253).

OPERATIVE SURGERY IN THE RHEUMATIC DISEASES

In this section, the general principles of the surgery of the rheumatic diseases are outlined in relation to the probable causation of the disability.

The application of these principles will then be discussed in relation to the joints involved in RA, ankylosing spondylitis, and OA.

Operative surgical treatment in the rheumatic diseases is never curative: rarely, the pathological process may be arrested or even reversed, more commonly its existence and progression have to be accepted and surgery offered to provide palliation of the patient's disability—pain, stiffness and deformity. The exact causation of this disability is sometimes obscure, but an attempt must be made to explain it in the rationale if operative surgery is to be understood.

The Ætiology of Pain, Stiffness and Deformity

Pain in the rheumatic diseases appears to arise in three tissues: the bones of the joint, its capsule and the muscles acting on it. Cartilage is not innervated and accordingly is insensitive to pain. Synovial membrane is poorly innervated and experiments with human volunteers have shown that it is almost insensitive to pain arising from mechanical stimulation. No doubt active inflammation of the synovial membrane, as of the capsule, is painful, but active inflammation of these tissues in the rheumatic diseases is not treated surgically.

Although histologically nerve fibres which might be pain afferents have been described in bone, the effective stimulus for the provocation of pain in bone is unknown. Experimentally, pain may be induced when the intra-osseous venous pressure is raised and venous engorgement of juxta-articular bone has been demonstrated in OA. (Traction on the periosteum is painful but this is not relevant to pain induction in intra-articular bone where the true periosteum is replaced by cartilage or synovium.) Whatever may be the precise nature of the effective stimulus for bone pain, it may be regularly observed in clinical practice that if the arthritic bony surfaces are replaced with appropriate prostheses securely fixed to the skeleton, the patient's arthritic pain both at rest and on weight-bearing is abolished at once. This observation proves that the major component of the pain of arthritis originates in bone and it may be speculated that this is due to the generation of abnormally high stresses in the bone, possibly leading to trabecular micro-fractures and hence to vascular engorgement.

The fibrous capsule and ligaments of all joints are well innervated both with mechanoreceptor afferents subserving perceptual and reflex proprioception and with pain afferents. The effective mechanical stimulus for the provocation of capsular pain appears to be excessive tension in the capsule. In the context of the rheumatic diseases excessive capsular tension may be brought about by two pathological events: firstly, an increase in the volume of material within the capsule, that is to say by an increase in the bulk of the synovial membrane or by an accumulation of intra-articular fluid, and secondly, by capsular fibrosis leading to shortening and tethering of the capsule. Joint movement and position affect the severity of this pain in two ways. In all joints some position within the normal range draws the capsule most tightly against the synovial contents and it is this position which provokes

pain when the bulk of the intracapsular tissues is increased. Secondly, the pain arising from capsular fibrosis is provoked only or chiefly when the capsule has shortened to such an extent that it limits movement: when this limited movement is attempted by the patient with sufficient force to apply tension to the capsule pain is provoked. Thus capsular pain interferes with movement and in an attempt to avoid it the patient tends to hold the joint immobile in a position of capsular relaxation.

Muscular pain is probably provoked by local anoxia or by the accumulation of metabolic end-products, arising from abnormally sustained muscular contractions. Such contractions—known clinically as muscle spasm—have not been convincingly demonstrated electromyographically but their existence may be inferred from the fact that the passive range of movement in a diseased joint is often increased under a general anæsthetic producing muscle relaxation. Muscle spasm of this type appears to be a reflex consequence of joint disease and imposes immobility on the affected joint.

Joint stiffness is a loose term covering two disturbances of function—a sense of added resistance encountered by the patient when he attempts to move the affected joint, and a total inability to move the joint through some part of its normal range. The first factor is perhaps in part due to a rise in intra-articular friction caused by disruption of the normal cartilaginous bearing surface, but seems mainly attributable to articular and extra-articular soft tissue thickening, adhesions and œdema, and probably to over-contraction (' spasm ') of the antagonist muscles.

A reduction in the passive range of movement may be due to pain and muscle spasm—in which case the range will return under anæsthesia—or to such mechanical factors as capsular and peri-capsular fibrosis, osteophyte formation and collapse of the articulating bony surfaces.

Deformity.—In its early stages deformity may be regarded simply as a loss of part of the normal range of movement: thus, for example, a patient who is unable fully to extend his knee may be said to have a flexion deformity or contracture of that joint. Deformity may progress to a point at which the two articulating bones start to take up a position in relation to each other which does not occur anywhere in the normal range of movement: in other words the joint becomes subluxated and finally dislocated.

Dislocation implies a mechanical derangement of the tissues of the joint and the presence of a deforming force. The factor chiefly responsible for dislocation in the rheumatic diseases is the collapse of the bony articular surfaces. In the leg the ligaments stretch little, if at all, but at the hand they are said to do so sufficiently to permit dislocation. The deforming forces at work consist of the body weight and the play across the joint of muscles of normally unequal power (such for example as the finger flexors and extensors), muscles in ' spasm ', or muscles suffering progressive fibrosis. In the leg, the position of a joint may be normal until weight-bearing is attempted. The deforming force of the body weight may then be sufficient to subluxate the diseased joint. Subluxation or dislocation of this kind affects particularly the knee and is referred to as *instability*.

The General Principles of Surgery in the Rheumatic Diseases

The surgical procedures used in the rheumatic diseases must be related to the underlying pathology if they are to be understood rationally. The following represents the author's view of the relevant surgical pathology.

OSTEOARTHROSIS (OA)

The fundamental cause of idiopathic OA is unclear but in general it appears to be due to mechanical or mechano-chemical degradation of cartilage. Sometimes an obvious mechanical cause for such degradation can be demonstrated—as for example joint incongruity after trauma, a reduction of the contact area in congenital dysplasia of the hip, or shifts in the distribution of force in a joint as for example at the knee in tibia vara. In so-called idiopathetic OA, the nature of the presumed mechanical abnormality is unclear: perhaps in such joints the collagen network in the cartilage fatigues in the face of normal loads or some clinically undetectable anatomical abnormality is present. All the obvious abnormalities which predispose to OA have in common the fact that they seem certain to increase the contact stress on the articular cartilage somewhere in the joint. Presumably as a consequence, the cartilage is destroyed and bone is exposed. Because cartilage is not innervated, it is usually not until the point of bone exposure that the patient experiences pain and hence seeks treatment. By this time surgical measures aimed at reversing the basic abnormality may not always be satisfactory—even if they are possible—since adult human articular chondrocytes can neither undergo mitosis nor, for all practical purposes, can they synthesize the collagen network in their matrix. Thus exposed bone is likely to persist in the reconstructed joint causing continuing pain and abrasive damage to the opposing joint surfaces. In spite of this, joint reconstruction may bring useful amelioration and since no other form of treatment be it physical treatment (such as exercise) or drug therapy can have any fundamental, beneficial effect whatsoever, such surgical procedures are often felt to be worthwhile. The operations specific for certain disorders which are known to be the precursors of OA (such as dysplasia of the hip) are outside the scope of this chapter. The operation of osteotomy is however of general relevance in OA, is used in this context, and will therefore be considered.

In the operation of osteotomy the bone adjacent to a joint is divided to allow the fragments to be displaced relative to each other in such a way as to bring residual cartilage into the area of the joint transmitting the major loads. For example: in OA of the tibio-femoral joint the cartilagenous and later, the bony defect initially affects one compartment (medial or lateral) only. Such defects produce an angular deformity in the weight-bearing knee concave towards the side of the defect. Thus for example a defect in the medial tibial condyle will produce a varus deformity. Any such deformity will displace the line of action of the resultant of the forces acting through the knee towards the concavity, that is to say towards the defect (fig. 251). Thus a vicious circle is established in which the defect results in a deformity which increases the loads acting on the defect to cause increasing deformity and

pain. One way in which this vicious regress could be interrupted is to angulate the tibia, by osteotomy, so as to return the overall alignment of the leg to normal (or even a little towards over-correction), thus off-loading the

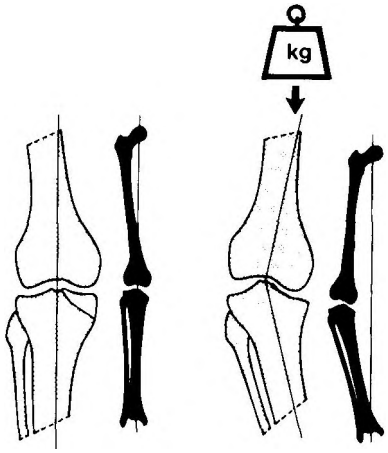


FIG. 251.—The normal non-weight-bearing knee is shown on the left. Note that the centre of the hip, knee and ankle lie in a straight line. If a bone defect (unshaded) were to be created in the medial tibial condyle, the femoral condyle would collapse into the defect on weight-bearing to produce varus malalignment as shown on the right.

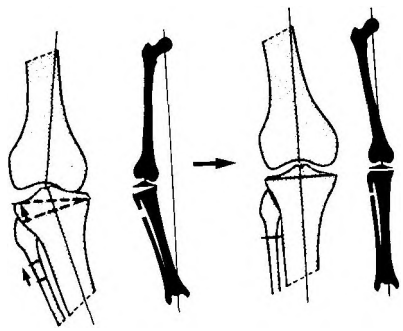


FIG. 252.—On the left the varus malalignment shown in fig. 251 is present. The bone resected from the proximal tibia and from the shaft of the fibula by a closing wedge osteotomy, is marked with dotted lines. When this bone is removed and the osteotomy surfaces are approximated, the leg is restored to its normal alignment as shown on the right.

defect and bringing the relatively healthy cartilage on the other side of the joint back into the line of action of the load (fig. 252).

In early OA of the hip, cartilage is often lost from the superior surface of the head and preserved elsewhere. Intertrochanteric osteotomy followed by angulation of the shaft of the femur may now rotate the femoral head sufficiently to allow intact cartilage to become weight-bearing.

In addition to an action of this mechanical kind, osteotomy has been thought to have a persistent analgesic effect associated with the healing of cysts in bone and of hyaline articular cartilage (with fibro-cartilage). This effect has been attributed to the division of the bone itself (perhaps mediated by an alteration in the innervation or the hæmodynamics of the involved articular bone rather than to the angulation as such) and has led to the suggestion that better symptomatic results could be achieved by dividing both bones, rather than just one. The existence of this 'biological' effect remains however controversial and most surgeons using osteotomy now plan their procedures on a mechanical basis. Even when properly planned and executed the symptomatic relief obtained by osteotomy is unfortunately unpredictable.

Osteotomy apart, nothing can be offered surgically which might, even theoretically, restore the tissues of the joint to their original, normal state.

Thus if it is felt that a joint has become too destroyed for osteotomy to be useful, the joint must be stiffened (arthrodesed) or replaced.

In arthrodesis the bone ends are resected and then held in contact in the hope that they will fuse together. If they do, pain is relieved and deformity corrected, but at the price of loss of movement. This price may be acceptable, as for example in the joints of the foot, or very undesirable as for example in the hip and knee. In the former joints, arthrodesis therefore offers the possibility of permanent pain relief at an acceptable price and for this reason it is frequently employed for, for example, osteoarthrosis of the subtalar joint or ankle. Unfortunately OA of these joints is relatively rare whereas it is very common in precisely those lower limb joints where movement is vital (the hip and knee). Thus arthrodesis has only a small part to play as a primary procedure in the surgery of OA.

A major surgical problem therefore exists in OA of the hip and knee: the disease is common, cannot satisfactorily be treated by arthrodesis and frequently presents too late to be treated by osteotomy (which is anyway an unreliable procedure). No similar problem exists in the upper limb since here OA is rare and does not usually cause severe pain. It is therefore particularly for the hip and knee that an alternative is needed. This alternative is replacement surgery.

The rationale of joint replacement is simple: the defective bone and cartilage is removed and replaced by a prosthesis in much the same way as an area of caries is removed from a tooth and replaced by a filling. If the height of the defect is restored precisely, deformity and instability will be corrected (fig. 253) whilst movement is permitted and pain is relieved by covering the opposing, exposed bone surfaces with prostheses (of appropriate shapes and made of inert materials) attached to the skeleton in such a way that they do not move against the bone. The materials in use today are high density polyethylene for the concave component of the joint and one of the implantable metals for the convex surface, both bonded to the skeleton by a cold curing acrylic (polymethylmethacrylate). The technicalities of these procedures are outside the scope of this text.

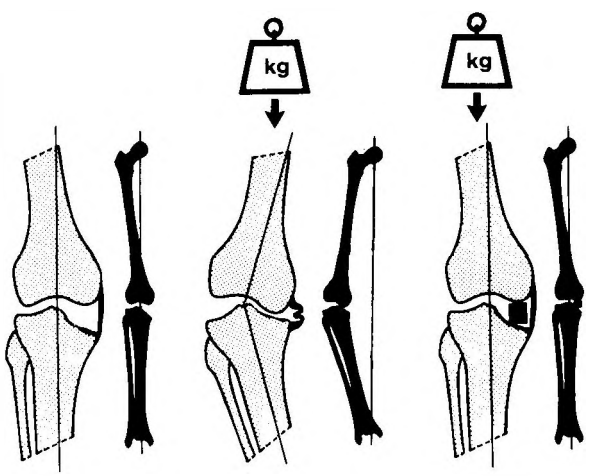


FIG. 253.—The left hand diagram shows a knee with a medial tibial defect but without a soft tissue contracture. When load is applied to this knee, the joint collapses into varus as shown in the middle diagram and in fig. 251. The knee may be restored to normal alignment by blocking open the defect with a prosthesis, shown diagramatically, on the right.

The immediate outcome of a successful replacement is easy to describe: pain is abolished and the joint is rendered mobile and stable. Technically however these procedures are not easy to perform and if a component becomes loose or infected, the pre-operative disability may return in full and may then be difficult or impossible to relieve. Since the chances of loosening or infection are related in part to the length of time the implant is required to function, joint replacement procedures should be used with great caution, if at all, in the young. For patients over 60 however they represent the treatment of choice for OA of the hip and knee.

RHEUMATOID ARTHRITIS (RA)

It might be thought that the likelihood of surgical procedures being useful in RA would be slight: the disease is after all said to be due to a chronic, progressive synovitis which unless arrested would be expected to destroy a joint however it might be reconstructed surgically.

Based on this view of the pathology, synovectomy was widely used in joints with an accessible synovium such as the knee and the joints of the hand. The results were disappointing: synovitis recurred in the regenerated synovium and the long-term fate of the joint was not influenced for the better. Nevertheless the procedure may still have a part to play in the hand with severe synovitis, resistant to rest and medication, involving the tendon sheaths and joints.

The failure of synovectomy of the knee to produce long-term remission may be contrasted—at first sight surprisingly—with the outcome of replacing the articular surfaces of the same joint. After the latter operation, which is unaccompanied by synovectomy, it has been the author's experience that any pre-operative synovitis remits and does not recur unless the joint replacement fails for mechanical reasons such as loosening of the prosthesis. This observation, combined with the failure of synovectomy, invites the question: is synovial inflammation really the fundamental element in the pathology of moderately advanced RA? In this author's view the answer is: No.

There can be little doubt that, in RA, the initial event in a given joint is the onset of a synovitis the ætiology of which is unknown. The immune response appears to be associated with this synovitis in most cases of RA—the evidence for the association being strongest in sero-positive RA—but whether it is in any way causative (and thus worth suppressing) is not clear. There is, after all, irrefutable evidence that the immune response is involved in tuberculous arthritis but this disease would not be arrested—indeed it would be made worse—if the immunological events were suppressed.

Regardless of the role of the immune response, it is clear that an invariable element of the pathology in rheumatoid synovitis is the exposure of articular cartilage to degradative enzymes, in particular collagenase, derived mainly from leucocytes. Experimentally collagenase destroys the collagen network in cartilage and with it, the mechanical properties of the tissue. Indeed it is probably true to say that the prognosis for a joint following an episode of synovitis depends upon whether collagenase is, or is not, present; if it is

present, as in septic arthritis, the prognosis is bad; if it is absent, as is probably the case in Rheumatic Fever, the prognosis is good. The mechanical forces of everyday life, acting on cartilage enzymatically weakened, now destroy the cartilage surface, not just in local areas where the loads are particularly heavy as in OA, but generally. Bone is now exposed in the joint surface so that the pain of the original synovitis blends into the pain of bone-to-bone contact. Bone being a poor bearing material, it is now destroyed by abrasion and crushing to produce progressive instability and deformity.

From the onset of these events, detritus—at first fragments of cartilage and thereafter of bone—is continually liberated into the joint cavity, an event which in other contexts is known to cause a chronic synovitis. Could it therefore be that the synovitis in RA is due to an initial inflammatory process of unknown ætiology but relatively short duration, followed by a chronic synovitis due to the presence of detritus in the joint? On this analysis the progressive destruction of the joint, i.e. the 'progression' of Rheumatoid Arthritis, would be due to mechanical factors and would be the cause of the continuing, chronic synovitis and an (idiopathic) synovitis would be thought to initiate the disease but not to be responsible for its progression. This concept would explain the two surgical observations mentioned above—that synovectomy does not arrest the progress of the disease locally and that resurfacing a joint abolishes the synovitis. In so far as it suggests that there is no mysterious progressive pathology at work, it would encourage the view that a resurfaced rheumatoid joint might stand the test of time. On this view it might be thought that because the progress of RA, as of OA, is mainly due to mechanical factors, it is unlikely to be influenced by physiotherapy nor by medication. Thus not only would surgery offer hope, it might offer the only hope.

Because the destruction of articular cartilage in RA affects the whole of the joint surface, little benefit can be expected (nor is it obtained) by attempts to alter the areas of the articular surface subjected to load. Thus osteotomy has little or no place in RA.

Arthrodesis should be avoided if at all possible in RA since to stiffen a joint imposes additional stresses on its neighbours, and in this disease these joints are likely to be abnormal.

Thus the surgery of RA is a matter of joint replacement with limited exceptions in the small joints of the feet, the hand, wrist and spine where synovectomy (perhaps in the hand), arthrodesis and excision arthroplasty have a part to play.

It might be thought that, however attractive in theory, it would be impracticable to treat established RA surgically since in principle every joint in the body is involved. Closer inspection makes the situation seem more hopeful. The disability in the upper limb although significant, is frequently tolerable since pain is not usually severe whilst the presence of other mobile joints and the astonishing versatility of the hand make it possible to offset significant handicap in one joint or even one limb: Nelson won Trafalgar with one arm. It is in the lower limb that pain may be intolerable and because the patient cannot walk he or she becomes unemployable, unable to care for herself and

socially isolated. Fortunately one joint—the knee—is responsible for the majority of severe disability. Involvement of the hip is relatively uncommon but when it occurs (in about 3% of patients) it is crippling. The foot is almost always involved but although this involvement is painful it is often not crippling. Thus replacement of the knees—now in some centres a routine and reliable procedure—may abolish, perhaps indefinitely, the major disability in RA. Hence surgery, especially replacement surgery of the knee, has a major part to play in the treatment of RA.

THE SURGERY OF OSTEOARTHROSIS

Primary OA affects certain joints, such as the hip, knee and carpo-metacarpal joint of the thumb, much more commonly than it affects others such as the shoulder and wrist. Secondary OA following injury or another primary disease process may affect any joint.

The Upper Limb

The Hand.—The only joint in the hand commonly requiring surgical treatment for primary OA is the carpo-metacarpal joint of the thumb. Here the disease may be treated by *arthrodesis* of the diseased joint or by *excision arthroplasty* of the trapezium. At their best these operations give indistinguishable results with an excellent return of painless function. Heberden's nodes occasionally cause pain at the TIP joints.

The Wrist.—Secondary OA following injury is treated, if the pain is sufficiently severe, by arthrodesis.

The Radio-Ulnar Joints.—The distal radio-ulnar joint may be affected by secondary OA following fractures adjacent to the joint, such as Colles' fracture. The proximal radio-ulnar joint is similarly affected following fractures of the head of the radius. In both cases the treatment of choice is *excision arthroplasty:* in the distal joint by excision of 2·5–5 cm. of the distal ulna, in the proximal joint by excision of the head of the radius.

The Elbow Joint.—Primary OA may affect all components of the joint. Should the joint be so painful as to require surgery, which is rare, the most commonly employed procedure is *arthrodesis* of the humero-ulnar joint in sufficient flexion to allow the patient to feed himself and wash, together with excision of the head of radius.

The Shoulder.—The shoulder is rarely affected by OA, primary or secondary, but if so, it may be treated by *arthrodesis.* Although fusion is not easy to secure, it is easier than in RA because the bone is hypertrophic rather than porotic. The functional results of a sound arthrodesis in the position of function (i.e., approximately the position taken up by the shoulder when the hand is brought to the mouth) are good.

The Lower Limb

The Toes.—OA and other degenerative changes affecting the toes are considered in Chapter 22.

The Foot and Ankle.—OA, primary or secondary, occasionally affects any of the tarsal (especially the subtalar) joints or the ankle. If surgical treatment is necessary, arthrodesis is the only procedure available at joints other than the ankle. The latter however can now be replaced.

The Knee

Chondromalacia Patellæ.—A condition which has been thought to be analogous to primary OA affects the patello-femoral joint of young adults (especially women). In this disease the articular cartilage covering the patella and subsequently that covering the patellar articular surface of the femur becomes softened, fibrillated and finally ulcerated. Although this

Abraham Colles, 1773–1843. *(See footnote, p. 359.)*
William Heberden, 1710–1801. Physician, London, England.

sequence of events is identical with that seen in primary OA elsewhere, in this joint it occurs in early life as a normal event so that its pathological significance is hard to ascertain. The condition has been treated by various surgical procedures aimed directly at the cartilage: the cartilage and subchondral bone have been *drilled* and the fibrillated cartilage *shaved.* It is doubtful if these procedures are beneficial, although the regeneration of fibrocartilage following drilling of the patella in advanced OA of the knee has been described. Recently the view has gained ground that the condition is due to an increase in the contact pressure between the patella and the lateral femoral condyle caused by a tendency for the patella to be drawn laterally in involved knees. Treatment based on this view takes the form of division of the lateral patellar retinaculum and occasionally of transposition of the tibial tubercle. Useful pain relief has been reported after these operations. As a last resort the patella may be excised.

Chondromalacia Patellæ apart, the knee, after the hip, is the joint most commonly affected by OA.

Arthrodesis should not now be carried out as a primary procedure. It is used as a last resort if one of the modern forms of total joint replacements fail and cannot be revised. Several methods of arthrodesis have been described but the Charnley arthrodesis is perhaps the most reliable and widely used.

Excision arthroplasty of the patella has a part to play in OA confined to the patello—femoral joints, e.g. after fractures of the patella.

Total replacement arthroplasty is now giving very encouraging results. At the time of writing its place in OA cannot be defined but it is being used on a steadily increasing scale and may well displace all other surgical procedures in patients over 60 years of age in the next 5 years. A variety of prostheses is available (p. 253).

Osteotomy is indicated in patients under the age of 60 with early osteoarthrosis causing disabling pain with neither significant loss of flexion nor fixed flexion deformity. The disease at this stage is unicompartmental, causing a varus deformity if it affects the medial side of the joint (fig. 245) and a valgus deformity if it affects the lateral side. If the defect in the articular surface affects the tibia, the bone is divided above the tubercle and angulated so as to correct the deformity. Defects in the femoral condyle are treated by supracondylar femoral osteotomy. By realigning the knee in this way the force acting on the diseased compartment is reduced and this in turn eases pain and retards the progression of the disease.

The Hip

OA of the hip requires surgical treatment more commonly than does any other rheumatic disease in any other joint.

Arthrodesis, excision arthroplasty, interposition arthroplasty and partial replacement arthroplasty have all been used in the past but have been abandoned in favour of total replacement arthroplasty.

Total replacement arthroplasty using a prosthesis replacing the acetabulum as well as the head of the femur was introduced by McKee and Charnley (both in England) in the late 1950s. The device now most widely used in the UK is the Charnley prosthesis in which the acetabulum is constructed of high density polyethylene, and the head of the femur of stainless steel. Both components are attached to the bone with a cold-curing acrylic 'cement'. It is still too early to give a definitive assessment of this operation since it has been progressively introduced over the past 15 years and has only in the last few years been universally available. The results to date, however, have been extremely encouraging: pain is relieved, a useful range of motion is regained, and the hip is stable. Post-operatively, the convalescence is short, weight-bearing may be allowed within days of operation, and the prolonged, tedious and sometimes painful physiotherapy which some other procedures require is unnecessary. If these results are maintained over the next 10 years the operation will be indicated for most cases of OA of the hip: at the present time, because of uncertainty surrounding the long-term

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Sir John Charnley, Contemporary. Formerly Professor of Orthopædic Surgery, University of Manchester, England.

results, the operation is avoided if possible in patients under 60 years of age unless the prognosis for life is shortened by intercurrent disease. In patients under the age of 60, a new form of total replacement in which both the femur and acetabulum are resurfaced is now under evaluation.

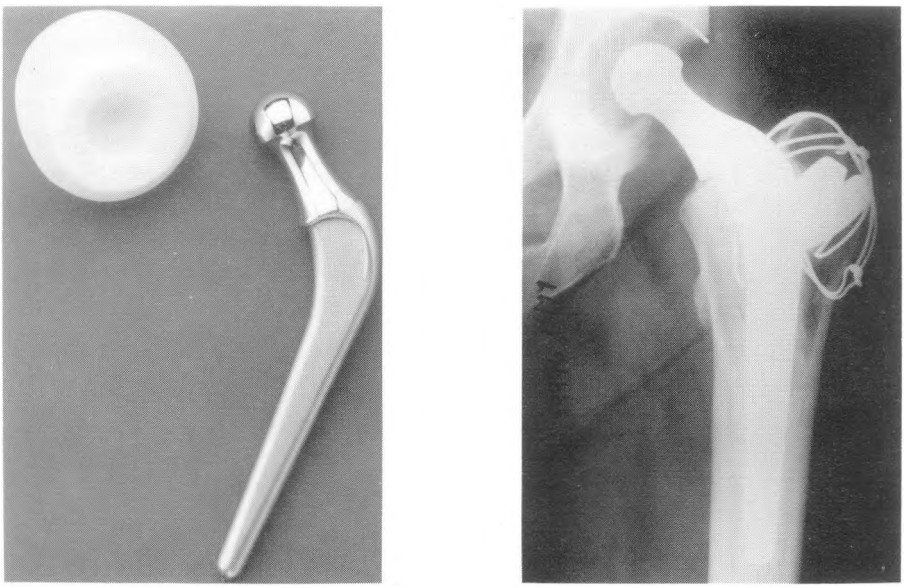

FIG. 254.—Total replacement of the hip using the Charnley prosthesis.

Osteotomy is employed—at the intertrochanteric level using internal fixation of the osteotomy site (fig. 255)—when pain, especially night pain, demands surgical relief but the range of movement is still good: the presence of 70° of active flexion being taken as a rough dividing line between a hip suitable for osteotomy and one suitable for arthroplasty. Since total replacement arthroplasty is avoided in patients under 60 years of age, osteotomy is employed particularly in this age group: over the age of 60 it has been generally replaced by total replacement arthroplasty. In some centres the operation has been abandoned in favour of replacement in younger age groups as well.

FIG. 255.—Intertrochanteric osteotomy at the hip with internal fixation using a Müller-Harris spline.

Correction of deformity now forms a secondary objective of osteotomy although the procedure was originally introduced for this purpose. There is some clinical evidence to suggest that osteotomy performed early in the disease may reverse the pathological process. If this is so, osteotomy represents the only operation capable of producing this result. It has additionally been argued that osteotomy can be used to improve the stability of a subluxing hip and to bring relatively healthy cartilage into the line of maximum weight-transmission.

Whatever may be the reason, osteotomy brings significant relief of pain, especially

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Nigel Henry Harris, Contemporary. Orthopædic Surgeon, St. Mary's Hospital, Harrow Road, London.

of night pain, to about 60% of patients who have the operation. Stiffness is often not relieved, however, so that in terms of the return of function osteotomy is by no means an entirely satisfactory procedure.

THE SURGERY OF THE INFLAMMATORY ARTHROPATHIES

Of the inflammatory arthropathies by far the commonest requiring surgical relief is RA and it is with this disease that this section is largely concerned. During the active phase of RA the joint surfaces may become partially or totally destroyed. Even if the active phase of the disease then remits, symptomatic progression in the involved joint is common as a consequence of secondary degenerative changes. Thus the surgery of chronic, 'burned-out', RA merges into that of OA.

Of the other inflammatory arthropathies, ankylosing spondylitis affecting the spine and hips and Reiter's disease (Chap. 59) affecting the feet may require surgical relief.

RA in childhood (Still's disease (p. 237)) does not readily lend itself to the surgical procedures available in the adult: operations on bones are avoided if possible because they may interfere with growth and soft tissue procedures are rarely indicated.

The Upper Limb

The Hand and Wrist.—*Synovectomy* is employed at the metacarpo-phalangeal joints of the fingers, the radio-carpal and inferior radio-ulnar joints, and for the extensor (and, less commonly, flexor) tendon sheaths.

At the metacarpo-phalangeal joints of the fingers the synovial tissue erodes the cartilage and bone. As a consequence the joints subluxate and finally dislocate. Since the finger flexors are stronger than the extensors, dislocation takes the form of palmar and then proximal displacement of the phalanx on the metacarpal. A similar mechanism produces anterior dislocation of the wrist, and disruption of the inferior radio-ulnar joint. The extensor tendons may rupture as a consequence of a nutritional disturbance, combined with abrasion over the prominent dorsum of the distal radius and ulna. As the fingers deviate ulnar-wards the extensor tendons come to lie to the medial side of the metacarpal head so that their line of pull tends to exacerbate this deviation.

By removing the synovium before subluxation has taken place this evolving deformity may be arrested. At this stage, however, marked pain from capsular distension is not a feature so that the surgical decision is a difficult one: probable slowing of the progression of deformity and some pain-relief have to be set against the loss of movement which often follows this procedure.

At the time of synovectomy the capsule may be incised and reefed so as to stabilise the joints and re-align the extensor tendons. The tendons themselves may be repaired if they have ruptured and their synovial sheath may be excised if their bulk interferes with the function of the hand (p. 248).

Once deformity has occurred, and especially when the metacarpo-phalangeal and wrist joints have subluxated, operations on the bones of the hand are indicated.

Arthrodesis of the radio-carpal joint is an excellent procedure and is one of the mainstays in the surgical treatment of the rheumatoid hand. The wrist can be rendered stable and pain-free so that the power of the grip is much improved. The loss of movement compared with that occurring in the normal wrist impairs function very little: compared with the pre-operative state of the wrist any loss is insignificant. Replacement of the wrist is starting to show encouraging results.

Excision arthroplasty of the metacarpal heads and of the bases of the phalanges from dislocated metacarpo-phalangeal joints makes it possible to realign the fingers with the metacarpus. The consequent improvement of function is limited by stiffness at the false joint due to adhesion formation. In an attempt to improve motion *total replacement arthroplasty* may be employed. Excision of the dorsally dislocated distal

ulna from the inferior radio-ulnar joint (a second example of *excision arthroplasty*) usually combined with fusion of the radio-carpal joint, produces a useful increase in the range of painless supination and pronation.

The Elbow.—*Synovectomy* is occasionally useful when the joint is painful, the synovial membrane thickened, but the bone intact.

Arthrodesis is occasionally indicated to confer painless stability, but the objections to this procedure in RA—that fusion is difficult to obtain and that the presence of other involved joints makes loss of movement functionally objectionable—usually outweigh the advantages.

Excision arthroplasty of the radial head may increase the range of painless motion—especially of pronation and supination. Excision of the entire joint may provide a good range of painless movement but the associated instability is such that little increased function is obtained.

Total replacement arthroplasty of the humero-ulnar joint is now giving increasingly encouraging results.

The Shoulder.—*Synovectomy* is not undertaken at this joint since the membrane is relatively inaccessible surgically and its bulk is hard to assess clinically.

Arthrodesis is hard to secure in RA, but is occasionally indicated in a severely painful joint. The loss of total shoulder movement is not as severe as might be expected since scapulo-thoracic motion is unaffected and by itself provides a useful range of shoulder movement.

Excision arthroplasty of the humeral head may be undertaken as an alternative to arthrodesis: the operation and the after-care are less arduous than for arthrodesis and the pain relief is comparable. However the false joint is too unstable to allow effective abduction so that the functional result is worse than that following arthrodesis. *Partial replacement arthroplasty* using a prosthetic replacement for the humeral head (the Neer prosthesis) has proved useful in RA. *Total replacement arthroplasty* is available but the results are not encouraging.

FIG. 256.—Excision of the metatarsal heads (Fowler's operation).

The Lower Limb

The Toes.—The commonest consequence of RA requiring surgical relief in the feet is a claw toe deformity. As a result of this deformity—which consists of dorsal dislocation of the phalanges on the metacarpal heads and flexion contractures of the proximal inter-phalangeal joints—the dorsum of the involved toes rubs on the upper of the shoe whilst the head of the metatarsal is driven painfully downwards through the sole on weight-bearing.

Synovectomy has not been used extensively in an attempt to prevent this deformity in the foot in the way in which it has been used in the hand. However *excision arthoplasty* of the metatarsal heads, together occasionally with excision of the base of the proximal phalanx, is used commonly (fig. 256). At the time of excision arthroplasty, the extensor tendons may be divided. This procedure allows the toes to be brought into line with the forefoot and very successfully relieves pain under the metatarsal heads. Post-operatively the gait lacks the 'toe-off' spring of normal gait, but such a spring has already disappeared by the time the operation becomes indicated. At the metatarso-phalangeal joint of the great toe the deformity is that of a hallux valgus rather than a claw toe, but again the operative treatment is *excision arthroplasty*.

As an alternative to excision arthroplasty, grossly deformed toes may be amputated through the metatarso-phalangeal joints.

Charles Sumner Neer II, Contemporary. Orthopædic Surgeon, Columbia-Presbyterian Medical Center, New York.
Alan William Fowler. *(See p. 216.)*

The Midtarsal and Ankle Joints.—Pain arising from the midtarsal and ankle joints is best treated by well-fitting shoes or boots with appropriate insoles. Isolated involvement of one of these joints may merit arthrodesis, or, at the ankle, total replacement.

The Knee.—Total replacement has now supplanted all other forms of surgical treatment for the arthritic knee. As at the hip therefore the basic surgical decision is simple: if the patients disability is tolerable it should be accepted; if it is not the knee

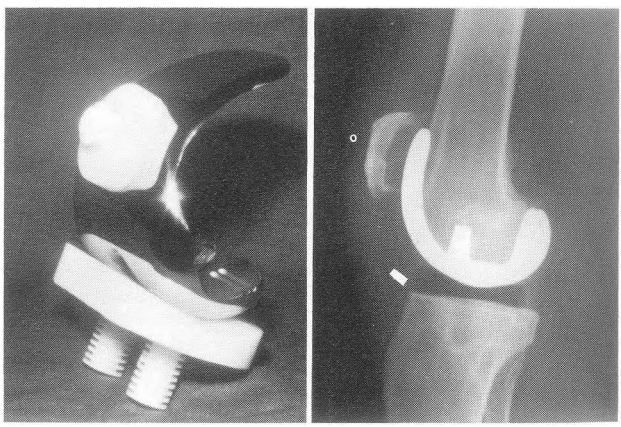


FIG. 257.—Replacement of the knee with the ICLH (Imperial College—London Hospital) prosthesis.

should be replaced. A number of procedures and prostheses is available, one of which is illustrated in fig. 257.

The Hip.—*Total replacement arthroplasty* using the implants already described (p. 249) is now the standard, indeed the only, surgical treatment for the hip in RA: at its best the operation results in almost normal hip function.

Synovectomy is not undertaken at the hip for the same reasons as those which apply to the shoulder. *Arthrodesis* is never indicated since it suffers from the same objections as those which apply to arthrodesis of the elbow and knee. Furthermore joints in addition to the involved hip have to be immobilised in plaster-of-Paris to obtain fusion, and in RA this risks disastrous stiffness affecting several joints. *Excision arthroplasty* is only used as a salvage procedure after failure of total replacement arthroplasty and takes the form of removal of the femoral head and proximal femoral neck (the Girdlestone operation). This operation relieves pain and permits a sufficient range of movement to enable the patient to lie and sit comfortably. The hip, however, is unstable so that walking is difficult and usually requires the aid of two sticks. *Interposition arthroplasty* and partial replacement has now been abandoned in favour of total replacement.

The Spine.—RA may affect all the facet joints of the spine but surgery is indicated only in the cervical spine, especially at the atlanto-axial joint, where the disease may lead to instability of the atlas on the axis with consequent danger to the spinal cord. This complication of RA is treated by *arthrodesis* (called ' fusion ' when applied to the spine) of the unstable level.

Ankylosing spondylitis produces progressive, rigid, flexion contractures particularly at the cervical and lumbar levels. Arthrodesis has not been attempted to arrest the progress of the deformity but lumbar and, more hazardously, cervical spinal *osteotomy* is used to correct the deformity followed by a fusion at the osteotomy site.

Gathorne Robert Girdlestone, 1881–1950. Nuffield Professor of Orthopædic Surgery, Oxford.

CHAPTER 16

TUMOURS OF BONES AND JOINTS

ALL primary bone tumours are rare: the incidence of osteosarcoma, the commonest primary malignant bone neoplasm, is only 1 per 230,000 of population. From the collective standpoint of the community therefore they are unimportant, almost insignificant. From the individual standpoint of the patient and his family, however, they may represent a tragedy, for many of the malignant tumours occur in the young (at which age they constitute one of the commonest malignant tumours), they are usually fatal, and the attempt to cure them involves mutilating surgery.

Nomenclature and Classification

These tumours are named after the predominant type of tissue matrix formed by the tumour. The benign tumours take the suffix—'oma' and the malignant tumours the suffix—'sarcoma'. Thus a benign tumour forming bone and/or osteoid is named an osteoma and the corresponding malignant tumour an osteosarcoma. Tumours forming a cartilaginous matrix are named chondroma and chondrosarcoma, the fibrous series fibroma and fibrosarcoma. Tumours differentiating to a synovial pattern are named synovioma and synovial sarcoma. A number of primary bone tumours arise from the cells contained in the bone marrow and these are also named, so far as possible, after the type of tissue differentiation. The matrix of some tumours is too poorly differentiated to allow specific classification.

Based upon the appearance of the cells and matrix of the tumour, Price has classified bone tumours into 11 groups, most of which may be subdivided into benign and malignant (Table 1). In this chapter only the clinically more important tumours will be considered, excluding those of dental and bone marrow origin.

BENIGN TUMOURS

Pathology

Most of the benign tumours of bone occur in children and young adults and often stop growing with the cessation of skeletal growth. Such lesions might more properly be regarded as localised dysplasias rather than neoplasias, and indeed those which are multiple, for example multiple osteochondromas and chondromas, may well be viewed in this light.

The exceptions to the general rule that the tumour ceases to grow at skeletal maturity are giant cell tumours, which are probably better regarded as locally malignant, and osteoid osteomas, the precise pathological nature of which is unknown. Aneurysmal bone cysts are almost certainly not neoplastic but can conveniently be considered under the heading of benign tumours.

Charles Hedley Godfrey Price, Contemporary. Formerly Research Fellow in Pathology, University of Bristol England.

TABLE I. TUMOURS OF BONES AND JOINTS

The Bristol Bone Tumour Registry Classification (Revised—1969)

Section A. Angioid tumours	
Benign	Angioma
	Aneurysmal Bone Cyst (? neoplasm)
	Glomus Tumour
Malignant	Haemangioendothelioma (Angiosarcoma)
Section B. Bone forming Tissue	
Benign	OSTEOMA
	Osteoblastoma
	Osteoid Osteoma (? neoplasm)
Malignant	OSTEOSARCOMA
	Juxtacortical osteosarcoma
Section C. Cartilage forming tumours	
Benign	OSTEOCHONDROMA
	CHONDROMA
	Chondroblastoma
Malignant	CHONDROSARCOMA
Section D. Dental and allied structures	
	Odontogenic cyst
	Cyst—not clearly odontogenic
	Ameloblastoma
	Composite odontome
	Malignant odontome
	Cementoblastoma
Section E. Embryonic Vestigial Tissue	
Benign	—
Malignant	Chordoma
Section F. Fibroblastic Tissue	
Benign	Fibroma. (Non-osteogenic: chondromyxoid: desmoplastic: alveolar)
	Myxoma
Malignant	FIBROSARCOMA (Inc. spindle cell sarcoma)
Section H. Heterotopic Tissue	
Benign	Dermoid
Malignant	Adamantinoma (of long bones)
	Central maxillary carcinoma
Section N. Non-osseous connective tissue	
Benign	Lipoma
	Neurofibroma
	Neurilemmoma
Malignant	Liposarcoma
	Reticulosarcoma
	Myeloma
	Leukaemia
	Ewing's tumour
	Leiomyosarcoma
Section S. Synovium	
Benign	SYNOVIOMA
	Chondroma
Malignant	SYNOVIAL SARCOMA (Malignant synovioma)
Section U. Undifferentiated connective Tissue	
Benign	GIANT CELL TUMOUR (osteoclastoma)
Malignant	Malignant forms of giant cell tumour
Section X. Undiagnosable primary tumours	

Osteochondroma.—This lesion is almost certainly the result of a localised disturbance of bone growth at an epiphysis. A portion of epiphyseal cartilage remains in the periosteum of the metaphyseal segment of the bone and endochondral ossification takes place in it, usually in continuity with the metaphyseal cortex. The cartilage cap proliferates, endochondral ossification continues, and thus the osteochondroma grows. With continued bone growth the osteochondroma moves away from the epiphysis and tends to become ' hooked ', its tip pointing away from the epiphysis (fig. 259). Growth of an osteochondroma usually ceases with the completion of skeletal growth. The lesion may be solitary or multiple (p. 281).

Compact Osteoma (syn. Ivory Exostosis).—This lesion consists of a small knob of extremely hard, dense, but otherwise normal bone usually arising on the inner or outer table of the skull.

Osteoid Osteoma.—This ' tumour ' consists of a small area of arborescent trabeculæ of osteoid and bone with a vascular connective tissue ground work. The tumour itself is usually less than 1 cm. in size and provokes dense new bone formation around it. Its neoplastic nature is unsure, and

James Ewing, 1866–1943. Professor of Oncology (Tumours), Cornell University Medical College, U.S.A

the lesion may possibly be a low grade osteitis. The tumour is most commonly seen in patients between 10 and 25 years old, is slightly more common in males, and usually occurs in the long bones. Any bone may be involved, the most commonly involved bones being the femur and the tibia. Lesions over 1 cm. in size are sometimes designated 'Osteoblastoma', but are histologically indistinguishable from Osteoid Osteomas.

Chondroma.—This tumour consists of a lobulated mass of cartilage. The tumour may arise in any bone, but is most frequent in the metacarpals, phalanges or metatarsals (fig. 258). Occasionally the tumours arise in synovial membrane. If situated in the medulla (*enchondroma*), the bone is thinned or expanded by the tumour so that pathological fracture is common. Tumours on the surface of the bone are known as *ecchondromas*. The matrix of the tumour may calcify and sometimes ossify. Like osteochondromas, chondromas may be solitary or multiple (p. 281). Malignant change is rare in solitary tumours, but may occur when the tumours are multiple.

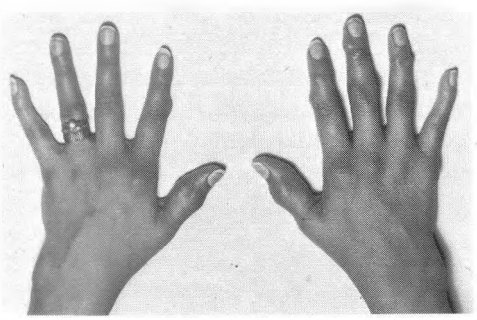


FIG. 258.—Multiple chondromas in the phalanges resulting in milder (and more typical) deformities than those shown in fig. 260.

Fibroma.—These occur in bone as islands of fibrous tissue of characteristic radiographic appearance. They range from a small oval cortical defect to quite large lesions. Areas of fibrous dysplasia are distinguished from fibromas by the presence within them of new bone.

Aneurysmal Bone Cysts.—These occur mainly in the ends of growing long bones. They form expanding osteolytic lesions containing bloody fluid through which pathological fractures may occur. Callus formation following such a fracture may obliterate the cyst. Care must be taken to distinguish them from osseous hæmangiomas.

Clinical and Radiological Features

Tumours growing on the surface of a bone, such as osteochondromas, may form an appreciable mass or they may interfere with the action of an adjacent soft tissue such as a tendon. The complaint of severe or persistent pain in such a tumour should raise the suspicion of malignant change.

Lesions which replace bone, such as chondromas or bone cysts, may present with pain, a pathological fracture, or deformity.

Radiologically, benign lesions have certain features in common which suggest their benign nature. These are:

1. Growth of the lesion is slow, and when situated within a bone it leads to cortical erosion and expansion.

2. The line of separation between the tumour and adjacent normal tissue

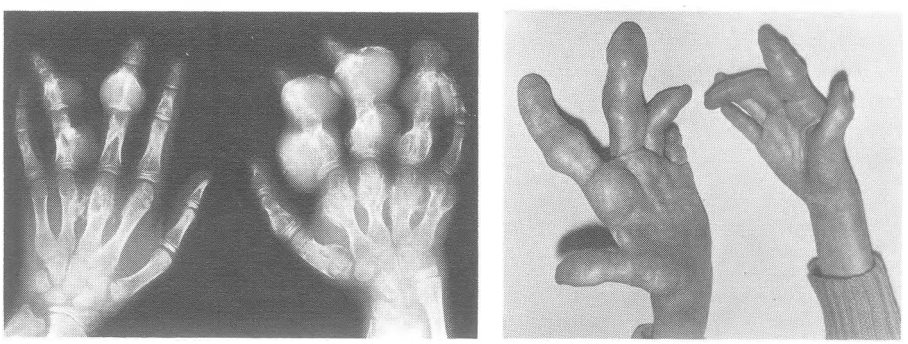
is usually sharply defined. This applies equally if the tumour is bony and lies in soft tissue, or if the tumour is radiolucent and lies in bone.

The radiological features of the common benign tumours are illustrated in figs. 259 and 260.

Biopsy.—A diagnosis can usually be made on the basis of the clinical and radiological appearances but, as a general rule, the lesion should always be biopsied, or when possible excised, since histological examination is advisable.

FIG. 259.—Multiple osteochondromas (exostoses) each of a typical hooked shape growing away from the epiphysis, of all the bones round the knee.

Osteoid Osteoma, alone amongst the benign neoplasms of bone, presents with pain which is severe, unrelieved by rest, but often specifically relieved by salicylate. The only abnormal physical sign is bone tenderness, and even this is often absent so that patients with osteoid osteomas may sometimes be regarded as hysterical. Radiologically, the bone in the area of the tumour is densely sclerotic. Towards the centre of the sclerotic area and often obscured by it, there is a radio-opaque ' nidus ' in a radiolucent zone which represents the tumour itself (fig. 261). If the tumour lies in cortical bone, there may be overlying subperiosteal new bone formation. Tomography may be required to demonstrate the nidus.



(*a*) (*b*)

FIG. 260.—(*a*) Multiple ecchondromas in the hand (Ollier's disease). (*b*) Appearance of the hands shown in (*a*).

Treatment

Osteomas can be ignored unless they give rise to symptoms which demand treatment. They should then be removed.

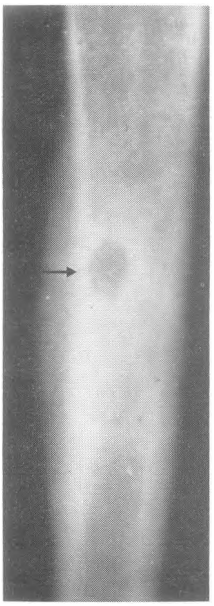


FIG. 261. — Osteoid osteoma in the tibia.

Lesions which significantly weaken bone such as chondromas or aneurysmal bone cysts should be treated, even if symptomless, to prevent pathological fracture. The lesion is excised by curettage and the resulting cavity in the bone is packed with autogenous bone chips. (In multiple chondromatosis (Ollier's disease), it may, of course, be impractical to treat all the tumours in this way.)

MALIGNANT TUMOURS

Pathology

Osteosarcoma. — This is a highly malignant tumour: reported 5 year survival rates vary, but few exceed 15%. Osteosarcomas arising in hitherto normal bone, occur mainly in the second and third decades of life and tend to occur in the metaphyseal region of the long bones, especially those of the leg. Paget's disease predisposes bone to develop a sarcoma, about 60% being osteosarcomas. Such tumours develop late in life (because Paget's disease is a disease of the elderly), arise in any bone involved by Paget's disease, and are almost invariably fatal. Excessive irradiation also predisposes bone to sarcomatous change.

The tumour is composed of pleomorphic cells which invade and destroy the bone producing a radiologically osteolytic lesion with ill-defined edges. Although they destroy normal bone, the tumour cells may themselves produce abnormal bone of irregular structure. If the tumour has invaded the soft tissue (fig. 262), neoplastic bone can be seen radiologically outside the confines of the normal bone, where it tends to be deposited as irregular spicules of bone radiating away from the shaft to give a ' sunray ' appearance. As the tumour grows in size, the periosteum is elevated so that above and below the tumour reactive new bone may be deposited subperiosteally. Radiologically, this bone appears as a triangle of new bone (*Codman's triangle*, fig. 265).

Metastasis occurs via the blood-stream to produce pulmonary secondary deposits. Locally, pathological fracture may take place. Finally, if the tumour is untreated, it fungates to produce an infected malignant ulcer sometimes overlying a painful pathological fracture.

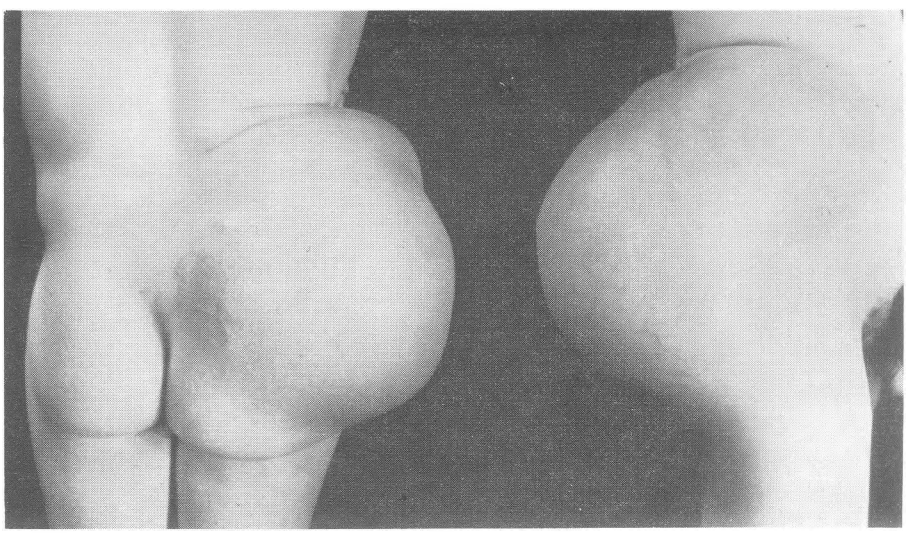
FIG. 262. — Osteosarcoma of the humerus. Note that the tumour is metaphyseal and eccentric and that the underlying cortex is eroded. The extraosseous mass seen here is much larger than that which might be expected from the mere trace of new bone which was present in the radiograph.

Louis Ollier, 1830–1900. Professor of Surgery, Lyon, France.
Sir James Paget, 1814–1899. Surgeon, St. Bartholomew's Hospital, London.
Ernest Amory Codman, 1869–1940. Surgeon, Massachusetts General Hospital, Boston, Massachusetts, U.S.A.

Chondrosarcoma.—This tumour arises usually in hitherto normal bone. It may however appear as a complication of any pre-existing cartilagenous tumour. It may arise in any bone, with a predilection for the flat bones and

263(*a*)

FIG. 263.—(*a*) Chondrosarcoma of the ilium. (*b*) The clinical appearance of the buttock shown in (*a*).



263(*b*)

ribs. Unlike osteosarcoma, it occurs mainly in middle aged persons, is very rare in children, and uncommon in teenagers.

Although malignant, the tumour may be slow-growing and mitoses so infrequent that malignancy may be impossible to decide on biopsy material. The tumour is locally invasive and, although often slow to metastasise, it may give rise to blood-borne pulmonary secondaries which are eventually fatal.

The matrix of the tumour is more or less cartilagenous. It may ossify in patches but more commonly calcifies.

The tumour invades and destroys bone, transgresses the periosteum and invades the adjacent soft tissues. In the spine, where it is rare, the tumour usually manifests itself whilst it is still small by causing collapse of a vertebra or by invading or compressing the cord or a nerve root. In the pelvis, on the other hand, the tumour may reach a considerable size before the patient notices a mass (fig. 263).

Fibrosarcoma.—These tumours are usually composed of spindle-shaped fibroblasts which produce a collagenous matrix but they may be pleomorphic. In bone, the tumour arises centrally or periosteally. (The tumour may, of course, also arise in any soft tissue.) They are of varying malignancy, produce blood-borne pulmonary secondaries, and are associated with a 5 year survival rate of about 30%.

Synovial Sarcoma.—This is histologically a bi-phasic tumour composed of elements simulating synovium mingled with malignant fibroblasts. It is extremely malignant, and may appear at any age.

The tumour usually arises close to a major joint (most often the knee, ankle or wrist), but it may arise, apparently primarily, in connective tissue, in muscle or subcutaneously. Metastasis appears to occur via the blood stream, the lymphatics and by the migration of cells through the tissue planes of the limb. Secondary involvement of regional lymph nodes is not unusual.

Giant Cell Tumour.—It is uncertain from what cell this tumour arises. It is composed of undifferentiated spindle cells and multinucleate giant cells in a vascular stroma. Microscopically it is indistinguishable from the so-called ' brown tumour ' of hyperparathyroidism and this condition must be excluded by biochemical and radiological investigations (p. 305).

The tumour occurs most often in the third and fourth decades of life and tends to be more common in women. The epiphyseal regions of the long bones, especially around the knee, are the most common sites of origin but tumours of the humerus, radius and ulna occasionally occur.

The tumour is osteolytic and expands the bone with thinning, often perforation, of the cortex to cause a pathological fracture in some cases. The bone is destroyed irregularly so that the tumour is traversed by remnants of the original bone and comes to lie in a cavity with heavily trabeculated walls. As a consequence, it has a typical ' soap-bubble ' radiological appearance (fig. 264). It rarely invades soft tissues but may do so when fracture occurs. Thus locally the tumour is only of low-grade malignancy. Metastasis is rare, but occurs via the blood stream to the lungs. Recurrence following local removal is common, and such recurrences are more likely to be frankly malignant than the original tumour.

Secondary Tumours in Bone.—Two thirds of all secondary malignant tumours in bone arise from carcinomas of the breast or prostate. One sixth arise from a variety of other primary neoplasms (especially those of the bronchus, kidney, and thyroid). In the remaining one sixth, no primary tumour is found at the time when the secondary presents. The majority of

tumours are osteolytic but a few, mostly arising from the prostate, stimulate new bone formation and are then called ' osteosclerotic '

Clinical and Radiological Features

Regardless of the pathology, these tumours present by causing pain, a noticeable lump or tender area, or a pathological fracture.

The diagnosis depends upon the clinical features of the tumour, its radiological appearance and its histology: the clinical features suggest a neoplasm; the radiological appearances indicate its size and whether it is benign or malignant; the diagnosis usually depends upon histological study of an adequate sample of tissue. Hæmatological investigations are unhelpful. An exact diagnosis can usually be made only by considering the clinical, radiological and histological features of the tumour in conjunction with each other, but even then, an exact diagnosis cannot be made in approximately 10% of tumours.

Clinically the important features are the mode of presentation (the more malignant the tumour the more likely is it to present with severe pain or pathological fracture rather than as a mass), the age of the patient, the fact that on physical examination the tumour is attached to bone, the location of the tumour, and the presence of any evidence of a primary neoplasm elsewhere. The clinical features of the more common bone tumours are set out in Table 2 (p. 262).

Radiologically two features suggest malignancy irrespective of the exact nature of the tumour. These are:

1. The presence of an ill-defined osteolytic lesion in the bone. Although the lesion is osteolytic, tumour new bone or calcification may occur within it or reactive new bone may occur around it.

2. Evidence that the tumour has breached the periosteum and invaded soft tissues.

The distinguishing radiological features of the commoner tumours are summarised in Table 2 and illustrated in figs. 263–266.

Biopsy.—Histological material should always be obtained by biopsy before treatment is undertaken. If possible, open biopsy is undertaken: the naked-eye appearance of the tumour is noted and material is taken from the edge of the tumour together with adjoining normal tissue. The cell morphology of the tumour, the type of matrix formed, the presence of abnormal mitoses, and the invasion of adjacent tissues permit a diagnosis of malignancy and usually serve to identify the tumour. Needle biopsy may be employed for tumours of the spine but since this technique yields only a restricted sample of tissue it is not as reliable as open biopsy.

Treatment

Osteosarcoma.—There is no general agreement as to how this tumour should be treated but the most widely followed practice is that first advocated by Sir Stanford Cade. The tumour is irradiated with 9000 rads over 3 months, a dose which locally controls most tumours. Over the following 6 months pulmonary secondaries become manifest in most patients and from these they die without having had (what would have been) an unnecessary

Sir Stanford Cade, 1895–1973. Surgeon, Westminster Hospital, London.

TABLE 2. *Malignant Tumours of Bones and Joints: The principal clinical and radiological features of diagnostic significance*

	Osteosarcoma	*Chondrosarcoma*	*Fibrosarcoma*	*Synovial Sarcoma*	*Giant Cell Tumour*	*Secondary Neoplasm*
Common Mode of Presentation	Pain, swelling and/or pathological fracture	Mass	Mass or pathological fracture	Soft tissue mass	Pain, swelling ± fracture or joint symptoms	Pain and/or pathological fracture
Age of the Patient	2nd and 3rd decades, or the elderly in Paget's disease	3rd and 4th decades	Any age—mainly 25–60	Any age	3rd and 4th decades	Middle-aged and elderly
Location of the tumour	Ends of shafts of long bones especially femur, tibia, humerus. Any bone with Paget's disease	Ribs, ilium, scapula. Proximal ends of femur and humerus	Any bone. (May occur in Paget's Disease.)	Usually a joint	Epiphyseal region of long bones especially distal femur, proximal tibia and fibula	Any bone
Radiological Appearance	Area of bone destruction with ill-defined edges. Tumour transgresses the bone to produce a shadow in the soft tissues. Codman's Triangle. Sunray spicules. Pathological fracture. Secondaries in the lungs	Area of bone destruction with ill-defined edges. Tumour transgresses the bone to produce a shadow in the soft tissues. Large tumour (in the flat bones) with ossifying or calcifying matrix	Area of bone destruction	Soft tissue swelling ± spotty calcification.	Expands the bone. Sharp edge—unless frankly malignant. Soap-bubble appearance	Area of bone destruction with ill-defined edges. Occasionally marked sclerosis in adjacent bone.

and mutilating amputation. Those without secondaries at 6 months are then treated by amputation since in this group amputation offers a real chance of cure. Patients whose tumours are not locally controlled by radiotherapy

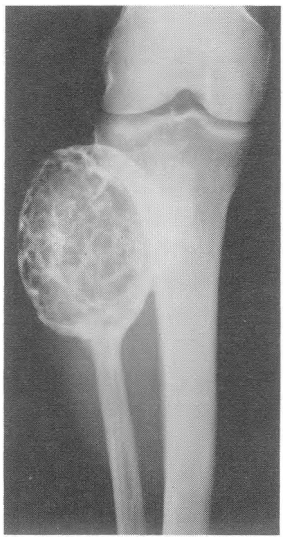


FIG. 264.—Giant cell tumour of fibula.

FIG. 265.—Osteosarcoma of the femur. Note Codman's triangle well seen in the proximal part of the tumour and 'sunray spicules'.

should be treated by earlier amputation to avert the suffering of death with painful fungating malignant ulcer and a pathological fracture.

Amputation should be carried out at the site of election through the bone or joint immediately proximal to that involved in the tumour. Thus for example, osteosarcoma of the tibia should be treated by amputation through the femur, and at the proximal end of the femur disarticulation of the hip or hind-quarter amputation is required. Solitary pulmonary secondaries have been successfully treated by lobectomy.

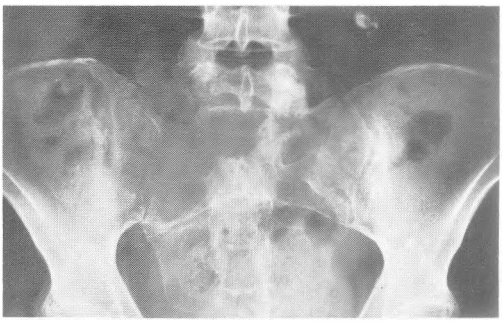


FIG. 266.—Multiple osteolytic secondaries in the pelvis.

Chondrosarcoma.—In spite of the difficulty that this tumour nearly always arises in the proximal limb skeleton, treatment is by amputation. The tumour is nearly always large when first diagnosed, and when large is radio resistant. Hence radiotherapy has no part to play in its treatment.

Fibrosarcoma.—This tumour is treated by early amputation or, in the trunk, by wide block resection.

Synovial Sarcoma.—This tumour carries an extremely poor prognosis regardless of treatment: few or no survivors have been reported after 5 years. The tumour should be treated by a proximal amputation together with irradiation of the stump of the limb.

Giant Cell Tumours are treated where possible (for example in the proximal fibula or distal radius) by block resection of the tumour and adjacent bone. Unfortunately such a resection is often disabling since the tumour may be located, for example, in a tibial condyle, block resection of which would render the knee useless. An attempt should then be made to eradicate the tumour by curettage followed by packing the cavity with bone chips. Unfortunately, the incidence of recurrence following curettage is high. Recurrent tumours may be frankly malignant and require amputation.

Ewing's Tumour.—There is uncertainty about the pathology of this lesion. Some hold that it is a malignant endothelioma arising from the endothelial cells in the bone marrow, others that it represents a secondary deposit from a neuroblastoma. The tumour usually occurs in the middle of a long bone in childhood or early adolescence. Radiography shows a number of layers of sub-periosteal new bone formation expanding the shaft and giving the characteristical appearance of a cut onion. Clinically a painful hot swelling is noticed and the patient may show intermittent elevations of temperature. In this way the condition may be difficult to distinguish from acute hæmatogenous osteomyelitis. Treatment is by radiotherapy although the long-term prognosis is guarded and widespread dissemination usually occurs. Amputation may be considered after irradiation.

CHAPTER 17

CONGENITAL DISEASES OF BONES AND JOINTS

THE congenital diseases of bones and joints are of two kinds: developmental anomalies and dysplasias.

The developmental anomalies are characterised by the fact that the fundamental abnormality of growth occurs *in utero* and is static at birth: the evolution of the disease thereafter is due to secondary changes.

The dysplasias are characterised by abnormal growth both *in utero* and throughout the period of post-natal skeletal development. Many dysplasias are clinically detectable at birth and hence are certainly congenital; others remain undetected until later in life. It is assumed, almost certainly correctly, that in the latter a tendency to abnormal growth was present at birth and hence that they are also congenital.

Some congenital diseases are genetic in origin, some are thought to be due to disturbances in the intra-uterine environment. For the majority, the aetiology is unknown.

DEVELOPMENTAL ANOMALIES

Certain of these disorders are of the greatest orthopædic significance and will be dealt with at length. Others are of minor significance and will only be mentioned.

THE LOWER LIMB

Congenital Dislocation of the Hip (CDH)

Overall Incidence.—Before it was realised that instability of the hip could be diagnosed at birth (p. 266) CDH was diagnosed in 1 child in 1,000. The reported incidence of unstable hips (see below) is much higher, varying from 8 to 20 per 1,000 live births.

Pathology.—The hip may be either dislocated or dislocatable at birth. The precise details of the morbid anatomy at birth are not certain, but the essential practical fact is that the bones of the pelvis and femur are broadly within normal limits, but the ligaments of the hip are unduly lax so that the femoral head can be dislocated from the acetabulum. The infant may lie with the hip in the dislocated position, or the hip may be in the reduced position but be easily dislocatable. Over the first few months of life the ligaments of the hip shorten so that if the femur is maintained in the acetabulum during this period, the hip becomes stable and normal development results. On the other hand, if the femoral head is allowed to remain out of the acetabulum during this period, shortening of the ligaments makes reduction of the dislocation increasingly difficult. If the hip is allowed to remain

dislocated until the child crawls and then walks, the acetabulum and femur fail to develop normally. It is then difficult to return the hip to normal whatever treatment is instituted, and osteoarthrosis in the involved hip may develop early in adult life.

Ætiology.—The condition may be unilateral or bilateral, and is familial. Three factors appear to be important in its ætiology:

1. An hereditary predisposition to joint laxity: the patient's relatives may either have dislocated hips themselves or may have hypermobile joints so that they can perform 'party tricks' such as extending their wrists and fingers until the fingers lie parallel with their forearm or extending their thumb until the thumb touches the radius;

2. Female sex on the part of the infant: the condition is 3 to 5 times more common in girls. This may be due to the fact that maternal *relaxin* crosses the placental barrier to enter the fœtus. If the hormonal environment of the fœtus is also female, relaxin acts on the fœtus' joints in the same way as it does on those of the mother to produce joint laxity in the pelvis.

3. Breech malposition of the fœtus: for infants who are either delivered in the breech position or who spend a substantial part of the last 2 months *in utero* in this position the incidence of instability of the hip is about 10 times as great as for infants with a vertex presentation. It is possible that in the breech position the uterus presses on the fœtal legs in such a way that, if the hip ligaments are already lax, dislocation may occur.

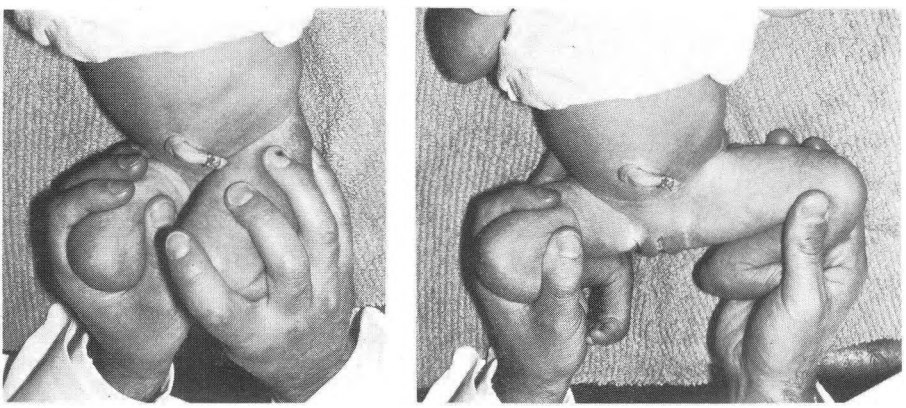
Clinical Features.—It is imperative that this condition be diagnosed in the first few days of life. This will only be done if the physician or midwife attending the delivery is aware of the existence of the condition and knows how to demonstrate its presence.

Diagnosis at Birth.—At birth the appearance of the infant is normal, and the hips can be put through a full range of movement. If the hip is dislocated, telescoping can in theory be demonstrated but in practice it is extremely difficult for the examiner to convince himself that this is occurring in a neonate. Thus conventional clinical examination of the hip reveals no abnormality. Radiologically, the epiphysis of the femoral head does not appear until the first year of life and the acetabulum is largely cartilaginous so that, even if the hip is dislocated (rather than dislocatable), there is no obvious radiological abnormality. Thus there is no way of detecting a dislocatable or dislocated hip at birth save by a specific examination designed to elicit instability. It is imperative that every doctor supervising a pregnancy should be familiar with this physical sign which is elicited as follows:—

The Demonstration of Instability of the Hip at Birth.—The demonstration consists of eliciting a click from the hip, and is known as Barlow's or von Rosen's sign. The legs are held as shown in fig. 267. The hip is then flexed to 90° and adducted. Whilst adduction is taking place, gentle pressure is exerted by the examining hand in a proximal direction along the long axis of the femoral shaft. As the femoral head rolls over the posterior lip of the acetabulum it may, if dislocatable (but not if dislocated), slip out of the acetabulum. The sensation which the examining hand receives as the hip

Thomas Geoffrey Barlow, 1915–1975. Orthopædic Surgeon, Salford Royal Hospital, Salford, England.
Sophus von Rosen, Contemporary. Orthopædic Surgeon, Malmö General Hospital, Malmö, Sweden.

slips over the posterior lip of the acetabulum is difficult to describe but is composed of a mixture of the feeling of abnormal posterior movement plus a distinct ' clunk ' as the femoral head leaves the acetabulum. If the hip is dislocated by this manœuvre or if it is already dislocated prior to examination, the second half of the physical sign can now be elicited. The hip is kept 90° flexed and, starting from a position of full adduction, is gently abducted. Now



(*a*) (*b*)

FIG. 267.—Von Rosen's or Barlow's sign. (*a*) The hip in full adduction. (*b*) The hip in full abduction: note that the knee touches the couch.

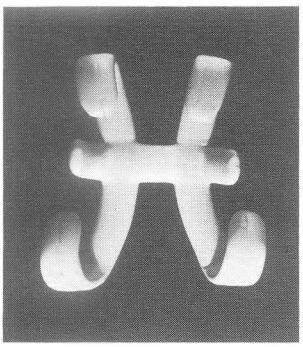
instead of exerting pressure proximally in the long axis of the femoral shaft, the examiner exerts pressure distally in the long axis of the shaft via his little finger placed proximal to the greater trochanter. The femoral head can now be felt to reduce with a soft ' clunk ' into the acetabulum somewhere during the course of movement from full adduction to full abduction. As abduction continues the femoral head remains in the acetabulum, demonstrating that the hip is stable in this position.

Occasionally a soft click can be felt in the hip which is noticeably different to the experienced examiner from the ' clunk ' of an unstable hip. The origin of these clicks is uncertain but they presumably come from soft tissue around the hip. Should the physician attending the delivery detect either a ' click ' or a ' clunk ' the patient should be referred immediately for an orthopædic opinion.

There are perhaps a few dislocated hips which cannot be reduced at birth. The diagnosis of dislocation in these hips at birth appears to be difficult or impossible.

The Treatment of CDH in the Newborn Infant.—The incidence figures quoted above show that in the absence of neonatal treatment only a minority of unstable hips at birth will develop into a persistent dislocation. All that is required to ensure that no unstable hip develops in this way is to place the femoral head in the acetabulum by the method described for the elicitation of von Rosen's sign (p. 266) and to keep it there until the hip is clinically stable and normal development is occurring radiologically.

Various splints have been devised to do this by keeping the hip in a position of 90° flexion and abduction (a position in which the femoral head is stable in the acetabulum) for a period of three months. All are satisfactory. One such splint is the von Rosen splint which is illustrated in fig. 268.



(*a*)

(*b*)

FIG. 268.—(*a*) The von Rosen splint made of padded malleable metal. (*b*) The splint applied.

The Diagnosis of CDH in a child of one year's age or more.—A discussion of the diagnosis and treatment of CDH in this age group used to fill a considerable part of textbooks of orthopædics, but it is very much to be hoped that no medical practitioner in the United Kingdom will ever in future have occasion to concern himself clinically with this problem. Unfortunately, because the method of making the diagnosis in the first week of life is not yet universally appreciated and implemented, it still happens that children present later in life with dislocated hips. This is a tragedy.

FIG. 269. — Asymmetrical skin creases in CDH.

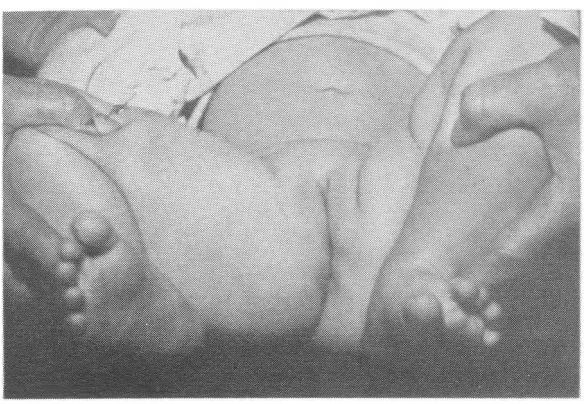


FIG. 270.—Limited abduction of the left thigh in CDH. (*J. C. R. Hindenach, F.R.C.S., London.*)

If one hip is dislocated, the skin creases in the groin and around the buttocks are asymmetrical (fig. 269). The affected hip has a full range of flexion, extension and rotation but cannot be fully abducted in 90° of flexion. In the infant, the normal hip will abduct until the knee nearly touches the

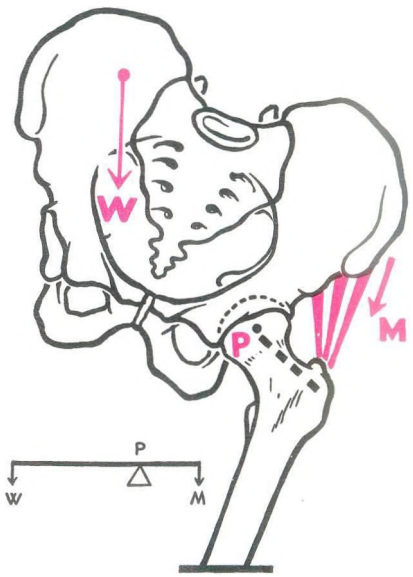
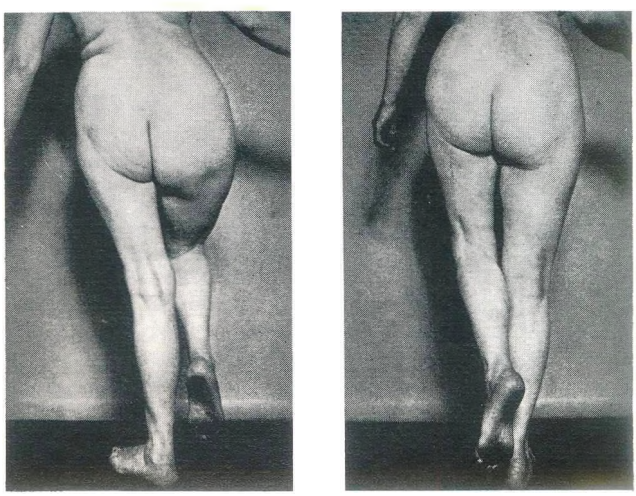


FIG. 271. — The physiological basis of Trendelenburg's sign. Contraction of the gluteus medius elevates the pelvis.

W = weight of body
M = muscle pull
P = pivot (the hip)

examination couch. The dislocated hip will abduct only to about 45° (fig. 270). Telescoping may be demonstrated.

If the patient is old enough to stand, Trendelenburg's sign is positive.



(*a*) (*b*)

FIG. 272.—(*a*) Trendelenburg's sign positive — standing on the defective hip. (*b*) Trendelenburg's sign negative—standing on the sound hip.

Friedrich Trendelenburg, 1844–1924 (see footnote, p. 75). Described this sign in 1895.

This sign is elicited by asking the patient to stand on the affected leg and noting the position taken up by the pelvis. If the sign is negative (i.e. the hip is normal), the pelvis rises on the unsupported side (figs. 271 and 272 (*b*)). If the sign is positive, the pelvis drops on the unsupported side (fig. 272 (*a*)). A positive sign is produced by any pathology which interferes mechanically with the abductor mechanism of the hip, for example dislocation of the hip, fracture of the neck of the femur, or paralysis of the abductors. If

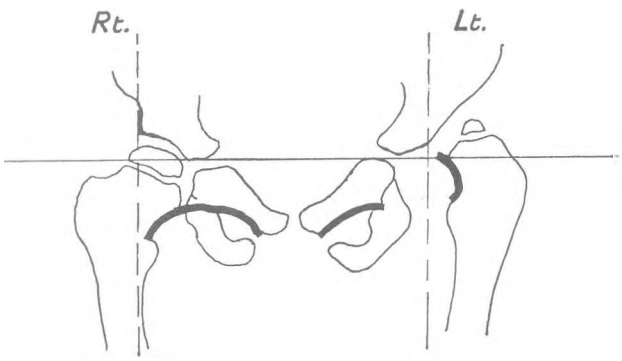


FIG. 273(*a*).—CDH (Lt). Shenton's line (black) broken on the left side. Note the small ossific nucleus on the dislocated side above the horizontal and lateral to the vertical line, and the shallow acetabulum with its undeveloped bony roof.

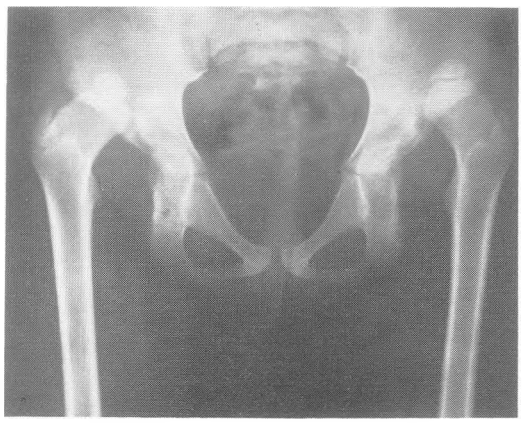


FIG. 273(*b*).—Bilateral CDH in a six-year-old girl.

Trendelenburg's sign is positive, the patient has a typical gait lurching downwards towards the unsupported side during the stance phase on the affected leg.

If both hips are dislocated, asymmetry of the groin creases is absent and the symmetry of the ' waddling ' gait (there being a Trendelenburg limp on both sides) often misleadingly makes it appear normal. Accordingly bilateral dislocation of the hips in infancy can be missed more easily than can unilateral dislocation.

Edward Warren Hine Shenton, 1872–1955. Surgical Radiographer, Guy's Hospital, London.

X-ray Examination.—In contrast to the situation in neonates, the hip is radiologically abnormal (fig. 273). The significant features are:—

1. Lateral and upward displacement of the epiphysis of the femoral head. This displacement can be demonstrated by drawing 2 lines, one horizontally through the tri-radiate cartilage and one vertically through the superior lip of the bony acetabulum: in CDH the femoral epiphysis lies above the first line and lateral to the second.
2. A break in Shenton's line. Shenton's line is a fortuitous radiological appearance consisting of the continuous curve produced by the inferior margin of the superior pubic ramus and the medial cortex of the femoral neck and shaft. If the hip is dislocated, this line will be broken by a step—because the femur moves upwards.
3. The capital femoral epiphysis is late to develop and when it does so is smaller than that on the unaffected side.
4. The superior lip of the bony acetabulum is also slow to develop so that it appears radiologically that the acetabulum is unduly shallow. If the hip continues to be dislocated throughout childhood, the final bony acetabulum is truly shallow. In infancy, however, the true acetabulum is often not particularly shallow since the cartilagenous development of the superior lip is not grossly abnormal.

Treatment between the ages of 6 months and 7 years (for unilateral dislocation) or 4 years (for bilateral dislocation).—Weight-bearing at the infant hip begins when the infant crawls at about 6 months. From this age until about 7 years for unilateral dislocation, and until about 4 years for bilateral dislocation, it is possible to reduce the hip and finally obtain fair function. Above these age limits, reduction is difficult and it is probably unwise to attempt it for reasons set out below; see Treatment in the Older Child (p. 272).

Treatment aims to replace the femoral head in the acetabulum and to keep it there whilst normal development occurs. Reduction is impeded by the facts that (1) the soft tissues have by this time developed in such a way as to conform with the abnormal position of the femur, and (2) the acetabulum has become filled with a mass of fibro-fatty tissue. Maintenance of reduction is difficult because: (1) the acetabulum is abnormally shallow and vertical, (2) the femoral head is smaller than normal, and (3) the femoral neck is usually retroverted on the shaft.

Closed ' Manipulation '.—It is sometimes possible under general anæsthesia to replace the femoral head in the acetabulum without any force being exerted on the leg. Should force be required the manœuvre is unwise, since it is likely to result in damage to the blood supply of the femoral head. If closed reduction under anæsthesia is thought likely to fail, it should be preceded by a period of traction in progressively increasing abduction, and for this a variety of special splints have been devised. The femoral head may reduce spontaneously during a period of traction, or it may be reduced under anæsthesia after a period of traction. Open adductor, and sometimes psoas, tenotomy is often needed to allow the hip to be fully abducted without forcibly compressing the femoral head.

Open Reduction is indicated if closed reduction fails. It may be difficult to determine if the femoral head has been truly replaced in the acetabulum by closed manipulation, or whether on the other hand it has been brought opposite the acetabulum, but held out from a full reduction by fibro-fatty tissue in the acetabulum or by the presence of a fold of capsule and acetabular labrum (the limbus) intruding between the femoral head and the superior

part of the acetabulum. The presence of such an impediment to reduction may be suspected if the femoral head persistently 'stands off' radiologically after an apparently satisfactory reduction, and may be confirmed by arthrography. The presence of a limbus, or clear-cut inability to reduce the dislocation by conservative means, are indications for open reduction. The hip is exposed and the soft tissues obstructing reduction are excised or released, so that the head can be repositioned in the acetabulum.

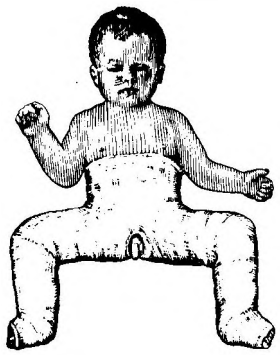


FIG. 274.—Plaster-of-Paris spica for the maintenance of the reduction of a bilateral CDH in neutral rotation and 90° abduction.

Maintenance of Reduction.—If the hip has been replaced either by closed or open means it must be maintained in the acetabulum for a period of months by some form of *splintage*. This may be done initially by traction on an appropriate splint or by a double hip spica applied with the hips held in the position of maximum stability. Not all hips are best immobilised in the same position, but stability is usually obtained in a position of abduction and flexion with some degree of external or internal rotation (fig. 274). Extreme internal or external rotation should be avoided since this tends to 'wring out' the vessels in the capsule of the hip and may interfere with the blood supply to the femoral head so that changes similar to those seen in Perthes' disease (p. 292) develop. After 3 to 6 months a hip spica may be exchanged for a Denis Browne splint if the child is under 2 years of age.

If reduction has been deferred until the bones of the hip have developed abnormally, *surgical reconstruction* of the hip may be required in addition to open reduction. The available procedures consist of:

1. *Pelvic Osteotomy* (Salter) which aims to rotate the acetabulum into a more horizontal position to cover the femoral head.

2. A *Shelf Operation* in which the superior lip of the acetabulum is reconstructed (so that it covers the femoral head) by a bone graft or by medial displacement of the acetabulum relative to the ilium after a pelvic osteotomy just above the hip.

3. *Intertrochanteric Rotation Osteotomy of the Femur* in which the proximal fragment is rotated, bringing the femoral head squarely into the acetabulum.

Treatment in the Older Child.—Putti suggested that the results of treatment in bilateral dislocations of the hip over the age of 4 years and in unilateral dislocations over the age of 7 years are worse than the results of the disease itself and thus that no treatment is indicated for these children in childhood. A lower age limit applies to bilateral dislocation because: (1) the limp is less noticeable; (2) as a consequence these patients, although they have a slightly abnormal posture and an abnormal gait, tend to live normal lives until their 40's or 50's when secondary osteoarthrosis may develop; (3) there is a danger that treatment may be more successful in one hip

Sir Denis John Wolko Browne, 1892–1967. Surgeon, Hospital for Sick Children, Great Ormond Street, London.
Robert B. Salter, Contemporary. Professor of Orthopædic Surgery, University of Toronto, Ontario, Canada.
Vittorio Putti, 1880–1940. Professor of Orthopædic Surgery, University of Bologna, Italy.

than in the other so that a bilateral dislocation is converted into a unilateral dislocation and (4) treatment may stiffen the hip, a tolerable outcome in one hip but a significant disability if it affects both.

Club Foot

Nomenclature.—' Club Foot ' is a loose term used to describe a number of different abnormalities in the shape of the foot. If the foot is fixed in a position of plantar-flexion so that it cannot be fully dorsiflexed, the deformity is described as one of ' equinus '. The opposite deformity is described as ' calcaneus '. If the foot is inverted and adducted at the mid-tarsal joint so that it cannot be fully everted the deformity is described as ' varus'. The opposite deformity is described as ' valgus '. Almost invariably two or more of the deformities are combined and by far the most common congenital combination is ' equino-varus '. The Latin synonym for club foot is ' talipes ' and the descriptive nomenclature for a club foot combines this term with the Latin description of the deformity. Thus the most common variety of congenital club foot is known as ' Talipes Equino-Varus ' (fig. 275).

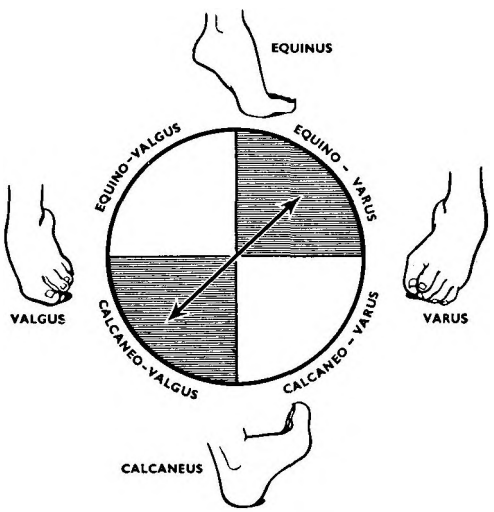


FIG. 275.—Nomenclature of talipes. Shaded quadrants indicate the common varieties.

Ætiology.—In the great majority of patients with congenital club feet the ætiology is unknown, and this group should properly be called idiopathic congenital club foot. But because this variety represents the majority of club feet, it is usually referred to simply as ' congenital club foot '.

It has been suggested that raised intra-uterine pressure forces the lower limbs of the fœtus against the wall of the uterus so as to mould the feet into the position of deformity. The presence of lesions in the skin over the convex aspect of the foot in talipes equino-varus which very much resemble healed pressure sores, lends support to this belief. It has alternatively been suggested that the primary disturbance is in the muscles of the calf or the soft tissues of the foot where some form of contracture, possibly ischæmic in nature, has been visualised as drawing the foot into a position of deformity. Neither of these postulated ætiological mechanisms have received widespread support and the most commonly held view is that the primary disturbance is a developmental defect of the soft tissues of the legs affecting particularly the ligaments on the concave side of the curve, or possibly, in the case of talipes equino-varus, the development of the neck of the talus.

Congenital club foot may be paralytic and secondary to myelodysplasia (Chap. 21). The deformities in arthrogryposis multiplex congenita may also include club feet. The club foot in this disease is gross, and nearly always associated with other deformities, in particular with clubbing of the hand (p. 277).

Morbid Anatomy.—Surprising as it may seem, there are relatively few accurate descriptions of the morbid anatomy of untreated club foot at or before birth and therefore the nature of the original morbid anatomy is un-

certain. Within a few months of birth the morbid anatomical features are as follows:—

1. Adduction and external rotation of the bones of the forefoot. The adduction occurs at the mid-tarsal and tarso-metatarsal joints, but also to some extent affects the bones themselves which are curved so as to be concave medially. The adduction may be so gross as to result in medial dislocation of the navicular from the talo-navicular joint. The external rotation is produced by torsion of the shafts of the metatarsals.

2. Inversion of the os calcis and of the navicular on the talus. The inverted os calcis and navicular carry the rest of the foot into inversion with them.

3. Plantar-flexion of all the bones of the foot including the talus. The talus, although plantar-flexed, is not as plantar-flexed as the other bones so that the head of the talus tends progressively to dislocate from the mid-tarsal joint in a dorsal as well as a lateral direction.

4. There is possibly an early abnormality in the neck of the talus which, it has been suggested, is angulated so that the head of the talus is displaced downwards and medially. Secondary changes occur in the head of the talus.

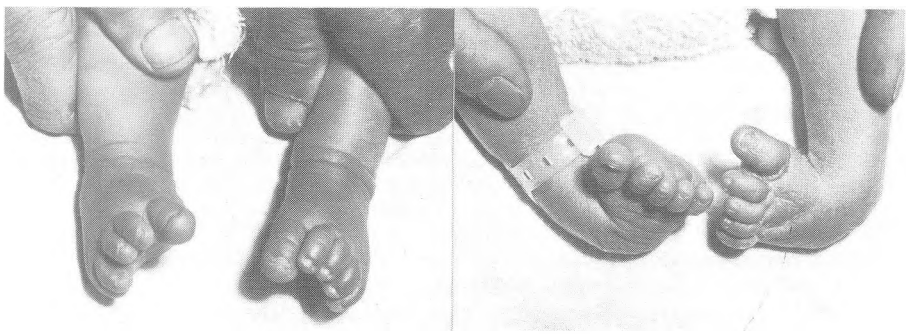
5. The os calcis may be small. As a consequence the heel is small.

6. All the ligamentous structures on the medial side of the foot and to some extent in the sole are shortened, but are histologically normal.

7. The muscles of the calf are hypoplastic, but histologically normal. As a result, the flexors of the foot and toes and the invertors of the foot are short. As growth takes place, hypoplasia commonly persists in spite of effective treatment for the deformity, so that finally the whole of the foot and calf are smaller than normal.

8. The skin shows adaptive shortening on the concave side of the curve, a fact which may present difficulties in skin closure following surgical treatment. On the convex side of the curve dimples are often present which may in extreme cases resemble scars, especially over the lateral malleolus.

The extent of these deformities is variable. In the mildest cases the deformity consists only of adduction and some rotation of the forefoot. In extreme cases the foot is so deformed that the sole rests on the medial side of the tibia (figs 276(*a*) and (*b*)).



(*a*) (*b*)

FIG. 276.—(*a*) Mild talipes equino-varus. (*b*) Severe talipes equino-varus.

Clinical Features.—The condition should be diagnosed at birth. If the deformity is severe the diagnosis is obvious, but mild deformities may be difficult to distinguish from the normal because the new-born infant tends to posture the foot in a position of equino-varus. The normal new-born foot has a greater range of dorsiflexion and eversion than the normal adult foot: it can be dorsiflexed until the dorsum touches the anterior aspect of the shin. If this is impossible, a minor degree of talipes equino-varus exists. Thus part of the examination of a new-born infant should include putting the foot through a full range of movement. If a club foot is detected, the spine and all other joints should always be examined carefully to exclude myelodysplasia and arthrogryphosis. Since the bones of the foot are at this stage largely cartilagenous, radiography is useless.

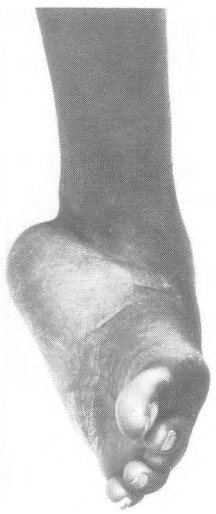


FIG. 277. — Untreated talipes equino-varus in an adult.

It must be appreciated that in the world as a whole most deliveries are not supervised by a physician and that therefore a club foot may not present until infancy or later life. By this time the deformity which was present at birth has become fixed and callosities will have developed on the skin of the foot where it bears weight. By adult life (fig. 277) secondary degenerative changes develop in the deformed joints.

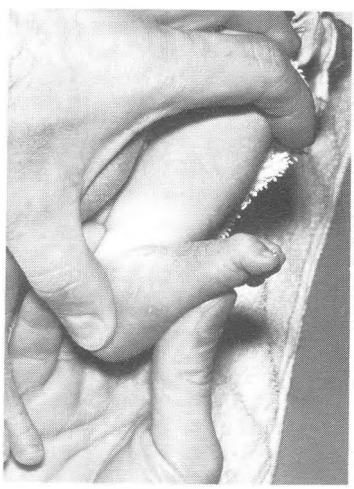


FIG. 278.—The technique of manipulation of the foot (in this case the left) in the treatment of talipes equinovarus. Note the range of dorsiflexion at this normal infant's foot. The physician's left thumb everts the heel while his right thumb dorsiflexes and everts the foot by pressing on the base of the fifth metatarsal.

Treatment

Conservative Treatment.—If the deformity is slight, so that the foot can be dorsiflexed to a little beyond the plantigrade position, the mother should be taught to manipulate her child's foot after every feed. The foot is dorsiflexed and everted as shown in fig. 278. Sufficient pressure should be exerted by the mother to blanch her own fingers and this pressure should be maintained for about 2 seconds. The pressure should be released and re-applied over a period of about 5 minutes. Minor deformity can usually be corrected on this regime but the infant should be examined at monthly intervals to ensure that the deformity does in fact steadily diminish.

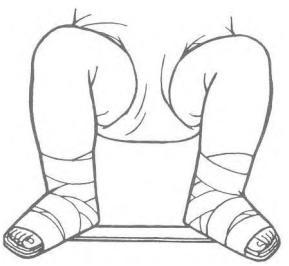
If initially the foot cannot easily be brought beyond the plantigrade position, the maximum correction obtained by manipulation must be main-

tained and gradually increased by serial splintage. The neonatal foot is too small to be treated satisfactorily in plaster-of-Paris but correction can usually be maintained by strapping. The foot may be taped to a padded aluminium splint using zinc oxide plaster by the method shown in fig. 279. The splint is removed and reapplied with a little more correction every week. By this method gradual correction of the deformity may be achieved so that, if all goes well, by 3 months the deformity can easily be corrected to beyond the plantigrade position. Splintage is then discontinued and maternal manipulation substituted. If by the age of 1 to 2 months the position of the foot is not satisfactory, plaster-of-Paris is substituted for the aluminium splint: a padded above-knee cast is applied with the knee at 90° of flexion so that the femur can be used to gain a purchase against which to abduct the foot. The cast is applied with the foot in the position of deformity and the foot is manipulated gently into a position of maximum correction as the plaster dries. The plaster is changed at 3 weekly intervals (a procedure which in an infant may require sedation) until a satisfactory correction has been achieved.

FIG. 279.—Splintage of the foot in the treatment of mild talipes equino-varus.

On this regime most club feet should have been corrected by the age of about 3 months. If they have been, splintage can be discontinued and maternal manipulation substituted.

Denis Browne splints (fig. 280) may be used throughout the day instead of maternal manipulation until the child walks and thereafter they may be used at night.



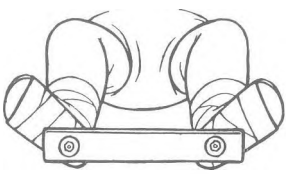


FIG. 280.—Denis Browne splint.

Treatment by Operation

A minority of club feet cannot be satisfactorily corrected by manipulation and splintage by the age of 6 weeks. In this event, the soft tissues on the medial side of the foot should be released surgically, the posterior capsule of the ankle divided, and the Achilles tendon elongated sufficiently to render the foot plantigrade.

The Operation of Medial Release and Elongation of the Achilles Tendon for Talipes Equino-Varus.—Through a curved incision posterior and distal to the medial malleolus the ligaments on the medial side of the ankle, talo-navicular and navicular-cuneiform joints are divided. The tendons of tibialis anterior, tibialis posterior, flexor hallucis longus and flexor digitorum longus are divided and elongated by Z-plasty. Division of these structures should allow the forefoot to be abducted and the foot to be everted. When this is done gaps open at the ankle, talo-navicular and navicular-cuneiform joints. Post-operatively, the cartil-

Achilles (*see footnote p. 331*).

age covering these bones proliferates rapidly so as to fill the gaps. The Achilles tendon is then elongated by Z-plasty. This may be sufficient to produce the required slight over-correction of the equinus deformity, but if it is not, the posterior capsule of the ankle joint is divided. During this operation the neurovascular bundle posterior and distal to the medial malleolus is carefully safeguarded. Closure of the skin may be difficult in the corrected position. Post-operatively the foot is splinted in a position of slight over-correction using an above-knee plaster as described above.

Treatment of 'Relapsed' Club Foot.—No matter what regime is employed for the initial treatment of club foot, there is a tendency for the condition to relapse over the early years of life and therefore children whose feet have been satisfactorily treated in infancy should be supervised until they are adult. If relapse occurs in a child less than 4 years of age, a soft tissue release as described above is usually sufficient, but in children over the age of 6 this procedure is insufficient. Children presenting with unsatisfactory feet at this age require not only medial release but also resection of bone from the lateral side of the foot. The foot may be likened for this purpose to a horse with two reins: the operation aims to pay out the medial rein (by medial release) and to pull in the lateral rein so as to turn the forefoot in a lateral direction. The lateral rein is shortened by resecting the articular surfaces of the calcaneo-cuboid joint and stapling the calcaneum to the cuboid so as to achieve calcaneo-cuboid fusion. Once calcaneo-cuboid fusion has been obtained, growth of the lateral side of the foot is slowed so that the tendency to relapse is diminished.

In adolescent children presenting with an unsatisfactory club foot, the foot may be corrected by the resection of laterally based wedges (followed by fusion of the exposed bony surfaces) from the mid-foot (to correct forefoot adduction) and the os calcis (to correct inversion of the heel).

If the condition presents in adult life, subtalar (triple) fusion with an appropriate resection of bone so as to bring the sole into a plantigrade position is the treatment of choice. Astragalectomy is the alternative if the deformity is severe.

Phocomelia of the Lower Limb may occur as in the upper limb (p. 278), and may be of varying extent up to total absence of the limb.

Absence of the Proximal Femur is rare. Radiologically the proximal third of the bone always appears to be absent, but the condition varies in severity since in some patients a cartilagenous femoral head is present although not radiologically visible. Treatment is difficult and the limb tends to become grossly short.

Absence of the Fibula is associated with forward bowing of the tibia, an equino-valgus deformity of the foot and absence of the 5th toe. The condition has some similarity to absence of the radius and its associated deformities. In both these conditions the fibrous tissue replacing the absent bone appears to grow more slowly than adjacent normal tissue so as to draw the limb into a curve concave on the side of the absent bone.

Absence of the Tibia is exceedingly rare and is inevitably followed by amputation.

Congenital Pseudarthrosis of the Tibia is probably a developmental abnormality although some are associated with neurofibromatosis and the possibility cannot be excluded that it is due to an ununited intrauterine fracture. Clinically the condition presents with marked forward angulation of the tibia at birth. Radiologically, a bony discontinuity can be seen at the junction of the middle and lower thirds of the bone and the adjacent bone is sclerotic and thinned. Treatment is by McFarland's operation in which the angulation is ignored, and a bone graft is placed posterior to the tibia extending from normal bone above to normal bone below the site of the pseudarthrosis.

Bryan Leslie McFarland, 1900–1963. Professor of Orthopædic Surgery, University of Liverpool, England.

Congenital Vertical Talus is a rare condition producing a congenitally flat foot. The long axis of the talus is vertically disposed so that the head of the bone is dislocated ventrally from the talo-navicular joint. The os calcis is in a position of equinus. As a consequence of these deformities the sole of the foot is convex downwards and the foot is rigidly flat. (fig. 281.) The condition must be distinguished from the normal flat foot of infancy in which, although the foot has no medial arch, the sole is flat rather than convex downwards and the foot is mobile. Treatment is difficult and consists of surgical re-position of the talus.

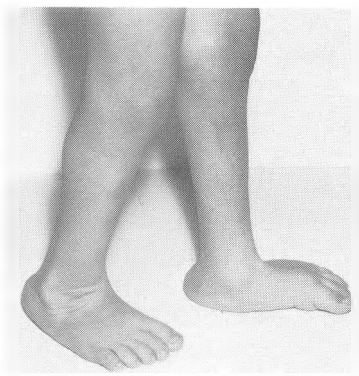


FIG. 281.—Bilateral untreated congenital vertical talus.

Abnormalities in the Toes correspond to those which may occur in the fingers (see below). In addition one toe, commonly the fifth, may override its neighbour. This is a familial condition which gives rise to no disability and which does not respond to simple conservative measures in infancy. No treatment should be given in infancy but the toe may require either correction or amputation in adult life.

THE UPPER LIMB

Phocomelia[1] is a condition in which the limb may totally or partially fail to develop. Although the condition occurs spontaneously attention has been drawn to it recently by the discovery that certain drugs, for example thalidomide, may interfere with the development of the foetus *in utero* to produce phocomelia. Such drugs are known as teratogens.

Treatment.—Under no circumstances should any portion of a phocomelic limb, for example a remnant of a finger attached directly to the trunk at the shoulder, be removed even though it is apparently useless, since such vestiges may enable the child to activate a powered prosthesis if they are capable of the slightest movement.

Minor degrees of phocomelia are represented by the *absence of one or more fingers*. The least rare anomaly of this kind is ' the lobster hand ' which has a thumb and only one finger. Function is surprisingly good in hands of this kind and no treatment is indicated.

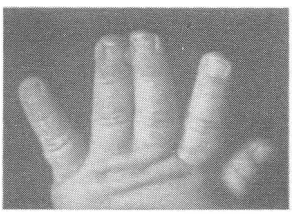


FIG. 282.—Syndactyly.

Syndactyly or Webbed Fingers may be primary, secondary or acquired (fig. 282). Primary syndactyly is often familial and in this condition not only does the skin bridge the two fingers but the bones of the fingers are fused as well. No treatment is successful. Secondary syndactyly is possibly due to amniotic adhesions and in this condition only the skin bridges the fingers. They can be separated by plastic surgical procedures (fig. 283). Acquired syndactyly may follow burns and can also be treated by plastic surgery.

Absence of the Radius is a rare anomaly as a consequence of which there is gross radial deviation of the carpus and hand (' club hand '). The thumb is often absent as well. Treatment consists of splintage to hold the deformity, but this is commonly unsuccessful. Fusion of the carpus to the ulna with an extensive release on the lateral side of the wrist may be required.

[1] Phoco (L) = a seal. i.e. Like a seal's flipper.

Radio-Ulnar Synostosis is an uncommon condition in which the radius and ulna are fused, usually in the proximal 2.5 cm of the bone. Pronation and supination are impossible. No treatment is advised.

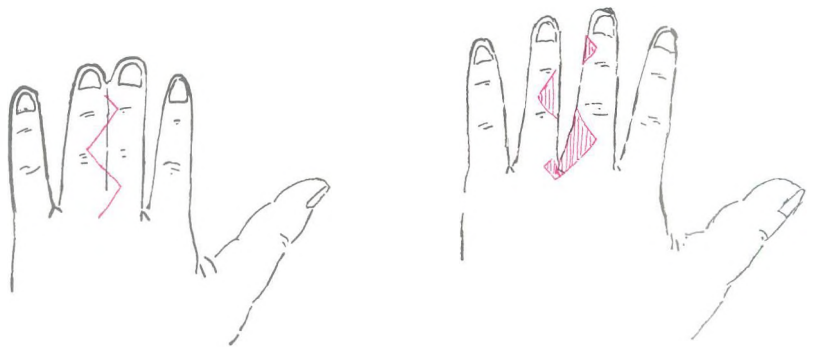


FIG. 283.—Syndactyly. The fingers are divided by means of a modified 'Z' plasty. There is insufficient skin to bridge the gap totally; the defect is therefore covered with a split thickness graft to both fingers. (Simple division with 'Z'plasty will nearly always result in scar contracture across each interphalangeal joint.)

Sprengel's Shoulder is a condition in which the whole shoulder girdle, including particularly the scapula, is higher on the affected side than on the opposite side. No treatment is available.

Webbing of the Neck occurs as part of the Klippel-Feil syndrome.

Congenital Pseudarthrosis of the clavicle is a condition similar to congenital pseudarthrosis of the tibia (p. 277). It is confined to females and only the right clavicle is affected.

Cervical rib is dealt with in Chapter 34.

THE SPINE

Fusion of cervical vertebræ occasionally occurs but is not noticeable clinically unless it is part of a more complex deformity such as the Klippel-Feil syndrome.

Hemivertebræ, especially in the dorsal region, may cause scoliosis (p. 285).

Myelodysplasia may occur in the sacral or more proximal spine.

Spondylolysis at L5 and L4 may possibly be congenital in origin although it is now widely regarded as being a fatigue fracture. The condition may progress to Spondylolisthesis (p. 497).

Sacralisation of the 5th Lumbar Vertebra, Lumbarisation of the 1st Sacral Vertebra and False Joints between the Transverse Processes of L5 and the Sacrum or Ilium are common congenital abnormalities. They may be revealed as a chance X-ray finding, sometimes in patients complaining of back pain. It is uncertain whether or not they themselves are actually responsible for back pain.

BONE DYSPLASIA

These diseases are sometimes referred to as Dysplasias, sometimes as Dystrophies. The term 'dystrophy' strictly implies that the condition is due to faulty nutrition, the term 'dysplasia', to faulty development. Since there is no evidence that any of these diseases have a nutritional basis, the term dysplasia is to be preferred. In some diseases the dysplasia appears to be

Otto Gerhard Karl Sprengel, 1852–1915. Surgeon, Grossherzogliches Krankenhaus (Grand Ducal Hospital), Brunswick, Germany. Described congenital high scapula in 1891.
Maurice Klippel, 1858–1942 and André Feil, 1884– , both of whom were neurologists in Paris. Described this disease in a joint paper in 1912.

genetic in origin but in many the cause is unknown. They may affect the skeleton generally, or may be localised to certain areas.

There are many dysplasias of bone and there is a regrettable tendency to expect the student to remember the names of all of them: very little useful purpose is served by doing so. Many have been given exotic names constructed from a random mixture of Latin and Greek, and in addition have been eponymously named: in this chapter wherever possible a simple English name is used. The majority are rare and only two or three are of orthopædic significance; the rest produce deformities for which no treatment is available or merely bizarre X-ray appearances.

Diseases of Orthopædic Significance.

Brittle Bones (Osteogenesis Imperfecta).—This is a disorder in which the bones are unduly fragile (and the sclerotics of the eyes are blue). As a consequence multiple fractures occur following trivial injuries which, because of their multiplicity, result in progressive deformity. The severity of the disease varies; some patients have only one or two fractures, others have many Children prone to a great many fractures may die before reaching adult life but if the child survives, the tendency for the bones to break seems to diminish and even to disappear when skeletal growth ceases.

Radiologically the long bones are often osteoporotic, bent and slender; the bone ends may appear large and sometimes cystic. The blood chemistry is normal. The bones are histologically normal and fractures unite uneventfully.

FIG. 284.—Exostoses of the humerus in three sisters.

Treatment.—Individual fractures should be treated on their merits. Should a long bone become unduly deformed it may be cut into several segments and each segment threaded on to an intramedullary nail to correct the deformity.

Multiple Exostoses (Diaphyseal Aclasis) (see also Osteochondroma p. 255).—This is a familial disorder more common in males (but see fig. 284). It appears to be due to a failure of bone remodelling at the metaphysis and perhaps to a failure of the control of circumferential growth at the epiphyseal plate. A variable number of exostoses develop in the metaphyseal region of the long bones. The exostoses usually stop growing at the completion of skeletal growth. They may occasion deformity and interfere with the movement of local soft tissues. Patients are otherwise unaffected by the disease, although they are often a little shorter than normal.

Five per cent of patients develop malignant changes in an exostosis. It is unfortunately impracticable to remove all a patient's exostoses in order to prevent this, but should an exostosis become painful, or should it continue to grow after the completion of skeletal growth, it should be removed *en bloc* and sent for histology.

Multiple Chondromatosis (Dyschondroplasia, Ollier's Disease) (see also Chondroma, p. 256).—This disease is rare and is not familial. Ossification of the cartilage at the epiphyseal plate is faulty and islands of cartilage remain unossified within the shaft and continue to grow. The disease tends to be unilateral and the affected limb is short. Involvement of the hand is common (fig. 260). The involved bones are often expanded by the tumour to produce deformity especially in the fingers. Radiologically the bone can be seen to be expanded by a lytic lesion in which areas of calcification may occur. Pathological fractures occur through the tumours. Malignant change develops in approximately 1% of chondromas.

Marble Bones (Osteopetrosis, Albers-Schönberg Disease).—This is a rare familial disease in which the bones appear excessively dense and structureless on X-ray because the medullary canal is for practical purposes absent. The spine is also affected. The bone is hard but at the same time brittle, and accordingly pathological fractures are not uncommon. Management of the fractures may be difficult since internal fixation is almost impossible.

Diseases of Little Orthopædic Significance

These diseases produce abnormal radiological appearances and often deformity. They do not however call for orthopædic treatment.

Achondroplasia.—This is a familial disease in which the growth of the long bones and of the antero-posterior axis of the skull are defective. Accordingly the patient is dwarfed and has a typical facial appearance. The trunk is less obviously shortened than are the limbs (fig. 285).

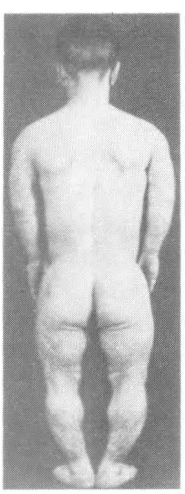


FIG. 285.—Achondroplasia.

Chondro-Osteodystrophy (Morquio-Brailsford's Disease).—This is a rare disease produced by a recessive gene so that consanguinity is important in its ætiology. The disease gives rise to dwarfism. The vertebral bodies may show typical 'tonguing' in the X-ray and the pelvis may be tri-radiate.

Gargoylism (Hurler's Disease).—In this condition a skeletal disorder is combined with a disturbance of lipid metabolism. Dwarfism and a typical facial appearance develop.

Epiphyseal Dysplasias.—There are several forms of epiphyseal dyplasia. Dyplasia epiphysialis multiplex is the least rare and affects all the epiphyses so that the patient is stunted because the limbs are short. The face and skull appear normal. The epiphyses look ill-formed, irregular and mottled radiologically. Dyplasia epiphysialis punctata is a variant of multiplex in which the mottling is more pronounced, but the shape of the epiphysis is more normal. Dyplasia epiphysialis hemimelica is a variant in which only the medial or lateral half of the knee epiphyses, the ankle epiphyses and the talus of one leg are affected.

Tibia Vara (Blount's Disease).—This is probably a mild version of dysplasia epiphysialis hemimelica in which only the proximal tibial epiphyses are involved. The disorder is particularly common in the West Indies and may be treated by early tibial osteotomy.

Cleido-Cranial Dysostosis (Anosteoplasia) (fig. 286).—In this condition there is faulty development of membrane bones, particularly of the clavicle and the skull. The clavicles are almost completely absent so that the patient can bring his shoulders together in front of the chest.

Louis Xavier Édouard Léopold Ollier, 1830–1900. Professor of Surgery, Lyons, France. Described dyschondroplasia in 1899.
Heinrich Ernst Albers-Schönberg, 1865–1921. Professor of Roentgenology, Hamburg, Germany. Described osteopetrosis in 1904
Luis Morquio, 1867–1935. Professor of Medicine, Montevideo, Uruguay.
James Frederick Brailsford, 1888–1961. Radiologist, Queen Elizabeth Hospital, Birmingham, England.
Morquio and Brailsford reported this condition independently in 1929.
Gertrud Hurler. Pædiatrician, Munich, Germany. Described this disease in 1919.
Walter Putman Blount, Contemporary. Professor of Orthopædic Surgery, Marquette University Milwaukee, Wisconsin, U.S.A. Described this condition in 1937.

Nail-Patella Syndrome (Osteo-Onychodysplasia).—This is a curious familial disorder in which the nails are hypoplastic or absent and the patellæ small. The radial head may sublux laterally and bony excrescencies may develop on the lateral aspect of the ilium.

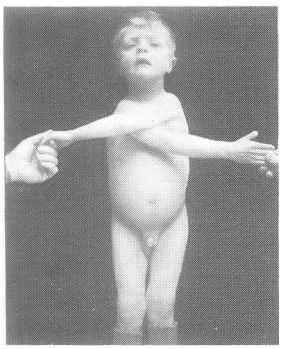


FIG. 286.—Cleido-cranial-dysostosis.

Progressive Diaphyseal Dysplasia (Engelmann's Disease), *Spider Finger* (Arachnodactyly, Marfan's Syndrome), *Acrocephalo-syndactyly* (Apert's Syndrome), *Candle Bones* (Melorheostosis, Leri's Disease), *Striped Bones* (Osteopathia Striata), and *Spotted Bones* (Osteopoikilosis) all produce abnormal radiological appearances of the bones and in some cases deformity.

MUSCLE DYSPLASIA

Arthrogryphosis Multiplex Congenita.—This is a rare condition in which there is extensive congenital failure of muscular development. The absent muscles are replaced by fibrous tissue. As a consequence of the mixture of paralysis and contracture, multiple deformities develop in the joints. The skin overlying the joints is strikingly creaseless. The most commonly affected joints are those of the feet and hands in both of which clubbing commonly occurs. No treatment is available for the underlying pathology. The deformities are hard to correct, and even if corrected, function tends to be poor. Excision of the talus may be required to render the foot plantigrade.

'Congenital' Torticollis is not a true congenital abnormality: it is almost certainly attributable to infarction of the central portion of one sternomastoid muscle at the time of birth. At birth the central part of the muscle may be found to be swollen: a 'sternomastoid tumour' (fig. 287). The infarcted muscle is gradually replaced by fibrous tissue which contracts. As it does so the neck is turned so that the face is directed away from the side of the lesion (as if the affected sternomastoid muscle were contracting). This state of affairs is exactly analogous to the development of Volkmann's ischæmic contracture in the limbs and indeed the condition should probably be regarded as an example of ischæmic contracture. Persistent torticollis often results in facial asymmetry.

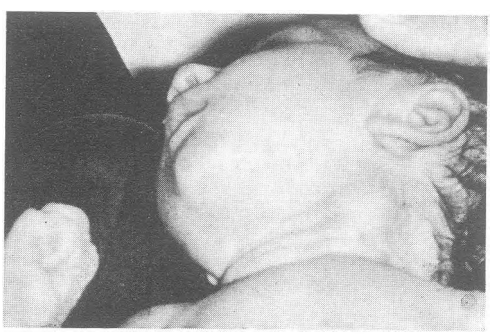


FIG. 287.—Left sternomastoid 'tumour' in a newborn infant.

Treatment.—If a sternomastoid 'tumour' is noticed at birth, the infant's head should be gently manipulated through a full range of movement every day in the hope that such a range can be maintained whilst the muscle is being replaced by fibrous tissue. More commonly the condition is not noticed until torticollis has developed. At this time the condition is treated by division of the sternomastoid muscle at its distal, or proximal and distal, attachment. Care should be taken at the proximal end of the muscle to avoid damage to the spinal accessory nerve.

Guido Engelmann. Orthopædic Surgeon, Vienna. Described this disease in 1929.
Bernard Jean Antonin Marfan, 1858–1942. Physician, Hospital des Enfants-Malades, Paris. Described this syndrome in 1896.
Eugène Apert, 1868–1940. Physician, Hospital des Enfants-Malades, Paris. Described this syndrome in 1923.
André Léri, 1875–1930. Physician Paris. Described this syndrome in 1921.
Richard von Volkmann, 1830–1889. *(See p. 343.)*

CHAPTER 18

DISORDERS OF THE GROWING SKELETON

CERTAIN disorders affect the epiphyseal plate or the epiphysis itself and hence are peculiar to the growing skeleton. Disorders of the epiphyseal plate may manifest themselves functionally, as *disturbances of growth*, or structurally because the epiphyseal plate is a point of mechanical weakness in a long bone, so that *displacement of the epiphysis* may occur. Certain other disorders of the epiphysis affect both its bony and cartilaginous portions and hence are termed ' *Osteochondritis* '.

DISTURBANCES OF GROWTH

The limbs and spine normally remain straight as they grow because growth on the two sides of the limb or spine proceeds at an equal rate. If the growth rate on one side exceeds the growth rate on the other, the structure will become curved, being convex on the side of more rapid growth. Unequal growth in the leg produces bow-leg or knock-knee, and in the spine is possibly responsible for idiopathic scoliosis. Although it is easy to understand how an imbalance in relative growth rates could produce angulation of a limb in this way, the production of torsional deformities is not so readily explicable. Nevertheless, disorders of growth may not only produce angulation of the limbs or spine, they may also result in torsion. In the legs, torsional disturbances of growth produce in-toeing or splay-foot; in the spine, combined with angular disturbances, they produce scoliosis.

Angular and Torsional Deformities of the Leg

Perfectly normal infants may be noticed by their parents to have some degree of knock-knee or bow-leg (fig. 288), in-toeing or splay-foot when they first stand and begin to walk. These ' deformities ' often worry the parents, although they do not upset the child. Typically, medical advice is sought when the child is 3 to 4 years of age. Of the two deformities, knock-knee is slightly the more common.

Both *knock-knee* and *bow-leg* occur as part of the normal pattern of growth and should only be viewed as deformities if they are severe and progressive. In countries where malnutrition is common, knock-knee is a frequent complication of rickets, but in Great Britain Vitamin D deficiency rickets is almost non-existent, save in immigrants. Rickets occurs (rarely) as a consequence of a metabolic disorder or malabsorption syndrome (Chap. 49). Epiphyseal dysplasia may cause bow legs (tibia vara) or less commonly knock-knee. Blount's disease, a variety of epiphyseal dysplasia, common in the West Indies, affects only the postero-medial segment of the proximal tibial epiphysis and thus causes bow legs but no other deformity.

Walter Putnam Blount, Contemporary. Orthopaedic Surgeon, Milwaukee, Wisconsin, U.S.A.

The degree of knock-knee can be measured by noting the distance between the medial malleoli. Bow-leg can be measured by noting the distance between the medial femoral condyles. The severity of both ' deformities ' is prone to increase when the child stands, so that a measurement should be made both non-weight bearing and weight bearing and should be related to the length of the leg.

In-toeing is somewhat more common than *splay-foot* and is due to torsion of the bones of the legs. If the child is placed supine on the examination couch with the patellæ pointing directly forwards, the degree of tibial torsion is revealed by the position taken up by the foot (fig. 289). Most of

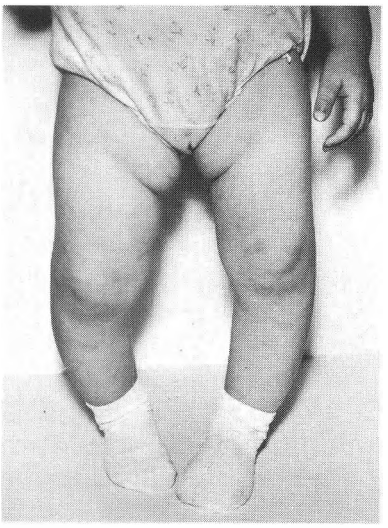


FIG. 288.—Infantile bow-leg. This appearance is within the limits of normal at this age.

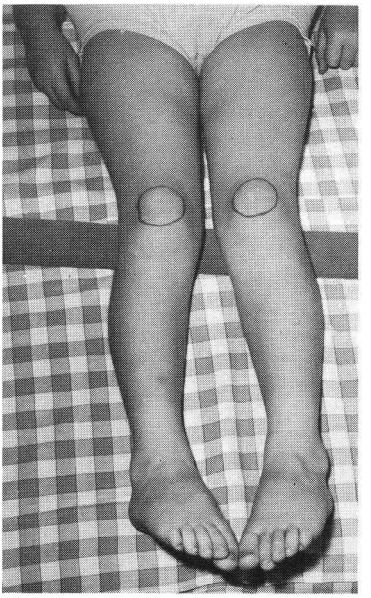


FIG. 289.—Internal tibial torsion (the patellæ are ringed) causing in-toeing, and associated knock-knee.

the torsion is usually in the tibia but some may be in the femur. Here it affects particularly the angle made in the coronal plane between the femoral neck and the back of the femoral condyles (the ' angle of femoral anteversion ').

Treatment.—No form of splintage, or adjustment to the footwear, has any effect upon any of these deformities. No treatment is indicated until the age of 6, since the deformity is usually self-correcting before that age. The parents should therefore be reassured and the child left to live a normal life. If significant deformity persists after the age of 7, the legs can be straightened by osteotomy or by slowing growth on the convex side of the curve. Growth can be slowed either by placing staples across the epiphyseal plates at the knee on the convex side of the limb or by excising this portion of the epiphyseal plate and fusing the epiphysis to the metaphysis with a bone block. The latter operation (epiphyseodesis) has the advantage over stapling

of being more reliable, but the disadvantage of being irreversible. These operations must be carefully timed in relation to the child's growth potential so as to avoid over-correction and under-correction.

Torsional deformities very rarely require correction, but in girls severe in-toeing, as well as being a slight functional disability, may represent a significant cosmetic defect. Should it be severe enough to merit treatment, the only treatment available is to divide the proximal shafts of the femora and de-rotate the bones at the osteotomy site. If the deformity really merits operations of this magnitude, they should be carried out when the child is about 11 years of age.

Scoliosis

Scoliosis is defined as a lateral curvature of the spine, but the lateral curvature is always complicated by a rotational deformity which, in the thoracic region, is transmitted to the ribs and produces asymmetry of the thorax (fig. 290 and 291). Scoliosis is classified according to its ætiology.

1. Idiopathic Scoliosis.—This is the most common form of scoliosis, and next to the paralytic form is responsible for the most severe curves, usually in the dorsal spine.

The fundamental cause of idiopathic scoliosis is, as the name implies, unknown. It has been attributed to paralysis of the intrinsic muscles of the spine caused by subclinical poliomyelitis, but its incidence appears to have been unaffected by the control of poliomyelitis, and girls are more commonly affected than boys (whereas poliomyelitis affects both sexes equally). The absence of paralysis in any other muscle group is also against the presence of a paralysing disease. It is, in fact, the complete normality of the rest of the body which distinguishes this condition from paralytic and other forms of scoliosis. A genetic element is almost certainly involved since the disease has a slightly increased familial incidence. Whatever the ultimate cause of the deformity, the proximate cause is a disturbance of vertebral growth.

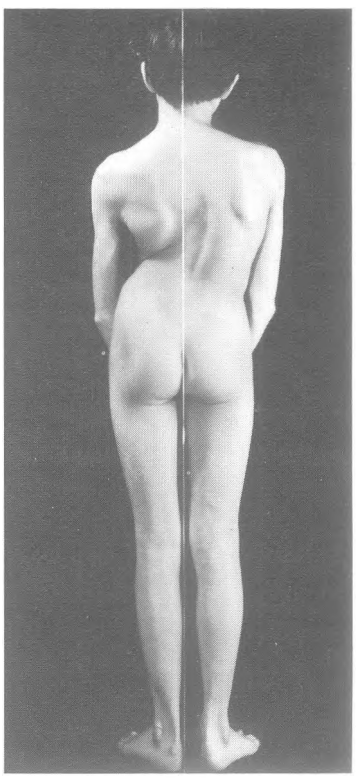


FIG. 290.—Scoliosis: the appearance of the back.

Clinical Features.—In order that the patient may stand upright, the primary curve in the spine is balanced by secondary curves in the opposite direction above and below it. These compensatory curves bring the shoulders over the pelvis so that in mild primary curves, no gross abnormality may be

visible when the patient is dressed. On the other hand, a severe scoliosis is very disfiguring for two reasons: firstly there is a loss of as much as 15 cm. of height as the spine becomes 'concertinaed', and secondly a rib hump develops which gives the appearance of a hunchback. Thus the adult picture in a severe case is that of a hunchbacked dwarf. The curves in the spine in a child are to some extent fixed (structural) and to some extent mobile. The latter are caused by the spine collapsing like a concertina under the influence of gravity: if the child is picked up by the head or the axillæ, the mobile part of the curve straightens so that only the structural component remains.

The rib hump is characteristic of idiopathic scoliosis and early in the disease is best demonstrated by standing behind the unclothed patient and asking her to touch her toes. Even in the absence of any very marked clinical deviation of the spine, this test will show the asymmetry of the chest (fig. 291). The rib hump is produced by rotation of the dorsal vertebræ (when they are involved in the curve) and, with them, of the ribs. The vertebræ rotate in such a way that the tips of the spinous processes move towards the concave side of the curve and hence, clinically, diminish its apparent severity. On the convex side of the curve, vertebral rotation displaces the ribs posteriorly to form the rib hump. Thus the appearance of the back is a poor guide to the severity of the spinal curve: rotation of the spine diminishes the appearance

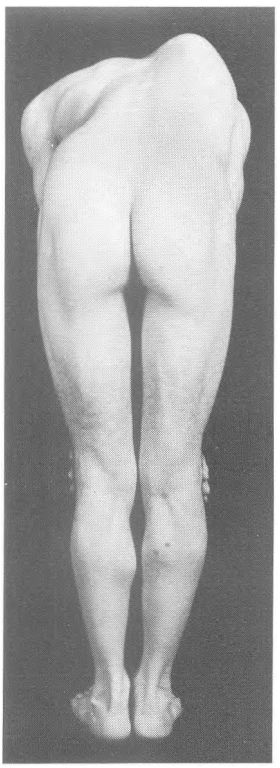


FIG. 291.—Scoliosis showing 'rib hump' revealed in flexion.

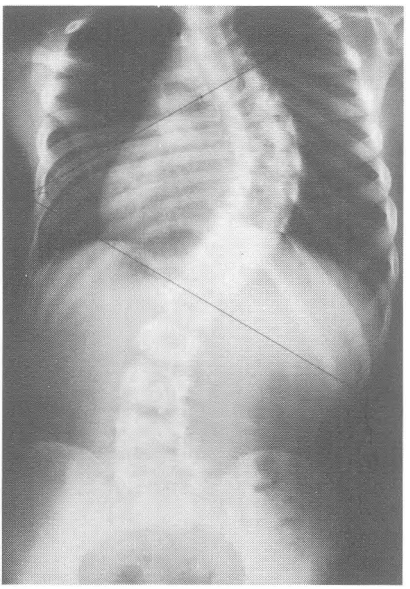


FIG. 292.—The radiological appearance of the spine in scoliosis. The angle of the curve is measured by the angle subtended by the upper surface of the first and the lower surface of the last vertebra in the primary curve.

of the deformity in so far as it brings the spinous processes nearer the midline, but increases it in so far as it produces a rib hump. The only way that the spinal curve can be assessed accurately is by the measurement of X-rays (fig. 292).

Because of the deformity of the ribs and dorsal spine, a dorsal scoliosis reduces the vital capacity, often seriously so. As a consequence the patient may eventually die of pulmonary infection. Alternatively, secondary pulmonary fibrosis may lead to Cor Pulmonale and right sided heart failure.

Clinical Management.—Idiopathic scoliosis may appear in infancy, in childhood, or during adolescence. In infancy it may resolve spontaneously but a few curves become progressive. All curves, once established as structural and irreversible, may deteriorate rapidly, particularly during periods of skeletal growth. After the completion of spinal growth (which coincides with the fusion of the iliac crest apophyses), deterioration may continue at a slower rate, or, more commonly, will come to a halt. Once advanced structural deformity has developed, nothing can be done to restore the spine to normal. Thus untreated, the condition may rapidly progress until the patient is an irrevocably dwarfed hunchback. Early diagnosis and, if necessary, treatment are therefore essential.

The rate of progression of deformity is hard to predict when the patient is first seen, so that the patient must be examined frequently once the deformity has been noticed. Comparable anteroposterior X-rays of the entire spine should be taken every 3 months during the first year and thereafter at longer intervals until skeletal growth is complete. The curves are measured accurately on these X-rays (fig. 292); it is useless to depend upon clinical impressions of the appearance of the spine. Without careful radiological supervision a deformity may develop, within a year of the curve first being noticed, which is too severe for full correction.

Treatment.—In most patients who develop a severe curve, a rapid deterioration takes place in the 2 or 3 years prior to skeletal maturity. Mild curves under 20° may be treated *expectantly*, the spine being checked regularly by comparable anteroposterior radiographs, taken in the erect position, until growth is complete.

Conservative or non-operative treatment by the Milwaukee brace[1] and exercises may be successful in preventing deterioration or even producing improvement in curves which advance beyond 20° but do not exceed 40° to 45°. The brace may be used in more severe deformity, particularly in young children, as a holding device until the child reaches the age of ten to twelve years, when definitive surgical treatment can be carried out.

Operative treatment is reserved for those patients who deteriorate during conservative treatment or who have severe curves (over 50°) when first seen. It is preferable to defer surgery until near skeletal maturity because of the unpredictable effects of further growth after spinal fusion. Fusion of the spine is also more difficult to achieve in the young child. Nevertheless

[1]The Milwaukee brace was introduced in 1946 by Walter Putnam Blount and his associates at Milwaukee, Wisconsin, U.S.A.

correction and fusion in a young child is sometimes carried out where deterioration cannot be controlled in any other way.

The aim of all surgery for scoliosis is correction and fusion of the primary curve. Correction may be pre-operative or it may be part of the surgical procedure.

Techniques of Pre-operative Correction

1. *Risser Turnbuckle Cast.*—This is the most widely used method of obtaining pre-operative correction. A plaster cast is applied, including the head and one thigh, with the patient's spine flexed laterally to produce the maximum correction of the mobile portion of the curve which can be tolerated. The cast is heavily padded with sorbo rubber at the points where pressure is encountered. When the cast is dry it is cut at the level of the apex of the primary curve and angulated so as to produce progressive correction. The new position is held by means of metal turnbuckles incorporated into the plaster. In this way a slow correction can be obtained from week to week

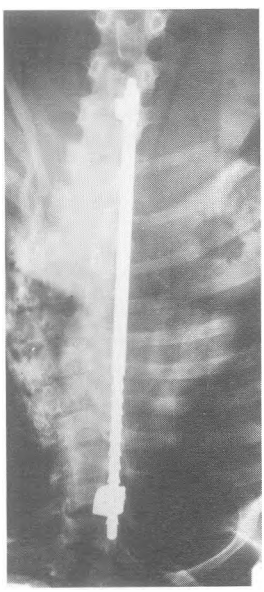


FIG. 293.—A single Harrington rod used to extend and internally fix the concave side of the curve.

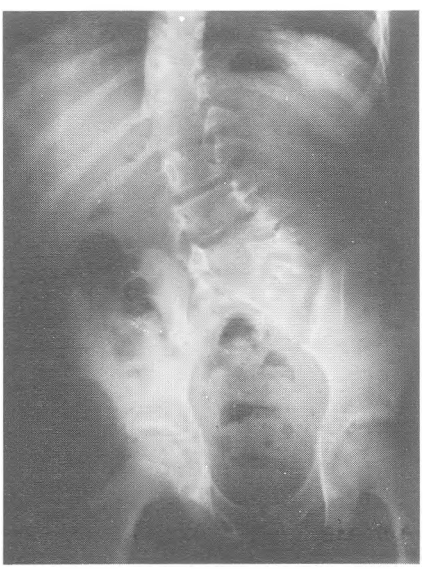


FIG. 294.—Congenital lumbar scoliosis due to a hemivertebra at L.3 fused to L.4. Note associated spina bifida, in this case occult.

and checked by X-ray. When the primary curve has been sufficiently corrected from the cosmetic and functional standpoints (which may not be the point of complete correction in an originally severe curve), a window is cut in the back of the plaster over the primary curve and through this the spine is fused posteriorly. The fusion must extend to include the vertebræ immediately above and below the primary curve. The bone used is generally taken as small chips from the patient's own spine and iliac crests, but the volume can be increased by adding banked bone. If the primary curve is very long, the operation may be performed in stages. After operation, the patient remains in the Risser jacket for 6 months whilst the fusion consolidates. The following year is spent in a carefully moulded corset or Milwaukee brace so that the soft new graft is not subjected to severe bending forces.

2. *Risser Localiser Cast.*—This is applied on a special frame. The patient lies on a narrow strip of canvas and correction is obtained by a combination of longitudinal traction and the localiser which applies pressure over the rib hump to correct the

Joseph Charles Risser, Contemporary. Professor of Orthopædic Surgery, Loma Linda University, Los Angeles, California, U.S.A.

angular and rotation deformities. A well fitting plaster cast is applied in the corrected position. A series of such casts may be used to obtain gradual correction.

3. *Cotrel Dynamic Traction.*—Mobilization of a rigid curve and some correction may be obtained by this method. The pelvis is anchored to the foot of the bed by a leather harness. A leather head halter is connected via a pulley at the head of the bed to a pair of stirrups through which the child applies longitudinal traction by extending the legs.

4. *Skeletal Traction* may be applied to the skull by means of a 'halo'. This may be combined with skeletal traction to the femora or pelvis. Great forces can be applied by this means and the method requires most careful supervision to avoid neurological and other complications.

Operative Correction

Harrington Instrumentation is the most widely used method of operative correction of scoliosis. After exposure and stripping of the posterior elements of the spine, hooks are applied to the vertebræ beyond the end vertebræ on the concave side of the primary curve. These hooks are connected by a rod which has a ratchet device so that gradual distraction can be applied to produce correction. A compression rod and hooks are sometimes used on the convex side of the curve.

Dwyer's Procedure.—Through an anterior approach the vertebral bodies are exposed on the convex side. A wedge of disc and body is removed at each level of the primary curve. The gaps so created are closed by means of a series of screws in the vertebral bodies. A tension cable passes through the heads of the screws. As each level is corrected the screw head is crimped on to the cable.

Technique of Fusion.—Correction, whether pre-operative or intra-operative, is always combined with spinal fusion. This may be an anterior fusion between the vertebral bodies, or a posterior fusion of the laminæ and transverse processes. Or a combination of the two is sometimes used. Additional bone, preferably autogenous bone from the ilium, is commonly employed.

Standard posterior fusion consists of meticulous stripping of the primary curve from the midline to the tips of the transverse processes. This is followed by excision of the apophyseal joints and decortication of the laminæ and transverse processes. Additional bone is packed into the bed so prepared and the wound is closed.

Following spinal fusion a corrective plaster is maintained until the graft is mature and solid. This may take about a year.

2. Congenital scoliosis is associated with congenital spinal anomalies, such as hemivertebra (fig. 294), errors of segmentation, spina bifida, diastematomyelia etc. Associated anomalies are common particularly of the spinal cord and kidneys. A myelogram and intravenous pyelogram are therefore indicated if surgery is contemplated.

3. Paralytic Scoliosis (Chapter 21).

4. Postural Scoliosis

This affects children of school age and is characterised by being a simple C curve without rotation of the vertebræ. The deformity is fully mobile and correctable. Formerly quite common, the condition is now almost non-existent, possibly because of the improvement in the general health of the population, the provision of physical

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Paul R. Harrington, Contemporary. Associate Professor of Orthopædic Surgery, Baylor University, Houston, Texas, U.S.A.

education in schools and the avoidance of bad posture when sitting at school desks. Treatment is by gymnastics and lessons in deportment.

5. Pulmonary Scoliosis

Pulmonary disease may produce scoliosis if disease of one lung results in contraction of the chest wall on that side. In the past the most severe deformities followed the treatment of empyema by prolonged drainage through tubes in the chest wall, but the modern treatment of chest disease has made this one of the rarer causes of scoliosis.

DISPLACEMENT OF EPIPHYSES

Slipped Femoral Epiphysis

Displacement of the proximal femoral epiphysis may occur in the face of the loads imposed by normal activities—possibly because the normal loads applied to this epiphysis are high, because the epiphyseal plate may not be disposed at right angles to the line of action of the resultant force applied to it, or because the epiphyseal plate is not mechanically normal. The latter possibility is entirely hypothetical. No other epiphysis is liable to displacement in this way. Rarely the normal epiphysis may be displaced by significant violence: this is really a fracture of the femoral neck which happens to involve the epiphyseal plate.

The condition affects children between the ages of 10 and 18 years. It is particularly common in boys in a ratio of 3:2 and in over-weight 'hypogonadal' children. It is bilateral in about 20% of patients, the two sides being affected at about the same time. Dunn has observed that, when unilateral, the side of the dominant hand is usually affected first. Relative to the neck, the epiphysis slips downwards and posteriorly. This is the equivalent of the femoral shaft and neck moving into adduction and external rotation in relation to the head.

FIG. 295.—Slipped femoral epiphysis.

FIG. 296.—Internal fixation of a slipped femoral epiphysis with 3 Moore's pins.

Two varieties of slipped epiphysis are encountered: 1. acute (i.e. sudden) displacement and 2. chronic (i.e. slow) displacement.

1. Acute Slipped Epiphysis.—Careful questioning will often reveal that

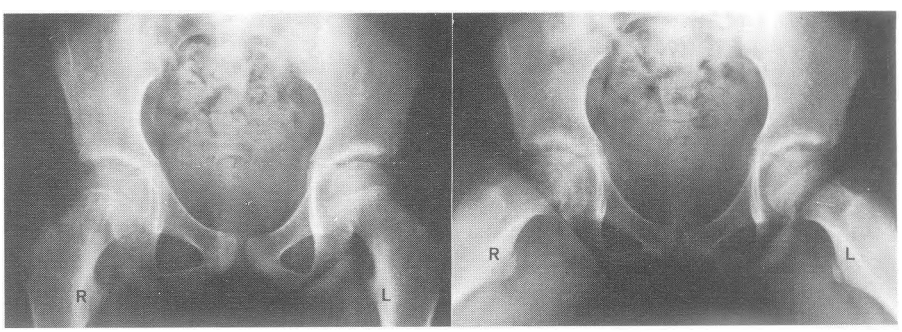
Denis Michael Dunn, Contemporary. Orthopædic Surgeon, Essex County Hospital, Colchester, Essex, England. Austin Tally Moore, 1899–1963. Orthopædic Surgeon, Columbia Hospital and the Moore Clinic (which he founded in 1939), Columbia, South Carolina, U.S.A.

the acute slip is really a final episode in what has been a slow slip, but the symptoms prior to the acute slip can be so slight as to pass without notice. The acute slip often follows an injury of some magnitude, such as a fall off a bicycle or at football, after which the patient is unable to move the hip and experiences severe pain. The clinical signs are identical with those of a subcapital fracture in an adult, and indeed this condition is a subcapital ' fracture ' through the epiphyseal plate. X-rays show complete displacement of the epiphysis (fig. 295).

Treatment is similar to that of a subcapital fracture of the femur in a young adult: the epiphysis should be reduced under anæsthesia without undue delay (by internal rotation and abduction) and fixed internally with 3 Moore's pins (fig. 296) under X-ray control.

2. Chronic Slipped Epiphysis.—In this condition the child complains of pain in the hip and of a limp, but is able to walk. On first examination muscle spasm usually causes limitation of movement in all directions, suggesting an inflammatory process in the hip (for example early tuberculosis). Rest in bed for a day or two relieves the pain and spasm: isolated limitation of internal rotation and abduction, and slight apparent shortening of the leg, are then demonstrable.

X-rays will reveal the displacement of the epiphysis (figs. 295 and 297) although if only a small slip has occurred it is very difficult to detect in an anterio-posterior view. In a lateral view of the hip, however, the posterior displacement of the epiphysis is obvious. Therefore a *lateral view of the hip should always be obtained if a slipped femoral epiphysis is suspected clinically.*



(*a*) (*b*)

FIG. 297.—A minimal slip of the right femoral epiphysis. (*a*) Apparently normal antero-posterior X-ray. (*b*) Minimal slip revealed on a lateral X-ray of the femoral neck.

In the absence of treatment there is a period of disability during which the slip progresses. This may be followed by spontaneous remission if healing occurs at the epiphyseal plate so that the head of the femur ' unites ' with the neck in the slipped position. The patient then recovers almost full function, though with the permanent physical signs of limited abduction (due to the adduction element in the deformity) and limited, or absent, internal rotation (due to the external rotation element in the deformity). Later in life,

osteoarthrosis may be expected to develop. Alternatively the period of slow, progressive slipping may culminate in an acute slip.

Treatment.—Attempts to reduce a chronic slipped epiphysis are difficult or impossible, and the attempt may damage the blood supply to the head. If the slip is slight, the position should be accepted and treatment should aim only to prevent further displacement by internal fixation of the epiphysis as for an acute slip. Because this condition is commonly bilateral, the other hip should be watched for any sign of a slip or should be pinned prophylactically.

Once internal fixation has been obtained, the child should remain in bed for 3 weeks, use crutches for 6 weeks, and then walk normally. Contact sports and physical education should be avoided for 1 year. If the child is overweight, he should diet.

If gross slipping has occurred by the time the patient is first seen, an osteotomy, either in the neck or trochanteric region of the femur, should be performed to correct the external rotation and adduction deformity. These operations are technically difficult and osteotomies of the neck run the risk of interfering with the blood supply to the epiphysis.

Displacement in Response to Significant Violence.—This may occur at any epiphysis: the condition is really a fracture through or adjacent to a previously normal epiphyseal plate and is described in Chapters 24 and 25.

Cartilage Necrosis (*Waldenstrom's Disease*).—Especially in Negro children, the articular cartilage in the hip may die after the epiphysis has slipped. The aetiology of this condition is unknown. The radiological joint space becomes reduced and the hip loses movement, sometimes completely. No specific treatment is available. Care should be taken to see that the hip, if it does become stiff, does so in the position of function (see Table, p. 224). Movement sometimes recovers to some extent spontaneously after a long period of stiffness.

Subclinical Displacement

It has recently been suggested by Murray that children, especially athletic boys, may sustain subclinical slips which are responsible in later life for many examples of so-called primary osteoarthrosis of the hip.

OSTEOCHONDRITIS

It is a confusing misfortune for the medical student that this one term is used to describe widely differing pathological processes all of which affect epiphyses. The exact nature of the pathological process occurring in the diseases which are given the title 'Osteochondritis' is uncertain because adequate human histological material has rarely been available. It is only possible here to state that three pathological processes appear to be at work: infarction of epiphyses, traction injuries to epiphyses, and transchondral fractures in epiphyses.

Infarction of Epiphyses

This disorder affects particularly the proximal femoral epiphysis where it is known as Legg-Calvé-Perthes' disease, the distal epiphysis of the second

Johan Henning Waldenstrom, 1877– . Swedish Surgeon.
Ronald Ormiston Murray, Contemporary. Radiologist, Royal National Orthopædic Hospital, London.
Arthur Thornton Legg, 1874–1939. Orthopædic Surgeon, The Children's Hospital, Boston, Massachusetts, U.S.A.
Jacques Calvé, 1875–1954. Orthopædic Surgeon, La Fondation Franco-Américaine, Berck-Plage, France.
Georg Clemens Perthes, 1869–1927. Professor of Surgery, Tübingen, Germany. Legg, Calvé and Perthes all described osteochondritis of the head of the femur independently in 1910.

metatarsal bone, where it is known as Freiburg's disease, the navicular where the whole bone may be regarded as the epiphysis and where it is known as Köhler's disease, and the lunate where it is known as Kienböck's disease. The last three examples may well be due to trauma. Of these, Perthes' disease is the most common and has the most serious consequences for the patient.

Perthes' Disease

Pathology.—Since this disease is not fatal and there is no therapeutic indication for removal of the femoral head in patients suffering from the disease, there have been very few observations of the morbid anatomy of this disease in man. On the basis of the histology obtained from epiphyses infarcted experimentally in animals, the little human histological material that is available, and the radiological appearances of the epiphysis in experimental animals and in man, it may be speculated that the sequence of events in Perthes' disease is as follows:—

The presence of an epiphyseal plate results in a peculiarity of the blood supply to the bone of the epiphysis, for blood vessels do not cross the epiphyseal plate. Thus the blood supply to the epiphysis is derived entirely from vessels passing around the periphery of the plate and along the ligamentum teres. (In the adult on the other hand blood not only reaches the head of the femur by the original route, but also by a route within the bone itself across the fused epiphyseal plate.) Since the epiphysis is entirely intra-articular all the epiphyseal vessels run beneath the synovial membrane before entering the epiphysis itself, and thus the epiphysis could be infarcted if, for example, fluid collected in the joint under sufficient pressure to occlude the vessels. It has been suggested that this is in fact the mechanism of infarction in Perthes' disease. (Why certain small bones such as the navicular may also infarct, is uncertain.)

That the spontaneously occurring variety of the disease is caused by infarction of the epiphysis is supported by the fact that similar changes in the femoral head may follow events which would clearly be expected to interrupt its blood supply such as traumatic dislocation of the hip and fractures of the femoral neck in children, immobilisation of the hip in positions in which the capsule is twisted around the femoral neck (as for example occurs in the treatment of congenital dislocation of the hip by immobilsation of the hip in positions of extreme internal or external rotation), and tense hæmarthroses in the hip in hæmophiliacs.

When the blood supply to the epiphysis has been interrupted the bone dies but the cartilage, nourished from the synovial fluid, survives and proliferates. Thus the bony epiphysis on the infarcted side appears smaller (because it fails to grow) and the radiological joint space appears larger (because the cartilage grows, and possibly because an effusion is present in the joint).

Although the cartilage survives for some weeks upon synovial nutrition, some chondrocytes eventually die, especially in the basal layers, perhaps because proliferation of the overlying cartilage makes it impossible for them to receive an adequate supply of nutrients by diffusion from the synovial surface. Dead cartilage (1) is probably softer than normal so that it may deform under normal loads, and (2) may not ossify.

Granulation tissue invades the infarcted epiphysis by overgrowing the margins of the epiphyseal plate. The dead bone is partly resorbed and partly entombed in new bone deposited on a scaffold of the old; a process known as creeping substitution. Before it is replaced, the dead bone may fail mechanically (i.e. may fracture) especially if the cartilage is soft, perhaps because it fatigues. Thus the bony nucleus of the epiphysis, like the cartilaginous portion, may deform in response to normal loads.

Once the trabeculæ become crushed, creeping substitution can no longer occur and the necrotic area must be removed or replaced by a fibrocartilaginous material having some of the appearances of immature cartilaginous fracture callus. This material is soft and biologically 'plastic' so that it may be moulded. As healing occurs it is replaced by woven bone by a process of membraneous ossification.

The epiphyseal plate is abnormal in all cases with loss of normal architecture and

Albert Henry Freiberg, 1868–1940.
Alban Köhler, 1874–1947 *(see p. 296).*
Robert Kienböck, 1871–1953. Professor of Radiology, Vienna.

also a failure of normal ossification producing some widening of the growth plate. This will result in a growth disturbance which will further contribute to the deformity of the femoral head.

As the bone is revascularised the deeper layers of the overlying cartilage are progressively, but patchily, converted into bone by endochondral ossification. The combination of creeping substitution of the bony nucleus and patchy endochondral ossification in the cartilage makes the epiphysis appear radiologically dense, fragmented, and deformed. Although the bony nucleus of the epiphysis always appears to be deformed radiologically, the femoral head itself may not be so: a fact which can be observed experimentally with the naked eye or clinically by arthrography.

Clinical Features.—Boys are affected more commonly than girls in the ratio of 4 to 1. The reason for this is uncertain. The condition usually presents between 3 and 10 years. 18 per cent of cases are bilateral. The child presents with pain often felt in the anterior thigh and knee, and is noticed by the parents to limp particularly in the evening and after exercise. There may be a history of injury in the past but there is commonly a silent period between this incident and the onset of symptoms.

Clinical examination will reveal apparent shortening of the limb, wasting of the quadriceps and restriction of hip movements by muscle spasm with pain at the extremes of movement. This muscle spasm will commonly resolve in a few days of bed rest with skin traction. Routine examination should exclude the presence of inguinal hernia and genito-urinary anomalies as there is known to be an association between these conditions and Perthes' disease. All hæmatological investigations are normal.

FIG. 298.—Radiological appearance of Perthes' disease of one year's duration.

Radiological Features. — This process passes through a series of stages. At first the ossific nucleus appears normal save that it is smaller than that on the other side. The radiological joint space however becomes greater than the opposite side. In part this is due to an effusion and in part to the continuing growth of the cartilaginous portion of the femoral head. In the second stage part or all of the bony epiphysis becomes crushed or fragmented producing a broad and flattened femoral head in many cases (fig. 298). Catterall has shown that the degree of this involvement varies and that in the long term the prognosis varies with the degree of this involvement. In the third phase the crushed area is reabsorbed and finally replaced by bone. This is followed by a long phase of remodelling continuing throughout the remainder of growth.

Anthony Catterall, Contemporary. Orthopædic Surgeon, Royal National Orthopædic Hospital, London.

At the stage of fragmentation it is possible to predict radiologically those cases in which severe flattening will occur. These cases may be considered to be ' At Risk ' and have the following radiological signs. A lytic area in the lateral part of the epiphysis and adjacent metaphysis, calcification lateral to the epiphysis, lateral subluxation, an extensive metaphyseal change and a horizontal growth plate. Treatment is indicated when two or more of the ' At Risk ' signs are present.

It is important to differentiate the changes already described from those conditions which produce a Perthes-like change. They are multiple epiphyseal dysplasia, cretinism, infection, sickle cell disease and Gaucher's disease (p. 875).

Prognosis.—The long term complication of this disease is osteoarthritis of the hip. The symptoms of this may be delayed for 30–40 years. The more deformed the femoral head at the time of healing the greater the chance of early symptoms. The prognosis depends on the age of the child at the onset of the disease. This is because younger children are lighter and less likely to damage the epiphysis and also have longer to remodel the femoral head after the disease has healed. Girls have a worse prognosis than boys.

Treatment.—There is no specific treatment for the underlying pathology. The object of treatment must be to prevent severe flattening of the femoral head. In the past it was the practise to confine the child to bed for 2–3 years to prevent this deformation but subsequent research has demonstrated that this will not alter the natural history.

Because the femoral head, particularly its lateral cartilaginous part, is outside the acetabulum and also because the movements of the hips are reduced, the principles of treatment have been redefined as:

(1) Restoration of movement by
    (i) Containment of the femoral head within the acetabulum
    (ii) Mobilisation of the reduced hip

(2) Prevention of further ischæmia by
    (i) Relief of further stress to the hip
    (ii) Prevention of injury

Containment of the femoral head within the acetabulum may be achieved by a Broomstick (Broomstick plasters) with the legs in abduction and internal rotation. Active movement is encouraged in the plaster to restore the normal range of movement to the hip.

Once the movement of the hip has been restored the clinician must decide how to maintain the reduction achieved. He can either do it by retaining the Broomstick plaster or by realigning the leg leaving the femoral head in the reduced position. This may be achieved by femoral osteotomy of the varus (adduction) and rotation type or by the innominate osteotomy of Salter. It must be stressed that 60 per cent of cases achieve a good result without treatment and therefore in mild forms of the disease where there is minor involvement and a good range, simple out-patient supervision is all that is required.

Robert Salter, Contemporary. Professor of Orthopædic Surgery, Hospital for Sick Children, Toronto, Ontario, Canada.

Infarction of the Navicular (Köhler's disease).—This condition presents with physical signs and radiological appearances analogous to those of Perthes' disease (fig. 299). The affected bone becomes painful and tender and apparent radiological deformation occurs. Treatment consists of relieving weight from the affected foot until revascularisation and a normal bony radiological appearance have been achieved.

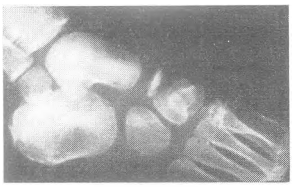


FIG. 299.—Köhler's disease.

Avascular Necrosis of the Lunate (Kienböck's Disease)

This may follow serious injury, for example dislocation, in which all the soft tissue attachments of the bone are ruptured so that the bone is infarcted. It may also follow trivial injuries, sometimes after an interval of years. Sometimes there is no history of trauma at all. The wrist becomes painful and stiff. Radiologically the lunate is dense and deformed (fig. 300). Long-standing cases are complicated by osteo-arthrosis.

Treatment.—The patient should be advised to adjust his occupation so as to limit the demands on his wrist. If this is impossible, the lunate or the proximal row of the carpus is excised, or the wrist is arthrodesed.

FIG. 300.—Kienböck's Disease.

FIG. 301.—Freiberg's disease of the second metatarsal. (*F.P. Fitzgerald, F.R.C.S.I., London.*)

Infarction of the Head of the Second Metatarsal (Freiberg's disease).—This condition (fig. 301) is not entirely similar to the two foregoing diseases in that infarction occurs in a rather older age group, namely in adolescents aged between fifteen and twenty-five years. The ' disease ' may in fact be due to a compression fracture of the epiphysis rather than to infarction (see Transchondral Fractures p. 298). Treatment is difficult and consists of attempting to weight-relieve the affected bone by an appropriately placed insole. If pain persists, the head of the second metatarsal should be elevated by an osteotomy of the shaft.

Traction Injuries to Epiphyses

Pathology.—Because the epiphyseal plate represents a point of weakness in the long bones, mechanical failure may occur in the region of the epiphyseal plate which is impossible in the adult. Slipped femoral epiphysis (p. 290) is one example of mechanical failure of this kind. Certain epiphyseal plates may be loaded in tension, a mode of loading which they do not appear to be well able to withstand, so that traction injuries form another example of

Alban Köhler, 1874–1947. Radiologist, Wiesbaden, Germany. Described this disease of the tarsal scaphoid in 1908.
Robert Kienböck, 1871–1953. Professor of Radiology, Vienna. Described this condition in 1910.
Albert Henry Freiberg, 1868–1940. Professor of Orthopædic Surgery, Cincinnati University, Cincinnati, Ohio, U.S.A. Described infarction of the second metatarsal bone in 1914.

such lesions. These injuries produce physical and radiological signs which in some ways resemble those associated with other disorders of the epiphysis and which for this reason have been named as ' Osteochondritis '.

Clinical Features.—These injuries occur in the knee, the heel, and the spine. In the knee the tibial tubercle, which is part of the proximal tibial epiphysis, may gradually be avulsed from the shaft, which is part of the metaphysis. This condition is known as ' *Osgood-Schlatter's disease* ' (fig. 302). A corresponding condition may occur at the distal pole of the patella where the condition is known as ' *Sinding Larsen's disease* '. At the heel the posterior epiphysis of the os calcis may be partially avulsed by the Achilles' tendon. This condition is known as ' *Sever's disease* '.

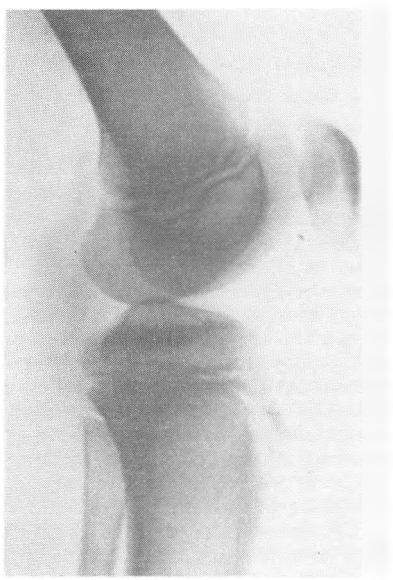


FIG. 302.—Osgood-Schlatter's disease; decalcification, fragmentation, and partial separation of the tongue-shaped portion of the epiphysis from the upper tibial shaft. (*Robert Maguire, F.R.C.S., Myrtleford, Vic., Australia.*)

In the spine the disease may occur in the vertebral bodies where it is known as ' *Scheuermann's disease* '. The disease affects particularly the 6th to the 10th thoracic vertebræ, and the lumbar spine. To understand this condition two facts must be appreciated: firstly, that in the normal hydrated intervertebral disc the nucleus pulposus functions as a fluid and accordingly is positively pressurised when the disc is loaded in axial compression. The pressure generated in the nucleus pulposus is applied to the annulus fibrosus which is therefore under tension when the disc is loaded. Secondly, the annulus fibrosus is attached to ring-shaped epiphyseal plates on the superior and inferior surfaces of the vertebral body. Thus traction injuries may occur at the cartilagenous attachment of these epiphyses to the vertebral body. This occurs particularly in the anterior portion of the epiphysis and results in interference with the growth of this portion of the vertebral body. The vertebral bodies become narrowed anteriorly, especially in the thoracic region, to produce a smooth, rigid kyphosis. In the lumbar region slight anterior narrowing occurs but this is not clinically evident since the normal lumbar spine is lordotic. The epiphysis may fail to fuse with the rest of the vertebral body and thus appears as a separate island of bone in the adult. Probably because the disease is associated with a disturbance of the intervertebral disc, the corresponding disc spaces are narrowed. It may even be that this condition is not strictly a traction injury to the epiphysis at all, but

Robert Bayley Osgood, 1873–1956. Professor of Orthopædic Surgery, Harvard University, Boston, Mass., U.S.A.
Carl Schlatter, 1864–1934. Professor of Surgery, Zurich, Switzerland. Osgood and Schlatter described osteochondritis of tibial tubercle independently in 1903.
Christian Magnus Falsen Sinding Larsen. Norwegian Surgeon. Described this disease of the patella in 1921.
James Warren Sever, 1878–1964. Orthopædic Surgeon, Children's Hospital, Boston, Mass., U.S.A. Described apophysitis of the os calcis in 1912.
Holger Werfel Scheuermann, 1877–1960. Radiologist, Municipal Hospital, Sundby, Copenhagen. Described juvenile kyphosis in 1920.

rather represents an extrusion of nuclear material through the epiphyseal plate, separating the epiphysis from the metaphysis. Whatever their cause, these events alter the mechanics of the spine and thus may cause back pain.

Treatment.—No matter where these conditions occur, treatment consists of resting the involved epiphysis until pain and tenderness have subsided. This usually takes about 6 weeks.

No persistent disability follows these conditions save in the spine where there may be a permanent increase in the normal dorsal kyphosis, sometimes accompanied by back pain.

Transchondral Fractures. (Osteochondritis Dissecans.)

Pathology.—A transchondral fracture is one in which the fracture line runs entirely in articular bone, that is to say in bone covered by articular cartilage. Because articular cartilage is more elastic than bone, strains which fracture the bone may not break the cartilage. Thus the bony fragment may be held in its bed by an intact bridge of cartilage. The cartilage is nourished from the synovial fluid and hence remains healthy. In contrast the bone dies because its blood supply is completely interrupted at the fracture line. If the cartilage all round the fracture breaks at the time of injury, the fragment separates to form a *loose body* in the joint. If the cartilage remains partly or completely intact, the fracture may unite and the fragment may become revascularised. Sometimes, however, especially if the fragment is large, revascularisation fails and the dead fragment of cartilage-capped bone may then separate to form a *loose body* long after the injury. Such injuries may occur in adults as well as in children, but in the latter, if the bone has an epiphysis, it is the epiphysis which is involved.

The connection between the injury and the formation of the loose body is often unprovable because the violence required to produce the fracture is slight and because the injury and the formation of the loose body are widely separated in time. The suspicion therefore arises that the formation of some loose bodies may not be due to injury at all. This suspicion is strengthened by the occasional spontaneous development of loose bodies in several joints of the same patient. It has been suggested that this may be due to thrombosis of an end-artery supplying the involved bone. In these cases of uncertain ætiology, it is reasonable to retain the term *Osteochondritis Dissecans*.

Clinical Features.—The condition may affect any convex articular surface but is particularly common in the femoral condyles, and the capitellum. Less commonly it occurs in the head of the femur and the dome of the talus (fig. 380 (*b*)). An unseparated fragment causes aching pain and an effusion in the joint. The lesion can be displayed radiologically but only if views are carefully chosen so as to place the lesion on the 'skyline' (fig. 303). Tomographs may be required. A separated fragment forms a loose body which may lock the joint. Radiologically the loose body and its parent cavity can be seen.

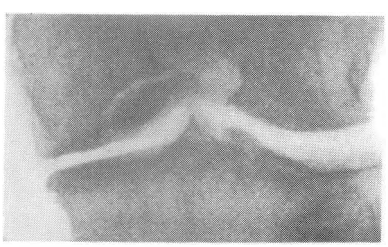


FIG. 303.—Osteochondral fragment separating from the medial femoral condyle. (*Professor Carl Krebs, Aarhus, Denmark.*)

Treatment.—If the fragment separates totally, the loose body must be removed. The crater from which it was derived should be ignored and will gradu-

ally be filled with fibro-cartilage. If the fragment is not displaced from its parent cavity when first detected, a state of affairs which applies particularly to the knee, it should be held in place by two pins (Smillie pins) in the hope that it will eventually re-unite. If the end-result of a transchondral fracture is an irregular articular surface, osteoarthrosis is likely in later life.

Ian Scott Smillie, Contemporary. Emeritus Professor of Orthopædic Surgery, Dundee, Scotland.

CHAPTER 19

GENERALISED DISEASES OF BONE

DISEASES OF UNKNOWN ÆTIOLOGY

Paget's Disease (syn. Osteitis Deformans)

This disease was described in 1877 by Sir James Paget. In three classical papers (1877, 1882 and 1889) he described the advanced polyostotic form of the disease and most of the known complications.

The disease is rare under the age of 40 but becomes progressively more common as age advances, finally reaching an incidence of about 10% in people over 90 years of age. Males are slightly more commonly affected than females. The condition can affect any bone in the body and many bones may be affected in the same patient. The spine, the skull, the pelvis, the femora and the tibiæ are the most commonly involved bones.

The pathological process begins with osteoclastic resorption of existing bone. This is followed by osteoblastic regeneration of a primitive coarse-fibred bone which is highly vascular. This process affects both cortical bone, which becomes increasingly thick and less well demarcated from adjacent cancellous bone, and cancellous bone where massive ill-formed trabeculæ replace the normal structure. Finally the affected bones assume the mosaic pattern of lamellar bone.

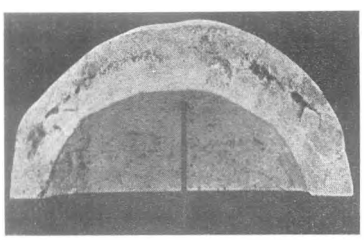


FIG. 304.—Paget's disease of the skull. (*R.C.S. Museum.*)

Clinical Features.—The majority of cases of Paget's disease are discovered incidentally in patients attending a physician for some other reason; relatively rarely a patient presents with a complaint arising directly from the condition. The disease gives rise to visible thickening of the involved bones and a complaint of increased thickness may be the presenting symptom. This applies particularly to the skull (fig. 304) where an inability to wear his normal size of hat may draw the patient's attention to the first changes. The pathological process may be painful, especially in the weight-bearing bones where deformity is taking place: the patient will then present complaining of a dull, continuous aching pain localised in the involved bone. The new bone formed is less strong than normal bone and, either because it deforms under load or because its deposition is irregular, it tends to bend under load producing typical bowing of the involved bones especially in the leg (fig. 305). The consequent deformity may be noticed

Sir James Paget 1814–1899. Surgeon, St. Bartholomew's Hospital, London.

by the patient. Attention is not infrequently drawn to the presence of the disease by the development of one of its complications (see below).

Physical Signs.—Subcutaneous bones may be obviously thickened and bowed, but otherwise there are no significant findings. Because of the massive increase in vascularity of the bones, the involved parts of the skeleton may become the functional equivalent of arterio-venous fistulæ and as a consequence lead to left ventricular failure (p. 302).

Radiological Appearances.—The involved bone is thickened, and patchily sclerotic. The normal clear line of demarcation between cortex and medullary canal becomes blurred. The long bones may be deformed. The final appearance of the bone is one of predominant sclerosis with a typical mottled appearance (fig. 306).

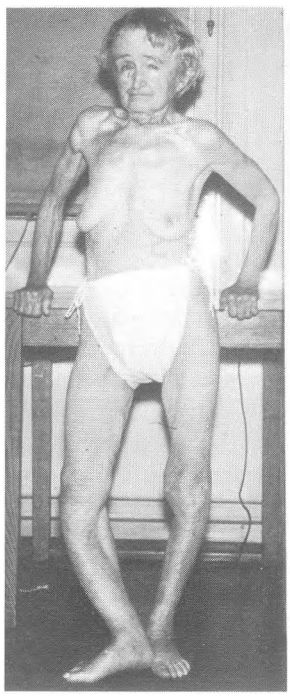


FIG. 305.—Paget's disease affecting the skull, which is large, and the legs (especially the right tibia) where the bones are thickened and bowed.

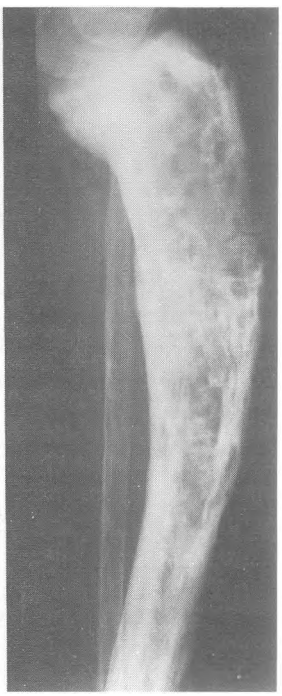


FIG. 306.—Paget's disease involving the tibia showing thickening of the bone, bowing, a pathological fracture anteriorly, and mottled sclerosis.

Special Investigations.—The serum calcium and serum phosphorus are usually within normal limits. The serum alkaline phosphatase is often very high, but this finding is in no way specific for it is a feature of most diseases in which osteoclasis is taking place. A bone scan, using one of the bone-seeking isotopes, shows markedly increased uptake in the involved areas of the skeleton (fig. 307).

Treatment.—The pain of Paget's disease can now be relieved by treatment with calcitonin. The extent to which this hormone also reverses the pathological process is uncertain but current evidence suggests that it does so to a significant extent. Whether or not it affects the incidence of complications, especially of malignant change, remains to be seen.

Complications

1. **Osteogenic sarcoma** develops in approximately 5% of patients with Paget's disease. The neoplasm is extremely malignant and death is almost invariable once this complication has developed. Treatment should be along the lines described for osteogenic sarcoma in general (p. 261).

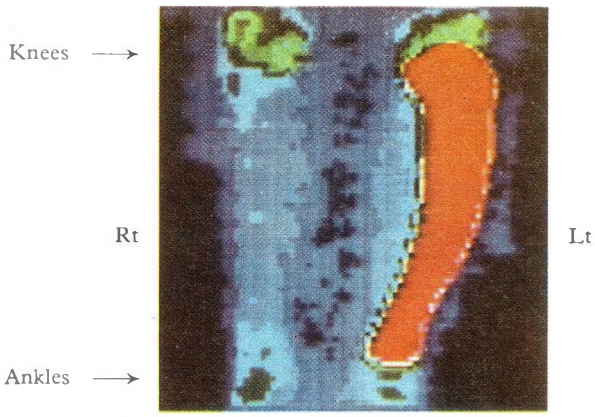


FIG. 307.—A bone scan of the lower legs in Paget's disease showing markedly increased uptake in the involved tibia on the left.

2. **Pathological fracture** may occur through long bones involved with Paget's disease. The fractures are usually transverse and the appearance of a transverse fracture in the tibia or femur in a patient over the age of 40 whose bone looks a little thickened should raise the suspicion of Paget's disease. Union of these fractures usually occurs uneventfully whether they are treated by internal fixation or by external splintage. Internal fixation may be indicated because of the age of the patient, but is usually difficult since the medullary canal has been lost and the bone is extremely vascular.

3. **Osteoarthrosis** occasionally develops in joints adjacent to deformed bone, but it is by no means certain that the incidence of osteoarthrosis is in fact higher than in the uninvolved population of similar age.

4. **Paraplegia** is a rare complication of involvement of the vertebræ.

5. **Heart failure** (high output) may occur due to the arteriovenous shunts within the affected bones.

6. **Deafness** may occur due to bony involvement of the middle ear.

Osteoporosis

This condition may occur in a localised form following local inflammation or disuse (due for example to paralysis or immobilisation). A generalised form may develop either primarily, and as such represents an almost invariable concomitant of increasing age, or secondarily following endocrine disorders.

Senile osteoporosis first becomes manifest in women shortly after the menopause and in men at around the age of 65, but it is rarely a clinical problem in women before the age of 65, and in men before the age of 85. There is no precise dividing line between normality and osteoporosis and the latter can be detected radiologically only when about 40% of the skeleton has been lost. There is now evidence that the major cause of osteoporosis in ageing women is the change in their hormonal environment after the menopause. Disuse (secondary to the generally reduced level of activity in the elderly) may also play a part. It is also suggested that loss of bone in the elderly may be due in part to osteomalacia (p. 304) as well as to osteoporosis.

The condition is characterised by a reduction in the amount of bone in the

skeleton, which is only radiologically manifest when approximately 40% of the skeleton has been lost. At this time the cortices of the long bones are noticeably thinner and cancellous bone less dense than normal. As a consequence of the latter change, the invertebral discs may intrude into the centres of the vertebral bodies to produce a typical 'fish head' appearance. Histologically the bone is perfectly normal and there are no demonstrable metabolic changes.

The disease itself is symptomless and only constitutes a clinical problem in so far as osteoporotic bone is more liable to fracture than normal bone. The fractures commonly associated with senile osteoporosis are Colles' fracture, fractures of the proximal humerus, crush fractures of the vertebral bodies, fractures of the femoral neck and intertrochanteric fractures of the femur. The first two and the last of these fractures are always associated with a clear-cut history of a fall and as such are perhaps as much due to the unsteadiness of the elderly as to their osteoporosis. Fractures of the vertebral bodies and of the femoral neck, on the other hand, may occur without a history of injury and there is now evidence that they represent the end-result of progressive fatigue fracture of isolated trabeculæ. Such fractures can be attributed to the increased stresses which osteoporotic bone must encounter because of its reduced mass.

Fractures present either obviously, in the case of the Colles' fracture or fracture of the femoral neck, or as back pain in the case of compression fractures of vertebral bodies. The fracture should be treated on its own merits, ignoring the presence of osteoporosis, which does not interfere with healing. No specific treatment is available for osteoporosis itself.

DEFICIENCY DISEASES AFFECTING BONE

A deficiency of certain vitamins may lead to abnormalities in the skeleton. In this section these diseases will be mentioned only in the briefest outline in so far as the bony complications demand orthopædic treatment. The student is referred for a more detailed treatment of the diseases themselves to appropriate textbooks.

Rickets.—Rickets is due to a dietary or absorption deficiency of Vitamin D. Uncalcified bone matrix is deposited normally but fails to calcify. Histologically, the bone contains large areas of uncalcified matrix which is known as osteoid. The disease affects both bones formed by endochondral ossification and bones, such as the skull, which ossify in membrane.

The disease usually presents in infants, but may develop at any time during the period of growth if the diet becomes deficient in vitamin D. Clinically, characteristic abnormalities develop in the skeleton: the costochondral junctions enlarge to cause the 'ricketed rosary', the long bones bow under the body weight, the skull develops frontal bosses, and the pelvis becomes triradiate. In general the stature is stunted (figs. 308–310).

Treatment.—The treatment of the disease itself consists of the administration of vitamin D. Early bony deformities may respond to appropriate splinting, but if they are more than trivial they will require correction by manual osteoclasis in the third and fourth year of life, or osteotomy thereafter. The tibia is the bone most commonly requiring orthopædic treatment.

Osteomalacia is the analogue of rickets occurring in the adult skeleton. Once again excessive osteoid is demonstrable histologically and the bones become unduly soft so that they deform under the body weight. Treatment consists of correction of deformities by osteotomy where necessary and practicable, together with the administration of vitamin D.

(*a*) (*b*)

FIG. 308.—Rickets: (*a*) The active state; (*b*) The healing state.

Other Causes of Excessive Osteoid in Growing and Adult Bones.—In children phosphaturia (synonyms—renal tubular rickets, vitamin D resistant rickets), and Fanconi's syndrome (amino acid-uria) both produce the changes of rickets. In adults renal tubular acidosis produces the changes of osteomalacia. Renal failure occurring from whatever cause in children may produce dwarfism and occasionally the appearance of rickets, whilst in the adult it occasionally produces osteomalacia and/or osteitis fibrosa. Osteoid tissue is often present in excess in hyperparathyroidism, together with the more specific lesion of osteitis fibrosa.

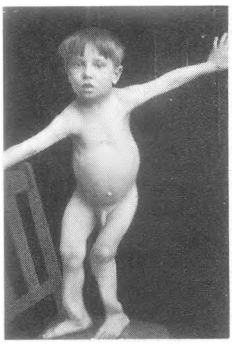


FIG. 309.—Rickets, showing the deformed legs and pot-belly.

Scurvy.—Scurvy is a deficiency disease attributable to a lack of vitamin C. In the skeleton endochondral ossification ceases but chondrocyte proliferation continues. As a consequence a chaotic state of affairs develops in the epiphyseal line: fractures may occur following insignificant trauma and hæmorrhages are common. Hæmorrhages occurring either around the epiphyseal line or subperiostally in the shaft, may ossify to give rise to bony swellings.

Treatment consists of the administration of vitamin C.

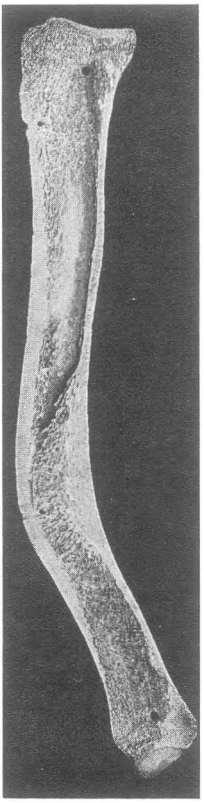


FIG. 310.—Rachitic tibia showing abrupt curvature in the lower third of the bone, and well-marked buttress formation.

ENDOCRINE DISEASES AFFECTING BONE

Adrenalcorticosteroids in excess produce osteoporosis. This may occur either secondarily following increased stimulation from the pituitary or primary as a consequence of an adrenal cortical tumour. High doses of steroids may be administered therapeutically and have the same effect of reducing skeletal

Guido Fanconi, Contemporary. Emeritus Professor of Pædiatrics, Zürich, Switzerland. Described this syndrome in 1936.

mass. This complication occasionally develops during the course of the treatment of rheumatoid arthritis and greatly increases the disability due to the disease itself.

Disorders of the pituitary may affect the skeleton not only indirectly via the adrenal cortex, but also directly. A deficiency of growth hormone developing during the growth phase produces pituitary dwarfism. Excessive growth hormone consequent upon the development of a pituitary adenoma produces pituitary gigantism if the tumour develops before the epiphyses have closed, and acromegaly if it develops after the epiphyses have closed. No orthopædic treatment is available for any of these conditions.

Hyperparathyroidism affects the skeleton. The disease itself is dealt with in Chap. 36. The bone changes in hyperparathyroidism retain the name (osteitis fibrosa cystica) which they were given when they were thought to represent a separate disease entity. Histologically, marked osteoclastic activity can be seen and the resorbed bone is replaced with fibrous tissue to produce the so-called 'brown tumours' (see Giant Cell Tumour, p. 260). As a consequence, patchy osteolytic lesions develop throughout the skeleton and pathological fractures may occur through them. It is of particular diagnostic importance that resorption affects the terminal phalanges in the hand and the alveolar margins in the jaws, so that the radiological appearances of the hands and jaws are characteristic. The blood chemistry is abnormal: the serum calcium is raised, the serum phosphorus lowered, and the alkaline phosphatase usually somewhat raised.

Orthopædic treatment is required only for pathological fractures. These are treated on their merits. They unite satisfactorily provided the underlying endocrine disease is treated.

CHAPTER 20

DISEASES OF MUSCLES, TENDONS AND FASCIÆ

NON-ARTICULAR RHEUMATISM

'NON-ARTICULAR RHEUMATISM' is a poor, but not easily abandoned, name for a group of common conditions the pathology (or pathologies) of which are not understood. The conditions present as pain localised to the aponeurotic attachment of muscle to bone. The commonest sites for this to occur are 1. the attachment of the extensor muscles of the forearm to the lateral epicondyle of the humerus (where the condition is called 'tennis elbow'), 2. the attachment of the flexor muscles to the medial epicondyle (' golfer's elbow '), 3. the attachment of the rotator cuff of shoulder muscles, especially the portion made up by the tendon of the supraspinatus muscle, to the greater tuberosity of the humerus, 4. the attachment of the ligamentum patellæ to the patella or the tibia, 5. the attachment of the Achilles' tendon to the os calcis, 6. the attachment of the plantar fascia to the os calcis. Similar symptoms commonly develop in the muscles of the back and it is possible that here they are due to the same pathological process affecting the bony attachment of one of the back muscles. In the back, however, localised pain of this kind has been attributed to reflex muscular spasm arising from some (unknown) deep-seated lesion in the spine or alternatively to ' fibrositic nodules '. (The terms ' fibrositic nodule ' and ' fibrositis ', like the term used here ' non-articular rheumatism ', are meaningless and do not advance our understanding of the condition but at least the latter does not imply a knowledge of the pathology.)

The tender area, especially at the lateral epicondyle of the humerus and at the attachment of the rotator cuff, has been explored on various occasions but significant morbid anatomical features have not always been demonstrated. It seems that the condition may be caused by a tear in the collagen fibres attaching the muscle to the bone which, because it is repeatedly loaded does not heal satisfactorily. The tear may be caused by overt injury, by the loads imposed in excessive but normal activity (as for example, at the elbow, by typing or tennis: hence ' tennis elbow ') or by weakening of the tissues following an interruption of their blood supply (as for example happens in the rotator cuff and possibly the Achilles' tendon).

The specific features of these lesions are as follows:

Tennis Elbow.—The patient complains of pain in the elbow at rest and in particular when he uses the hand and hence contracts the wrist and finger extensors. The attachment of the extensor muscles of the forearm to the lateral epicondyle is tender, and active resisted contraction of the extensor muscles of the fingers or the wrist provokes pain at the elbow.

Golfer's Elbow.—This is a precisely similar condition to tennis elbow,

save that it affects the attachment of the flexor muscles of the forearm to the medial epicondyle. Tenderness is often less well localised than in tennis elbow (and treatment is even less satisfactory).

Supraspinatus Tendonitis.—In this condition pain is felt in the shoulder, especially on abduction and rotation, and localised tenderness is present over the insertion of the supraspinatus tendon.

A feature of supraspinatus tendinitis or of any painful pathological process around the shoulder is the rapid onset of shoulder stiffness. This is probably to be attributed to the close proximity of the humerus to the acromion so that adhesions readily form between the two bones (involving the rotator cuff and the sub-acromial bursa) which limit movement of the humerus on the scapula.

In supraspinatus tendonitis, the pathology is a little more clearly understood than in other examples of this condition. Injection studies of post-mortem material have shown that the blood supply to the supraspinatus tendon commonly becomes defective in middle and later years—probably because of compression of the tendon between the acromion and the proximal humerus. As a consequence the tendon is prone to degenerate and may even rupture spontaneously (see Tendon Ruptures, p. 308). Progressive calcification of the tendon may develop and be obvious radiologically (fig. 311).

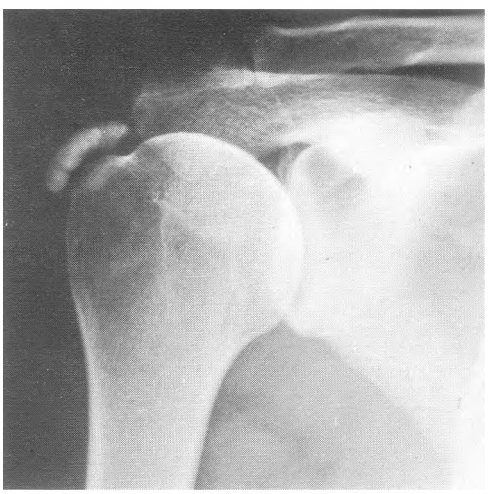


FIG. 311.—Supraspinatus calcification. Faint shadow of calcium seen lying above the head of the humerus just lateral to the outer margin of the acromion.

Pain occurs on moving the humerus, especially as the inflamed portion of the supraspinatus tendon passes between the head of the humerus and the acromion: this occurs in the middle third of the abduction arc so as to give a typical clinical picture, described as the *painful arc syndrome* in which the patient is freely able to abduct his arm through the initial 30°, has pain raising the arm through the next 30°, but thereafter is able to abduct the arm painlessly. Pain recurs as the arm is brought down to the side through the same mid 30° of abduction. As a consequence of the pain provoked by movement, such patients naturally tend to protect the shoulder by holding it still. Adhesions now form which limit shoulder movement mechanically. Thus the patient initially complains of pain in the shoulder and soon thereafter notes stiffness. The pain and stiffness tend, in the absence of treatment, gradually to increase for 3 months but thereafter remain static—usually for a further period of 3 months. During the latter half of this second period

pain gradually settles in the shoulder so that now the patient finds it is more comfortable to move it. Probably as a consequence of increasing comfort, the range of movement gradually increases until by about the end of 9 months the whole cycle is completed and the joint returns essentially to normal. This sequence of events has been given several names of which probably the best is *frozen shoulder*. It should be clearly understood that a frozen shoulder is not a specific clinical or pathological event: it is merely a response of the shoulder to any locally painful pathological process.

Patellar Tendonitis.—Occasionally the ligamentum patellæ may become painful and tender in adults either at its attachment to the patella or at its distal attachment to the tibia. Pain is felt on active extension of the knee.

Plantar Fasciitis is described on p. 331.

Achilles' Tendonitis is described on p. 331.

Treatment.—No satisfactory treatment is available for these conditions. The patient should be advised that the condition is normally self-limiting and that he should, so far as possible, avoid powerful contraction of the involved muscles without keeping the joint itself still: movement in the absence of powerful muscular activity is the objective. Local injections of 25 mg. of hydrocortisone and local anæsthetic into the tender area sometimes bring about dramatic relief. When the supraspinatus tendon is calcified it may suddenly become excruciatingly painful. Such pain can be relieved by incision and evacuation of the calcareous deposit. Surgical treatment is required, rarely, if these conditions persist and are severely painful. The procedures aim to release or decompress the involved tendon but, like conservative measures, they do not always bring symptomatic relief.

PATHOLOGICAL TENDON RUPTURES

The supraspinatus tendon may rupture spontaneously following interruptions in its blood supply in middle aged or elderly adults. The tear may include only the supraspinatus tendon or mav extend throughout the rotator cuff. The rupture often occurs following insignificant violence or even as part of normal everyday activities. The patient complains of sudden pain in the shoulder the severity of which usually masks the important physical sign: an inability to initiate abduction. For this reason and because once abduction has been started passively (for example by the patient tilting his body to the affected side so that abduction is initiated by gravity) the deltoid can continue abduction in a normal way, the diagnosis is frequently missed.

The disability, even if the lesion is left untreated, is very slight. This is fortunate since the only form of treatment available is suture of the tendon and this is frequently unsatisfactory—partly because the tendon holds sutures poorly, and partly because the operation often results in adhesion formation and hence in a stiff shoulder. Thus if the patient is prepared to accept the disability, it is probably best to leave the condition untreated.

The long head of biceps brachii may rupture in the bicipital groove. This is probably an attrition rupture and again occurs spontaneously. The

patient complains of sudden pain in the upper arm followed occasionally by a little bruising. The diagnosis is made by asking the patient to flex the elbow against resistance whereupon the affected biceps muscle bunches distally leaving a gap proximally (fig. 312).

Once again the disability is usually trivial and if the patient is prepared to accept it no treatment should be offered. Should the patient miss the slight loss of elbow flexor power which follows this injury, it is feasible to reattach the proximal end of the biceps to the humerus itself.

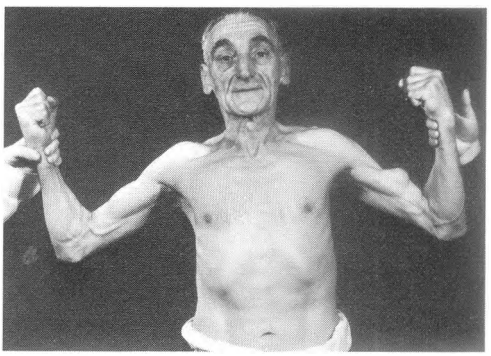


FIG. 312.—Spontaneous rupture of the tendon to the long head of the left biceps muscle. Note that when the muscle contracts the long head bunches near the elbow.

The rectus femoris muscle may be ruptured by normal loads at its attachment to the patella. This condition is discussed on p. 396.

The Achilles' tendon may rupture after insignificant violence—probably because it is weakened by vascular insufficiency as is the supraspinatus tendon. This condition is discussed on p. 386.

TENDON SHEATHS

Simple Tenosynovitis.—This condition follows excessive or unaccustomed use, and is commonly seen in connection with the extensor tendons of the hand and the Achilles' tendon. Pain and local œdema are present and a characteristic soft crepitus (like the rustling of tissue paper) is sometimes palpable when the fingers are moved. The condition is treated by resting the tendons involved, which in the case of the extensors of the hand must of course involve the fingers. A minimum of 3 weeks absolute rest on a splint is usually required, followed by a period of gentle but progressive activity.

Suppurative Tenosynovitis.—This condition affects particularly the flexor tendons of the hand since these sheaths are far more commonly injured than those elsewhere in the body. The condition is discussed on p. 204.

Stenosing Teno-Vaginitis.—This is a condition of unknown ætiology in which the sheath of a tendon thickens, apparently spontaneously, so as to entrap the tendon. Pain and limitation of movement result. The condition is most common at the common sheath of the abductor pollicis longus and extensor pollicis brevis tendons of the wrist (*de Quervain's disease*) and at the retinaculæ of the flexor tendons of the fingers and thumb in the palm ('*trigger finger*'). In the palm, the flexor muscles are sufficiently strong to continue forcing the tendon through the diminished gap in the flexor reticulum. The flexor tendon as a consequence gradually develops a constriction under the retinaculum and a bulge distal to it. Finally, the flexor muscles may force the bulge through the retinaculum but the extensor muscles may be insufficiently powerful to extend the finger thereafter. The finger now snaps as it passes through the constriction and finally locks in a

Fritz de Quervain, 1868–1940. Professor of Surgery, Berne, Switzerland. Described Stenosing Teno-Vaginitis in 1895.

position of flexion from which attempts passively to extend the finger are painful.

Both de Quervain's disease and trigger finger can be cured by dividing the appropriate retinaculum surgically. It might be expected that following this operation the involved tendon would subluxate but in practice this never occurs.

Carpal Tunnel Compression of the Median Nerve.—The carpal tunnel is formed by the flexor retinaculum anteriorly and by the distal row of the carpus posteriorly (fig. 313). The median nerve and the flexor tendons pass through it. Sometimes as a consequence of some clearly demonstrable pathology such as rheumatoid arthritis involving the synovial sheaths of the flexor tendons, the contents of the tunnel increase in bulk and compress the median nerve. Usually, however, signs of median nerve compression develop without any obvious increase in bulk in the contents of the tunnel.

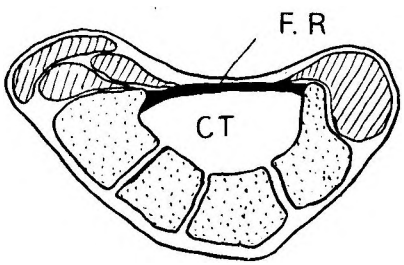


FIG. 313.—Carpal tunnel formed in the cavity of the carpus by the flexor retinaculum.

The ætiology of this condition is unknown but it may be an example of *stenosing teno-vaginitis*, in this case affecting the flexor retinaculum. The condition usually occurs in otherwise normal individuals but is associated with myxœdema (Chap. 35) and pregnancy.

The patient complains of pain which classically is localised to the distribution of the median nerve in the hand but which may be felt anywhere in the arm. The poor localisation of the pain may make the diagnosis difficult since it is easy to confuse the condition with other causes of pain in the arm, for example with cervical spondylosis. Commonly the pain is worse at night and the patient may notice that he is then relieved by hanging the hand over the edge of the bed, a feature of the history which is almost diagnostic of carpal tunnel compression of the median nerve. As the compression increases in severity more obvious neurological symptoms develop including paræsthesæ, numbness, inco-ordination, and finally frank weakness in the muscles of the hand innervated by the median nerve (of which the easiest to examine is abductor pollicis brevis, wasting of which is obvious). The fingers, apart from being inco-ordinated, may also feel stiff, especially first thing in the morning, a symptom which is probably attributable to compression of the flexor tendons themselves. Examination of the hand is frequently negative but may demonstrate slight tenderness over the carpal tunnel, an increase in pain if the fingers and wrist are held fully flexed for a few moments, and the presence of abnormal neurological signs in the distribution of the median nerve in the hand. Conduction studies on the median nerve demonstrate delay at the carpal tunnel.

Treatment.—Whatever may be the pathology, simple decompression of the tunnel by longitudinal incision of the flexor retinaculum cures the condition, often in a dramatic fashion. Rheumatoid tenosynovitis may in addition require synovectomy.

FASCIÆ

Dupuytren's contracture is a localised thickening of the palmar, and rarely the plantar, fascia. The fascia thickens to form nodules and contracts so that eventually the affected fingers are drawn into flexion. The overlying skin of the palm adheres to the fascia. Most commonly the disease starts near the base of the little finger and soon draws that finger into the palm of the hand (fig. 314). Later it involves the ring finger and, less often, the long and then index fingers, in the same way. In long-standing cases permanent changes take place in the metacarpo-phalangeal and the proximal interphalangeal joints which render futile any attempt to straighten the fingers.

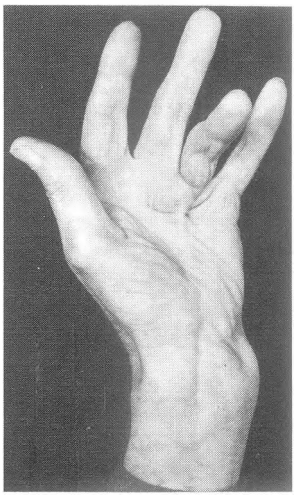


FIG. 314. — Dupuytren's contracture; note puckering of adherent skin in the palm.

The pathology of the condition is unknown. In the past it was believed to be due to repeated trauma to the palm. However, it is known to be familial and is frequently seen in patients who have never experienced such trauma. That it is so often bilateral and may involve the feet is also against any traumatic origin. It is more common in men than in women and may be seen in cirrhotics and epileptics who take sodium hydantoin.

Treatment.—Early cases can be treated by night splintage and gentle stretching by the patient. More advanced changes require excision or (less satisfactorily) multiple division of the affected fascia. This operation can give excellent results in all but severe longstanding cases where permanent joint changes have developed. In these cases it may be advisable to amputate the affected finger.

GANGLIA

Ganglia are localised, tense (but often painless) cystic swellings containing clear gelatinous fluid. They often communicate with, and are always adjacent to, a tendon sheath or the capsule of a joint. Their origin is uncertain but they are probably caused by leakage (and subsequent fibrous encapsulation) of synovial fluid through the capsule of a joint or tendon sheath. They are sometimes predisposed to by injury. Simple ganglia are most commonly found on the dorsum of the wrist and the foot (fig. 315). Occasionally minute ganglia develop on the flexor aspect of the fingers which although small, are exquisitely painful and tender.

Treatment.—Traditionally, ganglia were burst by striking them with the family Bible! Today, if they do not resolve spontaneously, they are excised.

Baron Guillaume Dupuytren, 1777–1835. Surgeon, Hôtel Dieu, Paris. Described Contracture of the Palmar Fascia in 1831.

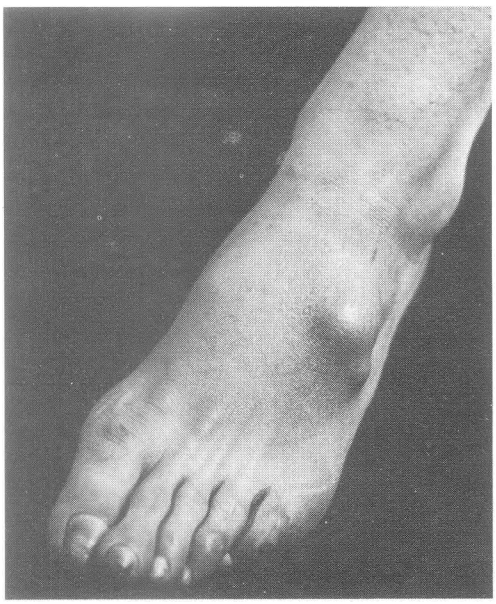


FIG. 315.—Simple ganglion on the tendon of the peroneus brevis muscle.

BURSÆ

Simple Bursitis. — Acute traumatic bursitis follows injury or unaccustomed exercise, for example inflammation of the bursa anterior to the Achilles' tendon may follow a cross-country run.

Chronic Bursitis.—This is the result of repeated pressure or slight injuries, to anatomical bursæ. Examples are involvement of the *prepatellar bursa* (housemaid's knee, fig. 316) or *olecranon bursa* (students or miner's elbow, fig. 317), and subsartorial bursitis (fig. 318).

Involvement of the *semimembranosus bursa* occurs particularly in children (fig. 319). The cyst is aspirated if it causes disability, but most cysts disappear spontaneously. Semimembranosus bursæ, if they communicate with the knee joint, sometimes enlarge when there is an

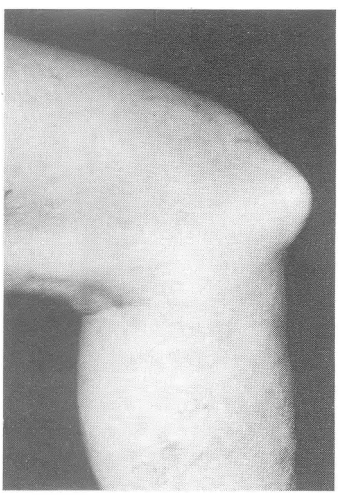


FIG. 316.—Housemaid's knee.

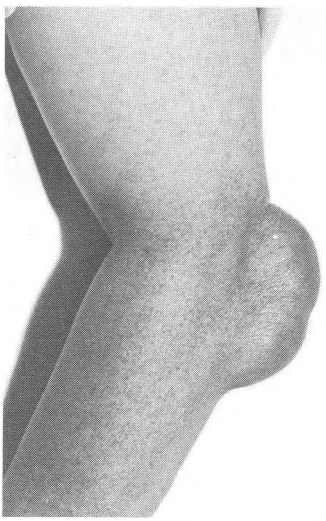


FIG. 317.—Chronic olecranon bursitis.

effusion in the joint, for example as a result of osteoarthrosis, and form one variety of *Baker's cysts*. (In general, synovial cysts in the popliteal fossa, which are known as Baker's cysts, may arise as described above from the

William Morrant Baker, 1839–1896. Surgeon, St. Bartholomew's Hospital, London. Described these cysts in 1877.

semimembranosus bursa or, in rheumatoid arthritis of the knee, from a posterior rupture of the capsule of the joint.)

Acute Suppurative Bursitis.—This is due to direct infection by penetrating wounds, or by the spread of local cellulitis. The prepatellar bursa is the most commonly involved. A ' sympathetic ' effusion in the knee joint sometimes follows. Confusion with infective arthritis should be avoided,

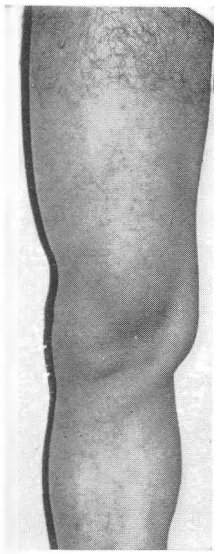


FIG. 318.—Subsartorial bursitis.

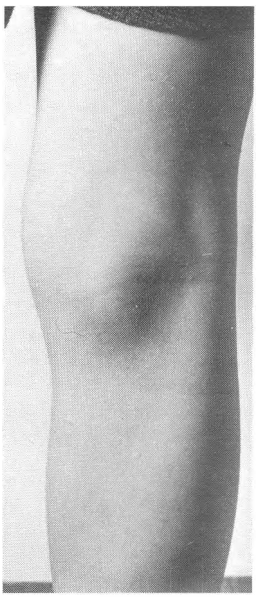


FIG. 319.—Semimembranosus bursitis.

as in the latter condition any attempt to move the joint is painful, and at the knee pain is elicited by pressure in the popliteal space (see p. 223). The infection usually responds to chemotherapy, but if pus is already present incision and drainage will be necessary.

Excision of Bursæ.—Persistent trouble with a subcutaneous bursa (e.g. olecranon, prepatellar) may only be resolved by complete excision of the whole of the endothelial lining and surrounding ' capsule ' under a general anæsthetic.

Adventitious bursæ form as a result of prolonged pressure over bony prominences. The term ' adventitious ' means that no anatomical bursa was present at the site of the newly formed cyst, and that it was generated in connective tissue as a result of repeated shearing motion in the tissue. One of the commonest of such bursæ is that over the medial aspect of the head of the first metatarsal in hallux valgus (p. 328). Like anatomical bursæ, adventitious bursæ can become infected.

CHAPTER 21

NEUROLOGICAL DISORDERS AFFECTING THE MUSCULO-SKELETAL SYSTEM

In spite of the wide variety of diseases which affect the nervous system the consequences for the locomotor system tend to follow a common pattern so that it is possible to lay down certain principles of management which have a general application. The loss of motor power and sensory faculty require separate consideration, although both are commonly together.

PARALYSIS

The consequences of paralysis are twofold:—

1. *Loss of Function.*—This results from the failure of action of the affected muscles and, while many of its consequences are self evident, the loss of joint stability is of particular importance.

2. *Liability to Deformity.*—In the adult this is seldom a serious problem for although there is a tendency for contractures to develop in the non-paralysed muscles these are relatively easily controlled by passive movements and splintage. In the growing child however the problem is much more serious for here there is a disturbance of growth. For reasons not well understood the non-paralysed muscle becomes relatively shorter than its paralysed fellow so that the joints concerned become fixed in the direction of the acting muscles. These changes may be followed by secondary bony deformity leading to even greater fixity of the joint. In addition to these particular deformities there may be an overall shortening of the limb, again for reasons which are not readily apparent.

It is important to appreciate that these forces of unbalanced growth are almost irresistible and just as a solid pavement yields before the persistence of a penetrating weed so also will splints and calipers fail if they are used alone in an attempt to correct or even to control these deformities. On the other hand it must be remembered that a totally paralysed limb has no tendency to deform since there is no imbalance to distort it, although its overall dimensions are usually reduced. In these circumstances gravitational force may have some distorting effects particularly in the production of equinus deformity of the foot, but this is easily resisted.

The Management of Paralysis

This has 3 main objectives as follows:

1. **The Elimination of Deformity.**—In the adult the emphasis should be on prevention, with the institution of passive movements and splintage as

soon as possible. If fixed deformity does develop simple tenotomy will usually be sufficient to bring it under control.

In the child splintage alone will not suffice and it will usually be necessary surgically to divide the contracted structures and in particular the tendons of the unopposed muscles acting on the joint. Once these deforming forces have been eliminated it may be possible to complete the correction by serial casts and splintage. Bony deformity may also be corrected at this stage providing that the epiphyses are not interfered with.

2. The Prevention of Recurrent Deformity.—In the child this implies the restoration of muscle balance across the joint. This can be achieved by reinforcing the paralysed group or by weakening the non-paralysed muscles, although in practice the methods are often combined.

Reinforcement of the Paralysed Group.—This may be achieved by *tendon transfer* in which the tendon of a normally acting muscle is inserted in a new site while preserving the origin of the muscle and its nerve supply thereby altering its mode of action. If this is to be undertaken there are certain principles which must be observed.

(i) The transferred tendon should itself be working to full power. Tendon transfer results in a significant loss of strength so that if the tendon of a partially paralysed muscle is transferred it may be reduced to total impotence in its new situation.

(ii) Deformity in the joint must have been previously eliminated. Tendon transfer alone cannot be relied upon to correct an established deformity.

(iii) The action which is expected of the transferred tendon should be as close as possible to its original action to facilitate re-education. This is of particular importance in the lower limb where the powers of re-education are limited.

(iv) The path of the transferred tendon should be as direct as possible.

(v) The transferred tendon should if possible be re-inserted into bone rather than into another tendon.

Weakening of the Non-paralysed Group.—This is the only method available if tendon transfer is not possible. It must always be remembered that a flail joint will not deform, so that if only one muscle is left acting across a joint muscle balancing will be impossible and it is better to eliminate all activity. Methods for weakening an active group are:—

(*a.*) *Tenotomy.*—Spontaneous reconstitution is possible so that if permanent loss of power is required it is better to resect a length of tendon.

(*b.*) *Elongation.*—Z-elongation of a tendon results in a considerable but not total loss of power.

Both of these procedures may be indicated for correction of the deformity and it is often convenient to carry out the rebalancing procedures at the same time.

Denervation.—Division of the motor nerve to the overacting muscle is sometimes indicated (for example, the soleus and adductor group).

3. The Re-establishment of Joint Stability and the Restoration of Function.—Joint stability may already have been achieved by tendon trans-

fer together with some restoration of function, but if not, then the choice lies between arthrodesis of the joint, which is normally possible only in the adult, or external splintage which is the solution usually adopted in children until they are old enough for bony procedures. The methods commonly employed in the lower limb are as follows:—

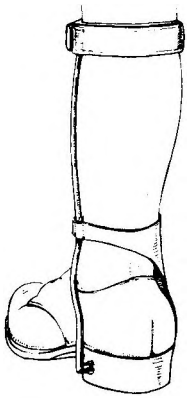


FIG. 320.—Outside iron and inside 'T' strap to control valgus deformity.

Subtalar Instability.—Below knee iron and T strap (fig. 320). The iron is usually placed on the side of greater weakness. Thus if the foot tends to valgus an outside iron and inside T strap is normally used. In many instances it is better to use a double iron. When skeletal maturity of the foot is attained (about 12 years) arthrodesis of the subtalar complex (triple fusion) can be undertaken (fig. 321) and the appliance discarded. Stabilisation at an earlier age can be achieved by means of an extra-articular graft between talus and calcaneum inserted across the sinus tarsi (Grice). Since this does not interfere directly with joint cartilage the disturbance to growth is minimised.

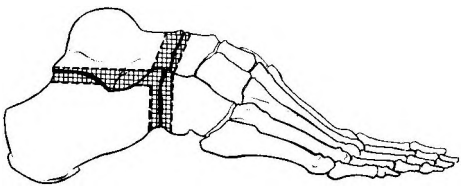


FIG. 321.—Shaded areas denote the bone to be removed in triple arthrodesis (triple fusion).

Ankle Instability.—A double leg iron is used. If there is a tendency to equinus this is resisted by a torsion bar in the heel or a toe raising spring. An appropriate stop can be incorporated if there is calcaneous deformity. A cosmetically more attractive solution for drop foot has become available more recently with the development of new plastic materials. This consists of a flexible back splint which is worn inside a normal shoe. Ankle arthrodesis is usually inadvisable and definitive correction of equinus or calcaneous deformity of the foot is usually obtained at the subtalar level by a modification of triple fusion.

Knee Instability.—This can be controlled by a double leg iron extending up to a thigh corset and may be provided with an unlocking device to permit sitting. Again these are gradually being replaced in suitable cases by more acceptable plastic orthoses[1]. The stability of the knee is dependent on the quadriceps muscle and if this is paralysed the patient cannot normally stand without a brace. If the knee is entirely flail, however, some patients can achieve stability by a trick action in which the knee is forced into hyperextension (fig. 322). Note that in this position the ankle is in some equinus. Arthrodesis of the knee is seldom indicated for paralysis.

[1]Orthosis = a form of splint.

David Stephen Grice, 1914–1960. Professor of Orthopædic Surgery, University of Pennsylvania School of Medicine, Philadelphia, U.S.A.

Hip Instability.—This is usually caused by paralysis of the glutei. Loss of the abductors results in a lurching gait which may be improved by the use of sticks but if the gluteus maximus and hamstrings are paralysed the patient is usually unable to stand upright without the trunk falling forwards. In these circumstances the bracing may need to be carried above the waist. Some patients with flail hips are able to stand by hyperextending the joints relying on the strong anterior capsule of the joint to support them. Arthrodesis is never indicated for paralysis affecting the hip.

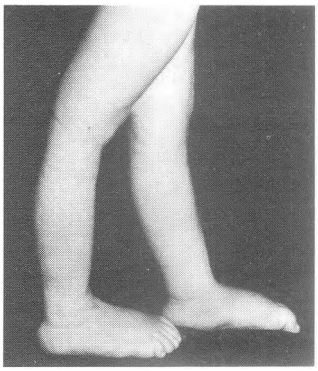


FIG. 322.—Complete quadriceps palsy in the right leg. The patient is able to stabilise the knee by bracing it back into hyperextension.

Gluteal paralysis creates a special problem for the hip if it arises within the first few years of life. In the presence of functioning adductor and flexor groups dislocation is likely and must always be looked for when there is flexion and adduction deformity.

Tendon Transfer.—The use of tendon transfer in the prevention of deformity has already been mentioned and these procedures may also be used for the restoration of function. The very fact that a joint has been arthrodesed may liberate certain muscles for this purpose.

On the whole tendon transfers are very satisfactory in the upper limb where each muscle appears to have separate cortical representation so that rapid re-education can be expected. In the lower limb, however, there appears to be central representation of whole joint movements rather than of individual muscles so that re-education of a single muscle within a group is more difficult. The expectation of useful function from a lower limb transfer is therefore much less than in the upper limb particularly if it is expected to act in a different phase of the gait than formerly and the ability to convert shows much individual variation. The chief value of such transfers is probably the prevention of deformity although certain operations, such as the lateral repositioning of the tibialis anterior and forward transfer of tibialis posterior through the interosseous membrane can usually be relied on to produce worthwhile function.

PARALYSIS FOLLOWING PERIPHERAL NERVE INJURIES (p. 504)

Approximately 18 months after nerve division irreversible changes will have occurred in the motor endplates unless re-innervation has occurred, so that further recovery cannot be expected after this time and secondary reconstruction may be indicated. The procedures which are commonly carried out are as follows:—

Circumflex Nerve.—Loss of shoulder abduction is the complaint and a useful range of this can be restored by arthrodesing the shoulder providing that the scapular rotators are intact. It must be remembered that this will entail a loss of rotation of the arm.

Musculocutaneous Nerve.—The brachioradialis may provide sufficient flexor power at the elbow to make reconstruction unnecessary but if not, additional flexor power can be obtained by advancing the origin of the forearm flexors further up the humerus (Steindler).

Radial Nerve.—Various combinations of tendon transfers are available

Arthur Steindler, 1878–1959. Professor of Orthopædic Surgery, State University, Iowa City, Iowa, U.S.A.

depending on the level of the lesion but at least one wrist flexor should be preserved to prevent hyperextension. A suitable combination for a high lesion is as follows:—

Pronator teres into extensor carpi radialis, longus and brevis.

Flexor carpi ulnaris into long finger extensors.

Palmaris longus into long thumb extensor.

Median Nerve.—In the more common low lesions of the nerve the main disability results from the sensory loss and weakness of opposition. The latter may be improved by tendon transfers, the simplest of which employs the flexor sublimis tendon to the ring finger. The direction of pull of the tendon is made appropriate by causing it to pass first through a pulley constructed on the ulnar side of the wrist. In high lesions the disability is much greater but some improvement in hand flexor function can be achieved by transfers using the brachioradialis and one of the wrist extensors.

Ulnar Nerve.—The disability from a lesion of this nerve is often slight and reconstruction may not be called for. However, paralysis of the adductor pollicis results in weakness of the pinch grip which may be reinforced by tendon transfer usually with one of the radial wrist extensors which has been suitably prolonged.

Brachial Plexus.—A wide variation in the pattern of residual paralysis can result from this and some of the above procedures may be appropriately combined. A total unrecovered paralysis results in a flail arm which is usually best treated by amputation through the humerus, together with the arthrodesis of the shoulder. If the scapular rotators are preserved some abduction at the shoulder will then be possible and may be used to activate a prosthesis. The decision to carry out this procedure should not be too long delayed or the patient will have become permanently adapted to the one-armed way of life.

Lateral Popliteal Nerve.—Many patients are content to control their foot drop with a toe-raising spring (p. 316) or orthosis but some active dorsiflexion may be achieved by a transfer of the tibialis posterior through the interosseous membrane on to the dorsum of the foot. Alternatively, a triple fusion operation may be performed, removing the greater part of the under surface of the talus (Lambrinudi). As a result when the foot is in 'neutral' the ankle is in maximum plantar flexion so that further footdrop is impossible.

MENINGOMYELOCELE (see also p. 486)

This is the commonest of the severe forms of spina bifida and is almost invariably accompanied by some paralysis in the lower limbs which is often extensive and comprises a mixture of both upper and lower motor neurone types. Deformities are often present at birth particularly flexion adduction contractures at the hips, which may be dislocated, and deformities of the foot (fig. 323). These are not to be regarded as separate associated abnormalities; they are secondary deformities and usually correspond with the

Constantine Lambrinudi, 1889–1943. Orthopaedic Surgeon, Guy's Hospital, London.

distribution of the paralysis. Without treatment progressive deformity corresponding with the imbalance can be expected.

Management.—The management follows general principles with the object of providing straight limbs with plantigrade feet at the same time preserving joint mobility and the important ability to sit comfortably. Certain special features should be mentioned.

FIG. 323.—Child with meningomyelocele. There is fixed flexion and adduction of the hips, extension of the knees and calcaneous deformity of the feet. Voluntary innervation was absent below the fourth lumbar segment.

Foot Deformity.—This has the first priority in the orthopædic management. Corrective splintage may be used as in non-paralytic talipes but caution must be used since the feet are anæsthetic. In very resistant cases it may be wise to perform early division of the deforming tendons. Once the deformity has been corrected muscle rebalancing should be carried out if possible but in many instances there will be insufficient muscles under voluntary control to do this. Spastic muscles should not be used for transfer and in these circumstances it may be better to abolish all muscle activity in the foot and to support it in a caliper until 11 or 12 years of age when it can be stabilised by triple fusion.

Hip Deformity.—Gluteal palsy is common in this condition. Where the flexor and adductor groups are strong the hips will either be dislocated at birth or will dislocate in the first few months. By the end of the first year about half of all untreated children with the condition will have dislocated hips. The deformity should be reduced as soon as possible by open tenotomy of the adductors, holding the hip reduced by some form of splintage in abduction and extension. As soon as the child is old enough (about 1 year) the muscle imbalance must be corrected. The adductors have already been weakened and the flexors must now be treated similarly by dividing the tendon of the iliopsoas which is the most important deforming force. At the same time an attempt may be made to reinforce the missing abductors and extensors by transferring the iliopsoas tendon to the back of the greater trochanter (Sharrard.)

Rotation and other deformities at the hip may be corrected by femoral osteotomy at any age since the epiphyses are not interfered with.

CEREBRAL PALSY (Spastic paralysis)

This condition which presents in childhood is the result of intracranial damage involving the cerebral hemispheres or the basal ganglia. It sometimes results from a congenital malformation of these structures, whilst in others the damage is acquired either during birth or soon after. The result in all is a varying degree of limb paralysis of the upper motor neurone type

William John Wells Sharrard, Contemporary. Orthopædic Surgeon, Sheffield Royal Infirmary, Sheffield, England.

in which spasm is prominent, together with difficulties in co-ordination (ataxia) and uncontrolled purposeless movements (athetosis).

The pattern is very varied and one or all limbs may be involved. The commonest pattern is of a symmetrical spastic paresis of the lower limbs with a tendency to flexion and adduction of the hips with flexion of the knees and equinus of the feet. In the upper limb there is typically flexion of the wrist and fingers with adduction of the thumb and pronation of the forearm but ataxia and athetoid movements are often present in addition.

The mechanism of deformity and disability is very complex and is by no means exclusively due to the overactivity of the spastic muscles. Some muscle groups may exhibit normal tone and yet be significantly weakened and, particularly in younger children, the limbs may be held in particular attitudes due to the persistence of primitive reflexes such as the tonic neck reflex. Many children have serious difficulties with balance and orientation in space and these may have an over-riding effect on their locomotor performance.

Management.—Physical therapy is important, particularly in the reinforcement of weak muscle groups by exercise. Passive stretching of muscles and splintage may help to lessen deformity but their limitations should be recognised. Severe fixed deformity can only be corrected by surgery.

When operations are planned the objectives must be clear. Deformity may require correction in order to assist with the child's general management as for example when tight adductors interfere with dressing and toilet care. If, on the other hand, improved function is the aim then this must be realistic, taking into account the child's I.Q., overall muscle power, co-ordination, etc. Failure to do this will lead to unduly ambitious and unnecessary surgery.

The objects of treatment are as before: the elimination of deformity and the restoration of muscle balance, but in the latter tendon transfer can play only a limited part because of the difficulty in finding wholly normal muscles to transfer. Rebalancing therefore has to be confined to procedures which weaken the stronger groups.

Hip.—Adduction deformity and spasm is the commonest problem and if it interferes with walking or toilet care is an indication for adductor tenotomy.

Adductor Tenotomy.—A skin crease incision is made close to the inguinal ligament and the tendons of adductor longus, brevis and gracilis divided through their tendinous parts as close to the pelvis as possible. It may also be necessary to divide the pectineus and even part of adductor magnus. Both branches of the obturator nerve should be identified. If spasm is prominent it may be justifiable to divide the anterior branch but the posterior branch must be preserved. If there is also flexion deformity the tendon of the iliopsoas can be divided or elongated through the same wound. The legs are immobilised in an abduction and extension cast for 3 weeks.

Flexion and adduction predominance predisposes to hip subluxation which will proceed to full dislocation over a number of years, and strict radiological vigilance is required in its presence. Adductor/abductor imbalance may be restored by weakening the adductors by adductor tenotomy and anterior branch neurectomy and in this way the tendency to dislocate controlled. Once dislocation has occurred restabilisation is extremely difficult. Another common problem at the hip is produced by internal torsion of the femur

tending to an ungainly intoeing gait. This may require a rotation osteotomy for its correction.

Knee.—Flexion deformity is the usual problem and is best dealt with by Z elongation at the hamstring tendons behind the knee.

Foot.—Equinus of the heel is extremely common and the child walks on his toes. Sometimes the contracture involves only the gastrocnemius in which case the deformity disappears on flexing the knee. In these circumstances division of the gastrocnemius part of the Achilles' tendon is required. If the entire group of calf muscles is involved an elongation of the Achilles' tendon is indicated. This must be done cautiously as overcorrection may lead to intractable calcaneous deformity.

Upper Limb Deformities.—The results of surgery in the upper limb are often disappointing usually due to deficient sensation and proprioception. If these are intact there may be a place for a flexor slide operation to reduce finger flexion contracting and for procedures to eliminate the common 'thumb in palm' deformity.

POLIOMYELITIS (Infantile Paralysis)

This is an acute febrile disease caused by a neurotropic virus whose permanent effect is an irregularly distributed flaccid paralysis caused by injury to the anterior horn cells. New cases of the disease have been practically eliminated in the United Kingdom as a result of mass immunisation. Occasionally cases of paralysis present without a clear history of the acute infection. These are recognisable by the distribution of the flaccid paralysis and the wasting which is almost invariably asymmetrical, involving particularly the proximal part of the lower limbs and the spine and trunk musculature. Sensory disturbance is absent.

Management.—In the initial and recovery phases of the disease orthopædic management is confined to simple splintage to minimise deformity, but after 2 years or so no further recovery can be anticipated and definitive reconstruction can begin. Although almost any pattern of paralysis is theoretically possible a number of deformities occur with some regularity.

Foot.—Pes equinus due to paralysis of the dorsiflexors of the foot. This can be corrected in childhood by a Z elongation of the Achilles' tendon possibly accompanied by a posterior capsulectomy of the ankle. Anterior transfer of tibialis posterior will help to control recurrence.

Pes calcaneus due to calf paralysis.—This can be treated by a transfer of the peronei and the tibialis anterior through the interosseous membrane to the heel. This is useful in preventing deformity but will probably provide no useful active function. Triple fusion will be required eventually in most cases (fig. 321).

Knee.—The ability to stabilise a knee with a paralysed quadriceps has already been mentioned (fig. 322), and if present represents a strong contraindication to correction of equinus deformity of the foot which may result in a loss of power to hyperextend the knee. If the knee flexors are strong it will

varus of the foot is the commonest problem and this can very satisfactorily be dealt with by elongation of the Achilles' tendon and a division of the tibialis posterior through the same incision. Hamstring tenotomy may sometimes be needed for knee flexion deformity. Upper limb surgery may also be occasionally indicated.

SENSORY LOSS

This has consequences for both skin and the bones and joints.

Skin.—Because of the lack of protective sensation ulceration is likely particularly in weight bearing areas of which the classical illustration is the penetrating ulcer over the metatarsal heads (fig. 64). The latter is particularly liable to occur if there is accompanying equinus deformity from associated motor loss. In the fingers sensory loss is of particular significance for, apart from the liability to damage, an insensitive finger will not be used even if its motor function is perfect. All attempts at finger reconstruction therefore must make provision for restoration of some innervation. If nerve repair is impossible sensation to the pulp may be restored by a pedicle graft of innervated skin from the tip of another finger, the pedicle including an intact digital neurovascular bundle.

Neuropathic Joints.—Tertiary syphilis was formerly the commonest cause of this condition but as this disease disappears an increasing proportion have become due to other causes including syringomyelia, peripheral neuritis (e.g. diabetes) and the rare condition of congenital indifference to pain.

Clinical diagnosis is usually easy due to the remarkable combination of a joint showing obvious gross disorganisation with deformity, swelling and grotesque hypermobility together with remarkably little pain (fig. 324)—the so-called Charcot joint.

Function is often astonishingly well preserved. The accompanying features of the underlying disease will also be present. Radiography shows a grossly disorganised joint with destructive features and irregular areas of new bone formation (fig. 325).

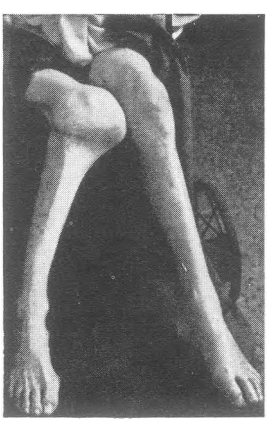


FIG. 324.—Charcot's disease of the right knee. *(Dr. Worster-Drought, London.)*

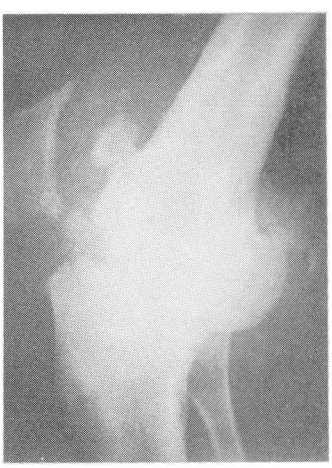


FIG. 325.—Charcot's knee joint. Hypertrophic type.

Treatment.—Although arthrodesis would appear to be the logical treatment this is often difficult to achieve in this condition and it is usually sufficient to stabilise the joint with a suitable appliance—a brace in the lower limb or a moulded leather support in the upper limb.

Jean Martin Charcot, 1825–1893. Physician, La Salpêtrière, Paris. Described tabetic arthropathy in 1868.

again be impossible to hyperextend the joint, when elongation of the hamstring tendons may be helpful.

Hip.—If the paralysis occurs in infancy and affects the abductors, subluxation or dislocation may occur. After dividing the adductors transfer of the iliopsoas to the great trochanter to act as an abductor (Mustard) will prevent progressive subluxation and may also improve the Trendelenburg gait. There is a tendency for the femoral neck to become valgus in these circumstances, further unstabilising the hip. Varus osteotomy may therefore be indicated.

Leg Length Discrepancy.—This may amount to many centimetres, so some correction is required. The normal limb may be shortened by driving staples across the growth plates of the upper tibia and lower femur so that normal growth is slowed, or in an adult the bone shortened by excising a segment of diaphysis. Alternatively the short limb may be lengthened up to 6 cm. by dividing the tibia or femur and producing gradual distraction with skeletal fixation and a screw apparatus. This procedure is contraindicated above the age of 12 years because of the risk of non-union. Whenever possible, the lengthening should be carried out in the tibia where the risk of complications is very much less than in the femur.

Scoliosis.—Muscle imbalance around the vertebral column may produce severe scoliosis with rotary changes as in idiopathic scoliosis (p. 285). However, the curve usually remains a good deal more mobile than in the latter condition. In its most severe form there is a collapsing spine so that the patient cannot even sit up and respiration is embarrassed.

Treatment.—Treatment follows the same lines as in idiopathic scoliosis with correction usually in plaster casts followed by fusion to maintain the correction attained. It should be recognised that this represents a conflict with the basic rules of management since the muscle imbalance remains uncorrected, but there is no alternative in this situation. It is not surprising therefore that there is often recurrence of deformity.

DISSEMINATED SCLEROSIS

This is a slowly progressive disorder resulting from patchy demyelination of the central nervous system. It results in progressive spastic paralysis, usually of the lower limbs. There is a tendency to deform and in particular to develop flexion of the knees and flexion and adduction of the hips. Treatment should therefore be prophylactic with the use of splints and passive joint movement. Unfortunately late cases are often seen and although no growth forces are involved deformity is often severe as it may represent the end result of many years of neglect.

The knees may be treated by tenotomy of the hamstrings followed by serial plasters to increase extension. Flexion and adduction of the hips may be so extreme that surgery in the groin is physically impossible. In these circumstances division of the psoas in the pelvis through a retroperitoneal approach with an intrapelvic division of the obturator nerve will make the situation more manageable. Tendon transfer is never indicated in this condition.

HEMIPLEGIA FOLLOWING A CEREBROVASCULAR ACCIDENT

The pattern of deformity here is very similar to that in cerebral palsy but since the patients are adult it is more easily controlled particularly if physical therapy and splintage are instituted early enough. Occasionally fixed deformity of sufficient severity to require surgery is encountered. Equino-

William T. Mustard, Contemporary. Orthopædic Surgeon, Hospital for Sick Children, Toronto, Canada.

CHAPTER 22

DISORDERS OF THE FOOT

THE common causes of pain in the adult foot, excluding RA and OA secondary to injury, are set out in fig. 326.

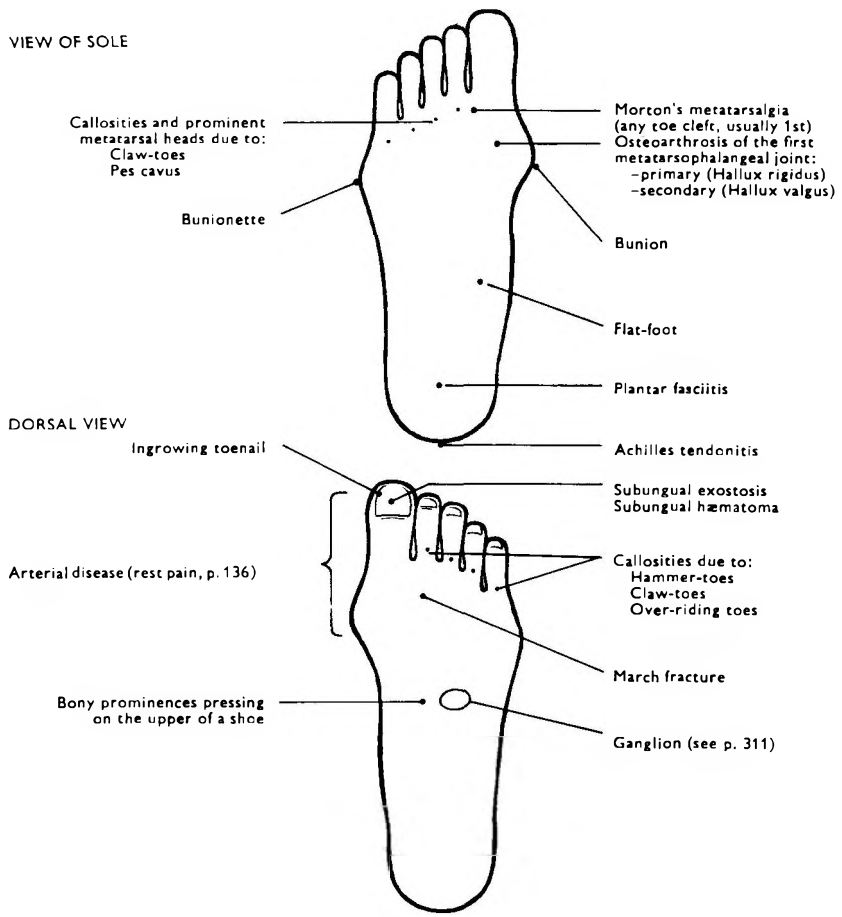


FIG. 326.—The causes of pain in the foot.

Flat-foot (*syn.* Pes Planus)

At birth the foot is flat, and the normal arch is only acquired when the infant stands. The arch is maintained in adult life by the shape of the bones of the foot, by their connecting ligaments and by the action of the muscles of the calf and sole. Flat-foot in adult life (fig. 327) may be due either

to the collapse of once normal arches, or to the persistence of the infant shape.

It is often difficult confidently to attribute a complaint of pain in the foot to anatomical pes planus. Under conditions of active service in first-class infantry regiments, for example, great variations in the shape of the foot, including flat-foot, are observed in unselected groups of men not complaining of painful feet. On the other hand patients will be found complaining of significant pain in the foot who have well-formed arches.

The presence of anatomical 'flatness' is not the most important aspect of pes planus, and two other physical signs should be sought in order to decide whether a patient's symptoms can be attributed to pes planus as such. These are: (1) limited mobility of the tarsal joints and (2) localised tenderness. In general, only patients with painful limitation of movement at the sub-talar, mid-tarsal, or tarso-metatarsal joints are likely to have a mechanical disorder in the foot. Similarly, consistently localised tenderness in one part of the foot is strong evidence of a local source of organic pain.

There are 4 varieties of flat-foot:

1. **Infantile.**—This is physiological.

2. **Peroneal Spastic Flat Foot.**—An uncommon, painful condition which presents insidiously in adolescence. The foot is everted and hence the medial arch appears flat. The tendons of the peronei and long toe extensors can be seen standing out apparently in continuous contraction. Attempts passively to invert the foot are resisted by these muscles and are painful. In about 50% of cases special radiological views of the foot reveal a congenital bar of bone between the talus and the navicular, but in the remainder no abnormality is to be found. In these, the ætiology is unknown.

Treatment is unsatisfactory. Under general anæsthesia a below-knee plaster cast is applied with the foot plantigrade (muscle relaxation may be required to allow the foot to be placed in this position). The cast is worn for 2 to 3 months. Persistent recurrence, especially if a talo-navicular bar is present, may require subtalar fusion or excision of the bar.

3. **Idiopathic Adult Flat-foot.**—This is the commonest form of symptomatic flat-foot in the adult. It may be the end result of repeated episodes of 'foot-strain': an ill-defined, uncommon condition in which the foot becomes acutely painful following excessive use. More commonly, the condition is gradually progressive and presents with aching pain in the foot after long-continued standing. It is seen most commonly in people such as waitresses and hairdressers who spend long hours standing (rather than walking). The medial longitudinal arch gradually collapses causing pain centred in the medial part of the sole. Local tenderness may be found in the sole under the apex of the arch, probably because the plantar ligaments are over-stretched. Pain is provoked if these ligaments are stressed by dorsiflexing the forefoot. In later stages the foot becomes painless and the gait shuffling and inelastic. In its final stages this type of foot can be painless, though rigid and flat.

Treatment.—Treatment is only indicated if the flat-foot is symptomatic: 'prophylactic treatment' is often prescribed (mistakenly) in schoolchildren. The pain of 'foot-strain' is relieved by rest. Its prevention may require arch supports or a change of occupation. Chronic flat-foot can only be

treated during the years when the foot is progressively breaking down. At this stage, arch supports moulded to the corrected shape of the foot may bring symptomatic relief but imply an acceptance of the disability. Exercises to improve the function of the intrinsic muscles of the sole and of the toe flexors may prevent further deterioration if the patient co-operates.

When the foot has finally collapsed, little can be done to improve its appearance or function and the patient should, therefore, be advised to accommodate his life to his feet by taking a sedentary job.

4. **Traumatic Flat-foot.**—This is caused by fractures which abolish the longitudinal arch. Fractures of the os calcis may produce a severe flat-foot with a rigid sub-talar joint. Fractures involving the sub-tarsal and mid-tarsal joints may lead to degenerative changes in these joints and hence to pain, the severity of which is out of proportion to the flatness of the foot.

Pes Cavus (*syn.* Claw-foot)

Pes cavus is an uncommon condition in which there is an increased concavity of the arch of the foot, so that the instep is unduly high (fig. 327). It is typically accompanied by clawing of the toes.

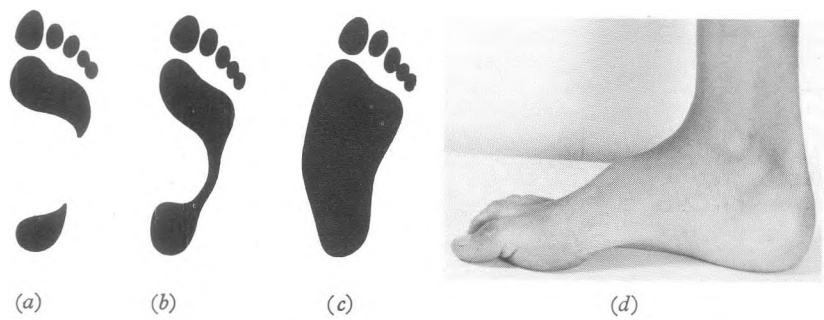


FIG. 327.—Imprints of the foot in: (*a*) Pes cavus. (*b*) Normal foot. (*c*) Pes planus. (*d*) The appearance of pes cavus.

Pes cavus is sometimes caused by neurological disease, for example poliomyelitis or Friedreich's ataxia, but in most cases it is idiopathic. The condition can perhaps be explained on the basis of weakness of the intrinsic muscles of the foot. These muscles flex the metatarsophalangeal joints and extend the interphalangeal joints. Without this mechanism the pull of the long flexors of the toes may cause the toes to assume the claw position and the foot to 'bunch'. Another factor which perhaps causes the deformity of pes cavus is shortening of the plantar fascia, which can be felt as a tight band stretched across the arch.

Treatment.—The treatment of pes cavus is unrewarding.

In childhood the foot is very rarely symptomatic so that the child is often not taken to a doctor or else is taken only because the parents have difficulty obtaining shoes. In such feet intrinsic exercises may be tried and, if unsuccessful, the relatively minor procedure of subcutaneous plantar fasciotomy carried out. It is difficult to justify more major procedures in a foot which is symptomless and may always remain so.

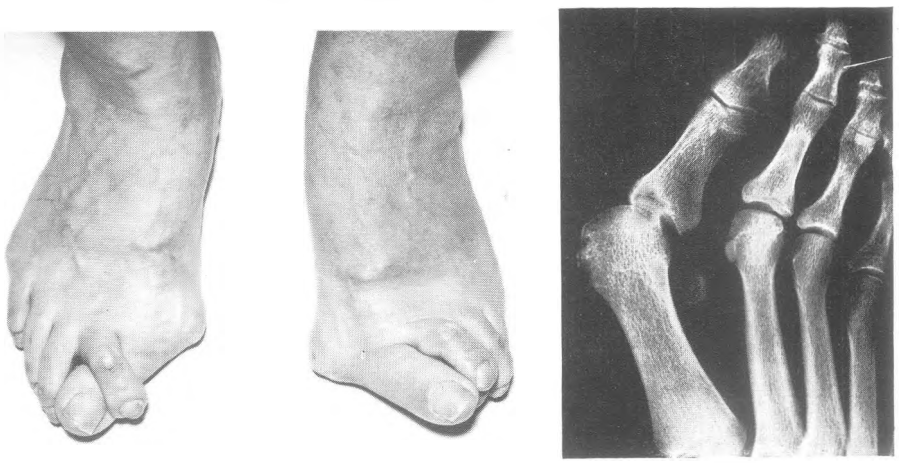
Nikolaus Friedreich, 1825–1882. Chief of the Medical Clinic, Heidelberg, Germany. Described this form of ataxia in 1863.

Fortunately many adult patients with pes cavus are able to play games and walk long distances. Such symptoms as are present are usually due to painful callosities under the metatarsal heads due to the ' high loading ' to which a small area of skin is subjected in this shape of foot and to the difficulty of getting a shoe which fits. Therefore before considering surgical treatment of the anatomical deformity, the surgeon must satisfy himself that the patient has sufficient disability to warrant surgery and that such disability as he has cannot be corrected by making his life more sedentary and by moulding insoles to support his high arch, thus relieving weight from his metatarsal heads. In practice surgical treatment is rarely required. If it is, in adolescents or young adults with a supple foot it may take the form of an attempt to abolish the ' tie-bar ' of the plantar fascia by Steindler's operation in which the plantar fascia and all the muscle attachments are erased from the lower surface of the os calcis by an open operation. The long extensor of the great toe may be transplanted to the neck of the first metatarsal to elevate this bone and so reduce the arch. In older adults with more rigid feet the metatarsals may be shortened and elevated by osteotomies through their shafts.

The treatment of claw toes in the absence of pes cavus is considered on p. 331.

Hallux Valgus

Hallux valgus means lateral deviation (valgus) of the big toe (figs. 328 and 332). It is initiated by wearing footwear which is too narrow for the fore-



(*a*) (*b*)

FIG. 328.—(*a*) The appearance of the feet with hallux valgus. A bunion is present in both feet, a bunionette only in the right. Note the variation in over-riding of the second toes: in both there is a callosity on the dorsum of the proximal interphalangeal (pip) joint. (*b*) X-ray of the foot with hallux valgus and metatarsus primus varus.

foot and which, therefore, tends to force the great toe laterally. Because women's shoes normally have a narrower forefoot than do men's, hallux valgus is much more common in women, especially the middle-aged. An abnormally broad forefoot predisposes to the condition and may be caused by a

Arthur Steindler, 1878–1959 (*see footnote, p. 317*).

congenitally varus position of the first metatarsal (metatarsus primus varus) (fig. 328(*b*)). Once the valgus position of the great toe has developed, it tends to be progressive, because the direction of pull of the extensor and flexor hallucis longus tendons further increases the deformity by a ' bowstring ' mechanism.

The following conditions are often associated with hallux valgus:

1. An exostosis on the medial side of the head of the first metatarsal, probably caused by pressure on the periosteum.

2. A *bunion*, which is an inflamed adventitious bursa, produced over the prominent head of the first metatarsal by pressure and friction. Infection of the bursa (to produce suppurative bursitis) is common and the metatarsophalangeal joint of the big toe may be secondarily infected. A similar lesion, called a ' *bunionette* ', may develop over the lateral aspect of the head of the fifth metatarsal (fig. 328(*a*)). The skin over a bunion may ulcerate if neglected.

3. Osteoarthrosis of the first metatarsophalangeal joint secondary to the malalignment of the proximal phalanx.

4. *Over-riding* or *under-riding* of the second toe by the first. In the latter condition (fig. 328(*a*)) a painful callosity may develop on the dorsum of the second toe. The second toe may be displaced laterally by the hallux to under-ride or over-ride the third (fig. 328(*a*)).

The patient may complain of an unsightly deformity, but more commonly the presenting symptom is pain caused either by pressure, infection or osteoarthrosis.

Treatment.—The conservative treatment of hallux valgus is unsatisfactory. As the patient is almost invariably a woman, it is usually useless to advise the wearing of shoes with low heels, wide fronts and straight inner borders since no such shoe is available in ordinary shoe shops, and since such shoes are unsightly. A small rubber pad can be worn in the cleft between the first and second toes, but this also demands the use of a wide-fronted shoe.

Most young adults with hallux valgus will eventually require operative treatment no matter what conservative techniques are tried, but it is important not to operate merely for cosmetic purposes if the foot is reasonably comfortable. A possible exception to this rule is hallux valgus associated with significant metatarsus primus varus and without degenerative changes in the metatarsophalangeal joint: in such a foot a ' prophylactic ' osteotomy of the first metatarsal so as to reduce the varus is worthwhile. The most satisfied patients after operation for the correction of hallux valgus are those in whom severe pain (as well as gross deformity) were present preoperatively: the indication for surgery in hallux valgus is pain.

Operation.—Local removal of the bunion together with the underlying osteophytes, without interference with the metatarsophalangeal joint, is not a satisfactory operation because symptoms almost always recur, and a more radical operation is later required. Exceptionally this operation may be performed in young people if the valgus deformity is slight, but the exostosis prominent.

The standard operation for hallux valgus is excision arthroplasty of the base of the proximal phalanx (Keller's operation) or of the head of the first metatarsal (Mayo's

William Lordan Keller, 1874–1959. U.S. Army Surgeon, who became Head of Department of Surgery at the Walter Reed General Hospital, Washington, D.C., U.S.A. Described this operation for hallux valgus in 1904.
Charles Horace Mayo, 1865–1939. Surgeon, The Mayo Clinic, Rochester, Minnesota, U.S.A. Described this operation for bunion in 1908.

operation). The Keller procedure in younger patients can be augmented by osteotomy of the base of the first metatarsal to abduct the first metatarsal and close the enlarged gap between it and the second metatarsal. The corrected position is maintained by inserting excised bone to hold the osteotomy open on the medial aspect.

After the operation, at least 3 months will elapse before the foot will tolerate an ordinary shoe, and the patient may not feel the full benefit of the operation for as long as 6 months.

Hallux Rigidus

This condition occurs as two distinct varieties, both more common in men. In both the metatarsophalangeal joint of the great toe is stiff and painful.

FIG. 330.—Hallux rigidus.

(1) The adolescent type is due to synovitis of the metatarsophalangeal joint following injury, and is associated with muscular spasm. The condition is often secondary to minor trauma, for example stubbing the toe or repeatedly kicking a football with the point of the toe instead of the dorsum of the foot. There are no radiological changes. It is relieved by wearing a metatarsal bar 2 cm wide and 1·25 cm thick (fig. 329). Conservative treatment may fail to arrest the condition which then progresses gradually to present as the adult type.

FIG. 329.—Metatarsal bar on shoe.

(2) The adult type is nothing more than monarticular osteoarthrosis (fig. 330), sometimes precipitated by injury. The limitation of movement is due to capsular fibrosis, deformation of the articular surfaces, and the interlocking of osteophytes.

Treatment.—The condition may be treated by fitting a metatarsal bar or by operation. The Keller procedure is the most widely employed operation, although arthrodesis is perfectly satisfactory in men, especially if hyperdorsiflexion has developed at the interphalangeal joint of the great toe.

Hammer-toes

This condition, which may affect the second, third and fourth toes, consists of hyperextension of the metatarsophalangeal and distal interphalangeal joints, and flexion of the proximal interphalangeal joint (fig. 331). Callosities form over the bony prominence on the dorsum of the proximal interphalangeal joint, and in long-standing cases adven-

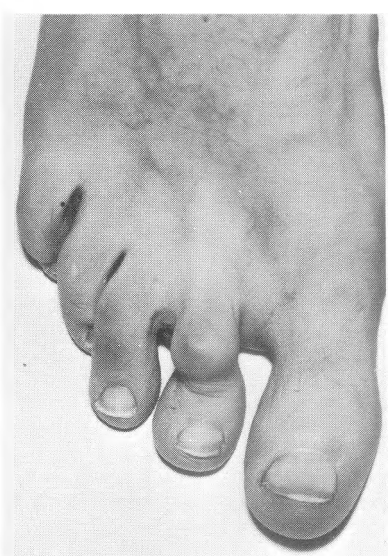


FIG. 331.—Second hammer-toe.

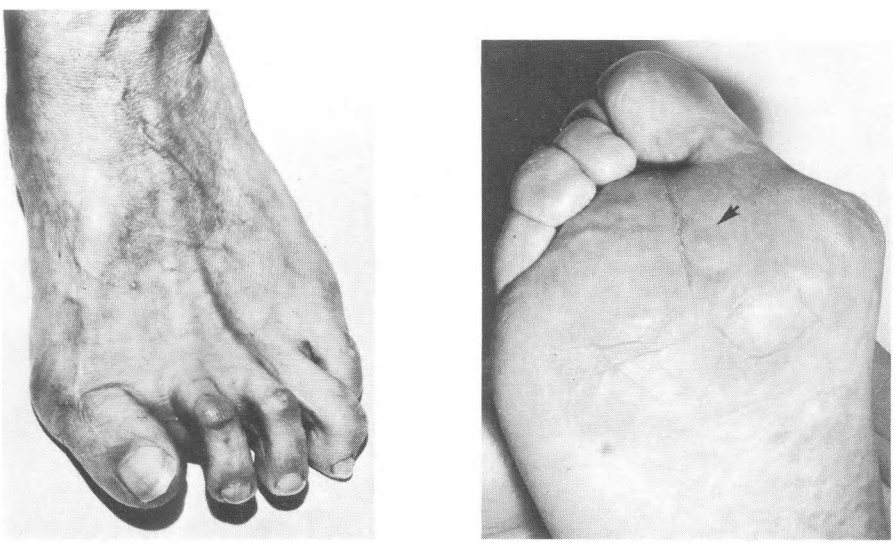
titious bursæ develop. The tendons and ligaments become secondarily contracted.

Hammer-toes sometimes develop from overcrowding, either by small or pointed shoes, or as a result of hallux valgus. Often there is no obvious cause.

Treatment consists of correcting any predisposing cause and wearing a corrective splint or a corn plaster. If the deformity is established, arthrodesis of the proximal interphalangeal joint by the 'spike' method (Higgs) combined with extensor tenotomy and dorsal capsulotomy of the metatarsophalangeal joint is the treatment of choice. The spike operation consists of drilling the base of the middle phalanx and impaling it on the shaft of the proximal phalanx after shaping the condyles into a spike. Alternatively, the proximal phalanx may be excised.

Claw-toes

This condition differs from hammer-toes only in that (1) the toe pads do not contact the ground when the patient stands and (2), probably as a consequence of the loss of contact, the distal interphalangeal joint is flexed, not extended (fig. 332(*a*)). Because the toe pads are not weight-bearing, an increased proportion of the body-weight is borne by the corresponding metatarsal head. Because the metatarsophalangeal joint is hyperextended, often to the point of dorsal dislocation, the toes are prominent dorsally and press on the upper of the shoe causing painful callosities on the dorsum of the toe and downward pressure on the metatarsal head. These two factors tend to



(*a*) (*b*)

FIG. 332.—(*a*) Second and third claw-toes. Note that the toe pads do not touch the ground. (*b*) Plantar callosity (arrowed) under the second metatarsal head. Note that the pad of the second toe does not touch the ground.

compress the skin and soft tissues between the ground and the metatarsal heads and so cause pain and callosity formation in the skin of the sole. Even-

Sydney Limbrey Higgs, 1892–1977. Orthopædic Surgeon, St. Bartholomew's Hospital, London.

tually the fibro-fatty tissue under the metatarsal head atrophies or is displaced distally so that the metatarsal head becomes subcutaneous and easily palpable in the sole. A callosity then develops in the skin under the metatarsal head which is painful to stand on and tender when pressed (fig. 332(*b*)).

The condition is often associated with hallux valgus and pes cavus but may occur as an isolated entity. Its ætiology is unknown, but it is probably related to the wearing of high heels and to poor function in the intrinsic muscles of the foot. Severe claw-toes may be caused by rheumatoid arthritis affecting the metatarsophalangeal joints.

Treatment consists of exercises for the intrinsic muscles of the foot if the metatarsophalangeal joints are fully mobile. If they are not, the foot can be made comfortable by the provision of well-fitting footwear, an insole to relieve weight from the metatarsal heads, and a corn plaster on the callosity.

If conservative measures fail, the deformity can be corrected by arthrodesis of the interphalangeal joint (see hammer-toes) combined with dorsal capsulotomy and extensor tenotomy of the metatarsophalangeal joint and transfer of the flexor digitorum profundus tendon into the dorsum of the proximal phalanx. Alternatively, the affected toes may be amputated through the metatarsophalangeal joints. If pain under the metatarsal heads is the main complaint, the head of the metatarsal may be elevated by osteotomy of the shaft or one or all the metatarsal heads may be excised. The latter operation is particularly useful in rheumatoid arthritis.

Morton's Metatarsalgia.—This syndome was thought to be due to the development of a neuroma on the interdigital nerve between the metatarsal heads, possibly caused by local compression of the nerve. It is now known that the true cause is intermetatarsal bursitis which does compress the nerve but does not cause a neuroma. The condition is rare and presents clinically with well-localised pain and tenderness between the metatarsal heads, together with paræsthesiæ and hypoæsthesia in the related interdigital cleft.

Treatment is by excision of the bursa.

Plantar Fasciitis.—The ætiology is uncertain. Pain and localised tenderness occur under the heel. The pain is often severe, is felt especially at heel-strike, and interferes with walking. It is probably caused by a tear in the attachment of the plantar fascia to the os calcis, but seems sometimes to be associated with gonoccocal and non-specific urethritis. The pain is not caused, as was once supposed, by bony spurs on the plantar surface of the os calcis.

Treatment consists of advising the patient to avoid walking and standing as much as possible for 6 weeks, fitting rubber heels, and supplying insoles to weight-relieve the tender area and to take tension off the plantar fascia by supporting the medial arch. If these measures are ineffective, local injections of 25 mg. of hydrocortisone and 1 ml. of 1% procaine may bring relief.

Achilles' Tendonitis.—The loose connective tissue in which the Achilles' tendon slides becomes inflamed, thickened and painful. Pain may also originate, as in other forms of tendonitis, from the attachment of the tendon to the bone. The condition is often caused by a period of greatly increased walking or by the wearing of shoes with an ill-fitting heel. Occasionally it appears to be spontaneous.

Treatment consists of rest, if necessary in a below-knee plaster cast, for up to 6 weeks.

Subungual Exostosis.—Occurs on any toe but is most common on the great toe. The toe-nail is lifted from its bed and becomes painful and often infected.

Treatment is by removal of the nail and exostosis.

Congenital abnormalities of foot and toes are described in Chapter 17.

Thomas George Morton, 1835–1903. Surgeon, The Pennsylvania Hospital, Philadelphia, Pennsylvania, U.S.A. Described this condition in 1876.

Achilles, the Greek hero, was the son of Peleus and Thetis. When he was a child, his mother dipped him in the Styx, one of the rivers of the Underworld, so that he should be invulnerable in battle. The heel by which she held him did not get wet and was therefore not protected. Achilles died from a wound in the heel, which he received at the Siege of Troy.

CHAPTER 23

INJURIES TO BONE, CARTILAGE, LIGAMENTS AND TENDONS: GENERAL PRINCIPLES

INJURIES TO BONE (FRACTURES)

TUBULAR BONE

Pathology. A fracture of the shaft of a long bone disrupts the bone matrix, the periosteum and endosteum, the blood vessels in the medullary canal, in the cortex and in the periosteum, and the neighbouring soft tissues. The matrix, being inert, does not contribute to subsequent bone healing, but the blood vessels, periosteum and endosteum, and possibly the osteocytes in the involved bone do so.

As a consequence of the disruption of blood vessels, the soft tissue spaces around the fracture site, the space between the fractured bone ends, and the medullary canal, become filled with extravasated blood which clots to form a hæmatoma as elsewhere in the body. The hæmatoma is invaded by granulation tissue. Macrophages remove the hæmatoma at the apices of the capillary loops of which the granulation tissue is composed. Cells which probably originate from the endothelium of the capillaries lay down a loose connective tissue composed of collagen fibres embedded in a dilute gel of proteoglycan. The periosteum and endosteum, torn by the fracture, may be avulsed from their attachment to the surfaces of the bone. The cells of which these membranes are composed are stimulated, possibly by the act of avulsion, or possibly by contact with humoral agents arising in the medulla, to proliferate and to lay down new bone. Thus at least 2 cell series, one arising from the endothelial cells of the blood vessels and one arising from the periosteal and endosteal cells are at this stage (that is to say approximately 2 weeks after the injury) contributing to connective tissue formation round the fracture site. It is possible that 2 other groups of cells play a similar role. These are: histiocytes which are believed to represent undifferentiated primitive mesenchyme cells present in all connective tissues, and, doubtfully, the osteocytes in the fractured bone itself. Many of the latter cells die as a consequence of infarction of the bone ends adjacent to the fracture, and such cells certainly contribute nothing to the healing process.

As the fracture hæmatoma becomes progressively organised and replaced by loose connective tissue, the bone ends come to be joined to each other but by a tissue which has very little rigidity and very little strength. From approximately 3 weeks onwards, cells around the fracture site not only lay down collagen in a proteoglycan matrix to form fibrous tissue and cartilage, but in addition they calcify the matrix and subsequently ossify it. Thus the tissue connecting the bone ends becomes osseous, and as such is visible radiologically and confers increasing rigidity and strength on the fracture site. The resulting tissue, known as *callus*, is distributed throughout the area once occupied by the fracture hæmatoma, and thus by 4 to 5 weeks after the fracture the bone ends are surrounded by a randomly distributed mass of fibrous tissue, cartilage and woven bone.

The process of vascular invasion by capillary loops, to which the original hæmatoma was subject, continues throughout this period and cells arising from the capillaries continue to lay down bone. Thus the callus, initially randomly organised, comes to be replaced by bone which progressively takes on an Haversian structure.

The original cortex is subject to the same process of osteoclasis and subsequent new bone formation as is the woven bone of the callus. In this way the anatomical dividing line between callus and cortical bone becomes increasingly blurred. Eventually the mass composed of callus and cortical bone is replaced by fully differentiated Haversian bone bridging the fracture site. At this point the bone returns to its original strength and rigidity but its anatomy and radiological appearance are still

Clopton Havers, 1657–1702. General Practitioner and Anatomist, London. Published his lectures on the anatomy of bone in 1691.

not normal. Over the ensuing years, the mass of bone in the medullary canal and surrounding the fracture site is remodelled so that the anatomy of the bone gradually returns towards the normal, although in the adult some internal or external irregularity commonly persists indefinitely.

The process described above occurs both outside the bone (to produce periosteal callus) and within the medullary canal (to produce endosteal callus). In the gap between the surfaces of the fractured cortex itself, a similar callus forms which is eventually transformed into cortical bone indistinguishable from the neighbouring, original cortex. If the fracture is perfectly reduced and then rigidly immobilized within a few days of its occurrence, callus formation does not occur as described above. Instead capillaries invade the dead bone adjacent to the fracture and grow across the fracture gap to fuse with each other. In the same way as described above, cells derived from these vessels both remove and deposit bone so that a living cortex is directly reconstituted. This process is described as Primary Bone Healing. It must be stressed that Primary Bone Healing occurs only after very accurate reduction and rigid internal fixation of the fracture. This method of healing is unlike healing by callus formation in that it is radiologically invisible and much slower (taking months instead of weeks).

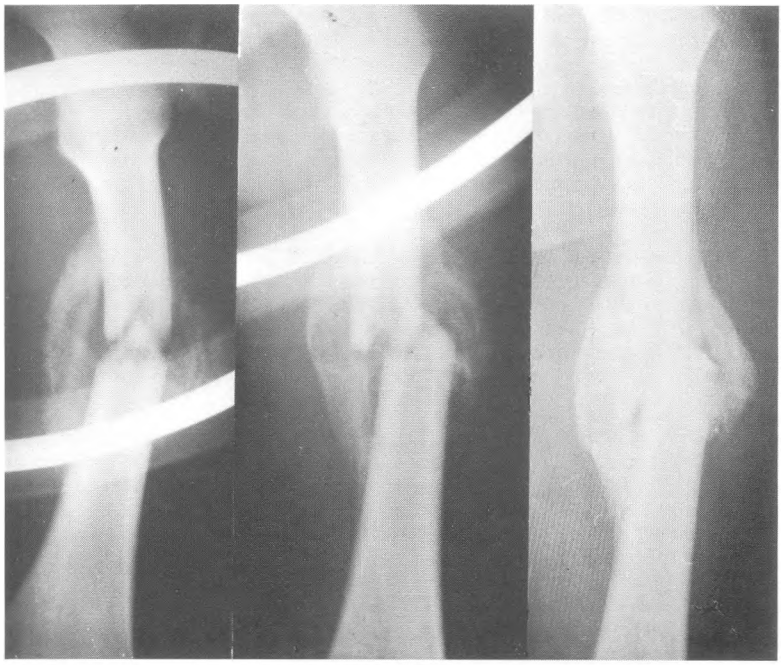
Clinical and Radiological Features

Immediately after the fracture has occurred the patient suffers severe local pain and finds that he is unable to use the injured part. The site of the fracture is tender, and within a few hours swelling and bruising appear. If the bone ends have been displaced in relation to each other, deformity may be visible. It is possible to elicit abnormal movement at the fracture site, but this physical sign should be sought with gentleness since such movements are painful. Crepitus (a sensation of grating when the bone ends are moved against each other) may also be elicited at the fracture site, but should never be sought deliberately since its demonstration adds nothing to the diagnosis and merely causes pain.

Radiologically, the bone can be seen to be fractured and the details of the fracture anatomy can be discerned. The first radiological examination of a fracture is only complete when the whole of the involved bone together with the joints in which it participates have been X-rayed in 2 planes at right angles to each other, since only in this way can the full extent of the bony injury be demonstrated and the presence of associated joint injuries be detected.

As the fracture hæmatoma becomes organised to form fibrous tissue and subsequently woven bone, the abnormal movement originally demonstrable at the fracture site diminishes. Slight movement can, however, still be elicited, stressing the fracture site is painful, and local tenderness persists. At this stage the fracture is described as being ' sticky '. Between the third and sixth week following injury, the calcification and subsequent ossification occurring in the callus become visible radiologically (fig. 333(*a*)) and as this process progresses the fracture site becomes decreasingly tender and increasingly rigid and strong.

By approximately 6 weeks (the exact time varies with the age of the patient and the nature of the fracture—see Table 1) healing has progressed so that (1) no abnormal movement can be detected when the fracture is stressed, (2) stressing the fracture site is scarcely painful, and (3) local palpation pro-



(*a*) (*b*) (*c*)

FIG. 333.—(*a*) Early callus formation. The calcified material does not have a bony texture and bridges the fracture site only as a vague ' cloud '. (*b*) Union. The callus now has a bony texture and bridges the fracture site but the fractured bone is easily visible, distinct from the callus. (*c*) Consolidation. The callus has a bony texture and is no longer clearly distinguishable from the femoral shaft.

TABLE I

In general:

(1) Lower limb fractures take twice as long to unite as upper limb fractures.
(2) Fractures in adults take twice as long to unite as fractures in children.
(3) Transverse fractures take longer to unite than oblique or spiral fractures.
(4) Compound and comminuted fractures are particularly slow to unite.
(5) No fracture unites in less than 3 weeks.

Thus, for example:

(1) An oblique fracture of the humerus in a child takes 3 weeks to unite.
(2) The same fracture in an adult takes 6 weeks,
(3) A transverse fracture of the tibia in an adult takes 12 weeks, and
(4) A comminuted fracture of the tibia in an adult may take 12–24 weeks.

vokes little or no tenderness. At this stage the fracture is described as being ' united '. Radiologically, bone can be seen to bridge the fracture, but since the bone is partly woven rather than Haversian in structure, it does not have a normal radiological texture (fig. 333(*b*)).

As the woven bone of the callus is replaced by normal Haversian bone the fracture site becomes entirely painless irrespective of the patient's activity and the radiological texture of the bony mass surrounding the fracture site gradually becomes identical with that of the rest of the bone (fig. 333(*c*)). Such a fracture is described as ' consolidated ' and at this point the healing process can be regarded as having come to an end.

Mechanism of Injury

A tubular bone may be broken either by direct or indirect violence. For example the tibia may be fractured by direct violence if the bumper of a motor car strikes the bone or by indirect violence if a skier twists his body whilst the ski is held fixed. A bone fractured by direct violence is likely to be broken into several small pieces. Such a fracture is described as 'comminuted'. Indirect violence may be applied to a bone as a twisting force, in which case a spiral fracture results, or as a bending force, in which case a transverse or oblique fracture is produced. Double oblique fractures may occur so as to leave a 'butterfly' fragment separated from the two main pieces of the bone (fig. 334).

(*a*) (*b*) (*c*) (*d*) (*e*)

FIG. 334.—(*a*) A transverse fracture. (*b*) A transverse fracture with an undisplaced butterfly fragment. (*c*) An oblique fracture. (*d*) A spiral fracture. (*e*) A comminuted fracture.

CANCELLOUS BONE

Pathological, Clinical and Radiological Features

The pathological events following a fracture of cancellous bone are similar to those following a fracture of a tubular bone save that now the healing process occurs around a multitude of small fractures—one in each trabecula of the involved bone—rather than between the two large fracture fragments of the tubular cortex. Thus, although the histological events are similar, the clinical and radiological appearances are somewhat different because the fracture callus is spread evenly throughout a honeycomb of cancellous bone

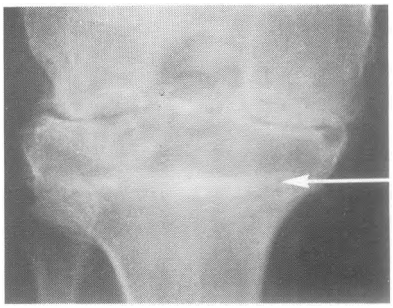


FIG. 335.—A uniting cancellous surgical fracture (an osteotomy) through the tibial condyles. All that is to be seen is a zone of sclerosis (arrow). Compare this with the appearance of the femoral shaft fracture in fig. 333 (*c*).

Clinically, the principal difference is that abnormal motion and crepitus may never be present if the honeycomb structure is interlocked by the chance arrangement of the trabeculæ at the fracture site. Such a fracture is said to be ' impacted '. Radiologically, the callus no longer appears as a bony mass surrounding the fracture site but instead may only be seen as a zone of increased radio-density (known as sclerosis) at the fracture site (fig. 335).

Mechanism of Injury

Cancellous bone may be fractured either by compression or by tension.

Compression fractures may occur for example if a patient falls from a height and lands on his heels: the compressive force travelling up the leg and through the trunk may produce a crush fracture of a vertebral body, of the tibial or femoral condyles, or of the os calcis. Such fractures are often impacted.

Traction injuries only occur in cancellous bone to which a ligament or tendon is attached, for example the medial malleolus. The bone is stressed by traction on the ligament. The fracture represents an avulsion of the bony attachment of the ligament and as such it is functionally equivalent to a rupture of the ligament. Such fractures are never impacted.

DISTURBANCES OF NORMAL HEALING IN BONE

Delayed and Non-Union

The times by which fractured bones have usually united have been listed in Table 1, but these times are often exceeded. When a bone still displays abnormal movement, pain when the fracture site is stressed, and tenderness

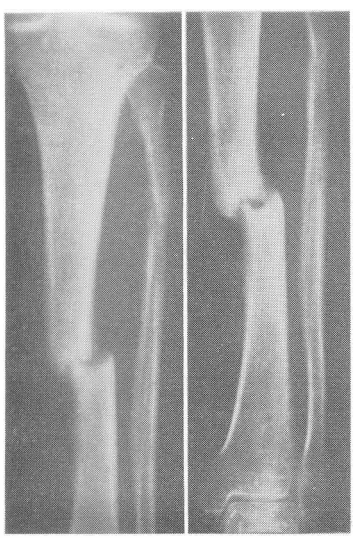


FIG. 336.—Delayed union—no sclerosis.

FIG. 337.—Non-union—sclerosis.

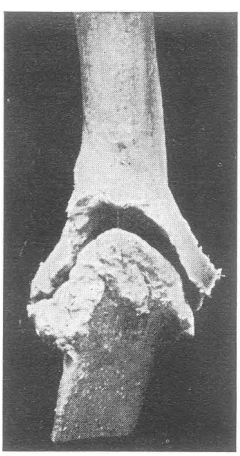


FIG. 338.—A pseudarthrosis following an un-united fracture of the shaft of the humerus.

over the fracture site at a time when union should have occurred, the fracture is said to display ' delayed union '. Because ossification of the callus has been delayed, the callus in such a bone may be invisible or poorly visible on an X-ray and, if visible, it may not bridge the fracture site (fig. 336). A fracture displaying delayed union may continue slowly to heal until finally it unites. The healing process may, however, come to a halt before union has occurred, so that abnormal movement and a radiologically visible fracture site persist permanently. Such a fracture is said to display ' non-union '. Histologically a fracture displaying non-union is bridged by fibrous tissue, the bone ends are sclerotic and the medullary canal may be obliterated (fig. 337). The fibrous tissue bridging such a fracture site may be dense and short, so that the bone ends are in close apposition to each other and little abnormal movement occurs between them. On the other hand the scar may be a long one and may even contain within it a cavity occasionally displaying all the tissue elements of a synovial joint. Such an abnormal joint is known as a ' pseudarthrosis ' (fig. 338). Persistent abnormal movement at a fracture site interferes with the function of a limb and the fibrous tissue in an un-united fracture is usually painful when stressed. Non-union is therefore usually unacceptable to the patient. Occasionally, however, a very firm fibrous scar forms so that, if the bone is not heavily loaded, as in the upper limb, the result is indistinguishable clinically from bony union.

The Causes of Delayed Union

1. **Excessive movement** at the fracture site during the healing phase may produce delayed union because the callus bridging the fracture site is constantly ' re-fractured ' by the movement occurring in the healing tissue. It may be speculated that shear, for example produced by torsion, is more harmful in this respect than is bending at the fracture site. Certainly minimal bending movements, such as occur in a fractured rib, are perfectly compatible with normal healing and may even promote it.

2. **Infection** of the fracture hæmatoma usually leads to non-union and, if it occurs, is a disaster (p. 339). Healing fails because the cellular elements which are required for the production of bone at the fracture site are diverted to the production of pus.

3. **A poor blood supply at the fracture site** interferes with healing since the invasion of the hæmatoma by blood vessels is slow or absent. Cells with osteogenic potential are therefore slow to appear in the fracture hæmatoma. Furthermore there is some evidence that a low oxygen tension produced by a poor blood supply interferes with the production of bone by undifferentiated mesenchyme cells so that even such osteogenic cells as are present may fail to produce bone, and instead produce cartilage or fibrous tissue.

The anatomy of the fractured bone may result in a complete loss of the blood supply to one of the fracture fragments. For example in fractures of the neck of the femur, the vessels ascending in the neck of the femur to the femoral head may be ruptured by the fracture. The femoral head may then be completely infarcted, so that the healing process does not consist of the

union of two vascularised bone fragments but, instead, involves the bridging of the fracture site from one side only, followed by revascularisation of the whole of the infarcted fragment. As might be expected, such injuries are either slow to unite or fail to unite altogether.

4. **Interposition of soft tissues** between the fracture fragments may so separate them that it is physically impossible for them to unite. For example, at the medial malleolus the tendon of tibialis posterior may come to lie between the medial malleolus itself and the rest of the tibia, or in spiral fractures of the femur a spike formed by one of the fracture fragments may become impaled in the muscles of the thigh. Such a fracture cannot be reduced (i.e. the bones ends cannot be accurately apposed) and healing by bone is obviously impossible.

5. **Systemic disease,** for example uræmia developing as a consequence of renal failure secondary to blood loss following injury, may interfere with healing generally.

FIG. 339. — Mal-united fractures of the radius and ulna. This patient was unable to pronate and supinate the forearm.

Mal-Union

A fracture which unites in a position of deformity is said to be *mal-united.* For example a long bone shaft fracture may unite with significant angulation at the fracture site. This complication of normal healing is due to imperfect reduction. Mal-union usually, but not always, results in impaired function and cosmetic loss (fig. 339).

Impairment of Function After Union

Healing may be imperfect in the functional rather than the anatomical sense. For example *adhesion formation* may occur between the fracture site and the muscles or other soft tissues adjacent to the bone even though reduction has been perfect, so that movement at the adjacent joints is limited and muscle power reduced.

Following a fracture, there is normally an increase in the blood flow through the injured area associated with a loss of bone matrix (i.e. osteoporosis, p. 302) in the skeleton adjacent to the fracture. Immobilisation also causes these changes so that if an injured limb has to be immobilised, they may be severe. Occasionally very marked osteoporosis, associated with thickening of the soft tissues, vascular stasis, pain and joint stiffness complicates a fracture irrespective of its severity. This syndrome is

FIG. 340. Sudeck's atrophy.

known as *Sudeck's Atrophy* (fig. 340). Its ætiology is unknown but it is thought to have either a reflex vascular basis or possibly to be due to the extreme reluctance of some patients to use the limb after the removal of splintage, a reluctance which accentuates the changes normally produced by immobilisation. The patient should be encouraged, with supervision in a physiotherapy department, to use the limb. No other specific treatment is available.

Compound Fractures

A compound fracture is one in which a laceration in the skin (or mucous membrane) communicates with the fracture hæmatoma. Bacteria can therefore reach the fracture site. It may not always be possible to tell preoperatively whether a laceration adjacent to a fracture actually communicates with the fracture hæmatoma or not, but a combination of a laceration and a fracture should always be regarded as a compound fracture until surgical exploration proves otherwise. If the laceration proves not to communicate with the fracture, the latter may be termed 'technically compound'.

Lacerations associated with fractures may be caused either by a fracture fragment penetrating the skin from its deep surface (in which case the fracture is said to be compound 'from within'), or by the object causing the fracture, e.g. the bumper of a car, breaking the skin as well (in which case the fracture is said to be compound 'from without'). The chances of significant bacterial contamination of the fracture are greater in the latter and hence fractures which are compound from without have a poorer prognosis.

A break in the skin surface may be due to skin death from ischæmia rather than to a laceration. Fractures of subcutaneous bones with displacement may lead to pressure on the deep surface of the skin by a bony fragment and thus endanger the circulation to the skin. The reduction of such a fracture is urgent in order to preserve the skin. Injuries causing fractures by direct violence may crush the blood vessels in the skin but this may not be obvious at the time of the first examination. During the following 2 to 3 weeks the skin may slough and if this possibility is not borne in mind, a fracture may be encased in a plaster-of-Paris splint in which the skin may slough unnoticed.

It must be assumed that any fracture in which the overlying skin is broken is contaminated with bacteria, either from the skin itself or from the surrounding environment. Such bacteria remain dormant in the fracture hæmatoma for a few hours following injury but soon thereafter start to multiply so that their number rises rapidly as time goes by. An inflammatory response develops locally and the fracture, from being contaminated, becomes infected. The chances of sterilising the fracture hæmatoma become increasingly remote as the number of bacteria at the fracture site rises. Therefore the treatment of a compound fracture is a matter of urgency (p. 347).

TREATMENT

The management of a fracture can be considered under two headings: (1)

Paul Hermann Martin Sudeck, 1866–1938. Professor of Surgery, Hamburg, Germany. Described this type of osteoporosis in 1900.

the management of the patient as a whole, and (2) the local management of the fracture itself.

General Management of the Patient

(1) *Pain.*—All fractures are painful and it should never be forgotten that the immediate responsibility of the physician is to relieve pain. This can be done by local splintage and by analgesics such as morphia, pethidine, paracetamol or salicylates.

(2) *Blood Loss.*—All fractures are associated with some blood loss. This may be negligible, but in fractures of the major long bones, the spine, and the pelvis it can be considerable. Its loss, however, may not be immediately obvious. For example a patient suffering a fracture of the pelvis or of the shaft of the femur can lose 2 litres of blood into the surrounding tissues without any obvious swelling or bruising. Such blood loss must be replaced.

(3) *Associated Injuries.*—Fractures are commonly associated with other injuries, for example fractures of the pelvis may be associated with injuries to the bladder, or fractures of a long bone shaft may be associated with injuries to the blood vessels or nerves in the limb. The possibility of such injuries must always be borne in mind since they may be missed unless they are deliberately sought at the time of the first examination.

Local Management of the Fracture

Locally, the management of a fracture is directed both to the injured bone itself and to the associated soft tissues.

So far as the bone is concerned there are two objectives: (1) *Reduction* of the fracture (strictly, reduction of the deformity at the fracture site) and (2) *Immobilisation* of the fracture. Reduction restores the normal anatomy. Immobilisation—(*a*) maintains the reduction whilst healing takes place and (*b*) promotes union.

FIG. 341.—An undisplaced fracture of the shaft of 3rd metacarpal. The fracture is splinted by the neighbouring soft tissues and bones: neither reduction nor external splintage is required.

For the soft tissues the objectives are: (1) to maintain muscle power and (2) to promote joint mobility. To some extent there is a conflict between these objectives and that of immobilising the fracture.

Severed blood vessels and nerves, and lacerations of the skin, require specific treatment.

Reduction of the Fracture.—Restoration of the normal anatomy at the fracture site is termed ' reduction ' of the fracture. If the original injury did not produce significant displace-

ment, as for example in the fracture of the metacarpal displayed in fig. 341, no reduction is required. Should reduction be required it may be accomplished in one of two ways: (1) by closed manipulation or (2) by open operation.

Closed manipulation has the great advantage that the skin remains intact so that there is no possibility of the fracture site becoming infected. Therefore if closed reduction is possible, it is, in general, the treatment to be preferred. The patient is either given a general anæsthetic or the fracture site is rendered analgesic by local infiltration or by i.v. local anæsthesia distal to a tourniquet. Anæsthesia has the double effect of producing muscle relaxation so that the manipulation becomes easier, and of making the manipulation painless for the patient. The bone ends are then manipulated in an attempt to restore the normal anatomy. The specific manipulations which are required for the reduction of common fractures are described in the following chapters, but the same general principle underlies the closed manipulation of all fractures: the surgeon seeks to reverse the mechanical events which led to the production of the original fracture. Thus an understanding of the mechanism of the injury is essential if closed manipulation is to be used intelligently.

For the reduction of some fractures, e.g. fractures of the shaft of the humerus or the femur, a formal manipulation is unnecessary and traction (see p. 343) alone suffices.

Open operation (see p. 344).

Immobilisation.—The object of immobilising the fracture is (1) to maintain the alignment of the bone produced by reduction whilst healing occurs and (2) to promote union. Immobilisation may be provided in one of four ways:

(1) Externally by: (*a*) splints
or (*b*) traction
} Conservative treatment

or (2) Internally by: (*a*) adjacent soft tissues
or (*b*) surgical implants (p. 348).
} Operative treatment

Conservative Treatment

Splints.—The strength and rigidity required in the splint are proportional to the forces applied to the fracture. Thus immobilisation is usually achieved partly by the use of splintage and partly by deliberately reducing the loads on the fracture site, for example by resting an injured arm in a sling or by walking only on the uninjured leg and crutches.

The most commonly used material for external splintage is plaster-of-Paris applied by wrapping the limb in a wet plaster-impregnated bandage. The plaster is allowed to set so as to hold the fracture in the desired position. Because it is almost impossible to obtain sufficient ' grip ' on a single bone to withstand the forces imposed on the bone by contraction of the muscles attached to it, a plaster splint must always extend proximally and distally to include the joints adjacent to the fracture site. This consti-

tutes a significant disadvantage to the use of plaster-of-Paris since it often means that when the plaster is removed the adjacent joints are stiff, the muscles weak, and the blood flow diminished. Other disadvantages include the facts that wounds cannot easily be treated in plaster, that adjustments in the reduction may be difficult to achieve, that the material is relatively heavy and radio-opaque and that it is not always easy to maintain a reduction whilst the plaster bandage is being applied. Having said this, plaster remains overall the safest, cheapest and most convenient form of splintage available. It has however one serious hazard attached to its use.

The Hazard of plaster-of-Paris Splinting.—Plaster-of-Paris is inelastic. When a plaster is applied soon after an injury it is to be anticipated that swelling will occur at the fracture site. Such swelling cannot distend the plaster and it will, therefore, result in a gradual rise in pressure in the soft tissues at the fracture site. This rise in pressure may occlude the venous return from the limb producing venous engorgement distally and, shortly thereafter, an interruption in the arterial flow to the limb. If the arterial inflow to a limb is obstructed for an hour or more, the muscles and nerves become infarcted and subsequently replaced by fibrous tissue. This sequence of events represents a significant hazard to the use of plaster-of-Paris and if it is allowed to occur, it constitutes a disaster the consequences of which are very much more grave than any which could possibly follow total failure to treat the fracture. Thus if plaster-of-Paris is to be applied to a fresh fracture, it is essential that the plaster should not completely surround the limb. This may be achieved either by applying a slab of plaster to the limb leaving a wide gap on one side of the limb, or by splitting a completely encircling plaster (fig. 342). Any such plaster should be well padded with wool so that swelling may occur without compressing the soft tissues and the limb should be elevated after its application.

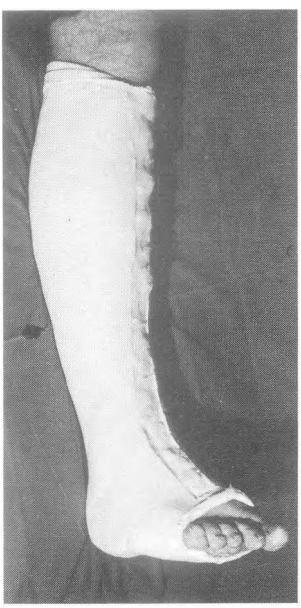


FIG. 342.—A split plaster-of-Paris cast.

The clinical features of an impaired blood flow are progressive in nature. The circulation in the exposed skin distal to the fracture should be examined carefully in the hours following the application of the plaster and any sign of an interruption of the blood supply (i.e. pallor or poor capillary filling in the limb) should be regarded as an indication that the blood supply is possibly impaired. In this event the whole plaster should be opened and all padding should be separated so that the skin is visible throughout the length of the split in the plaster. Should significant swelling occur, the patient will notice increasing discomfort and pain both at the fracture site

and distal to it. These symptoms should also be regarded as an indication for easing a plaster encircling a limb. If the arterial inflow actually becomes interrupted so that the blood supply to the muscles is impaired, active movement of the muscles will become impossible (e.g. the fingers will be stiff) and attempts to stretch the involved muscles by passive movement will be painful. The pulse is weak or absent. *The detection of these signs is an urgent indication for the removal of plaster* and, if this does not improve the circulation, for exploration of the appropriate main artery.

If infarction is allowed to occur so that fibrosis of the muscles and nerves ensues, the resulting fibrous tissue contracts to produce deformity, stiffness, and weakness at the involved joints. This syndrome is known as *Volkmann's Ischæmic Contracture* (fig. 343).

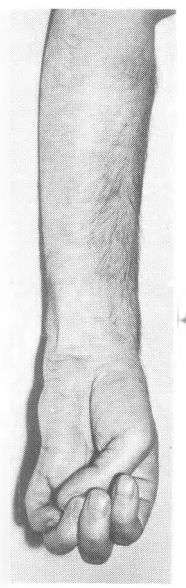


FIG. 343.—Volkmann's ischæmic contracture following a supracondylar fracture of the humerus in childhood.

External Skeletal Fixation.—As an alternative form of splintage to plaster-of-Paris, Steinmann or other pins may be passed through the bone proximal and distal to a fracture and the pins then connected to a special metal frame. This method of fixation is particularly useful if the fracture is compound so that access is required to a wound. It provides very rigid fixation, is easily adjustable and is light. The disadvantage is the necessity of using pins to transfix the bone: these expose the bone to the risk of infection and are difficult to pass in other than subcutaneous bones such as the tibia. In practice therefore the method is most useful for compound fractures of the tibia (fig. 344).

Traction.—Fractures may be immobilised by exerting traction on the involved limb. The way in which traction immobilises a fracture may be understood by likening the fracture and the adjacent joints to a bicycle chain. Each link of a bicycle chain is mobile and can be displaced on its neighbour if the chain is not under tension. If tension is applied in the long axis of the chain, the chain will straighten and movement of one link on another becomes difficult or impossible. Exactly the same principle applies to a fracture when traction is applied in the long axis of the limb.

Traction may be exerted on a limb by either (1) attaching an inelastic bandage (special materials are available) to the skin, and then a weight to the bandage (*skin traction*) or, (2) passing a metal wire or pin through the bone and attaching weights directly to the bony skeleton in this way (*skeletal traction*). The former technique has the advantage that there is no possibility of infecting the bone but it has the disadvantage that only a small weight (about 2·25 kg.) can safely be attached to the skin. The weight used must be sufficient to overcome the shortening and deforming force of the muscles acting across the fracture site and thus skin traction is almost only of use in infants and children where the strength of the muscles can be

Richard von Volkmann, 1830–1889. Professor of Surgery, Halle, Germany. Described ischæmic contracture in 1881.
Fritz Steinmann, 1872–1932. Surgeon, Berne, Switzerland. Described a pin for skeletal traction in 1907.

FIG. 344.—The Day External Fixation Frame being used to treat a severe compound fracture of the tibia. The frame stabilizes the fracture whilst allowing full access to the soft tissues.

matched by a pull of 2·25 kg. In adults, skin traction is only used as a temporary measure and if a fracture is to be treated definitively by traction, skeletal traction is required.

Plaster-of-Paris or the weight of the limb itself may be used to provide traction in the upper limb. For example, a fracture of the shaft of the humerus may be treated in a collar-and-cuff sling which simultaneously rests the limb and, by taking advantage of gravity acting on the elbow and forearm, exerts traction on the fracture site. The traction force may be increased by bandaging a slab of plaster-of-Paris to the arm. Such a slab not only provides more effective traction but also provides splintage at the fracture site.

The Adjacent Soft Tissues.—Certain bones, for example, a single metacarpal shaft (fig. 341), are satisfactorily splinted by adjacent soft tissues, provided the violence of the injury has not displaced the bones and in so doing torn their soft tissue attachments. No additional splintage is required for such fractures.

Operative Treatment

Indications.—Operation may be required for a fracture because:

(1) reduction is necessary but is impossible by closed means;

(2) having achieved reduction by closed means, it is impossible (or dangerous for the general health of the patient) to maintain it by external splintage; or

(3) the management of associated soft tissue injuries is difficult or

William H. Day, Contemporary. Engineer.

impossible in the presence of external splintage and requires rigid immobilisation of the fractured bone.

Closed reduction may be impossible if soft tissues are interposed between the fracture fragments. The fracture must then be exposed at operation to allow the soft tissue to be extracted and the bone ends brought into apposition. Alternatively it may be impossible to reduce a fracture by closed means because the surgeon cannot obtain any purchase on one of the two fracture fragments. For example in fractures through the anatomical neck of the humerus associated with anterior dislocation of the head of the humerous, closed manipulation is impossible since the head of the humerus is inaccessible save by operation. A third situation in which adequate reduction cannot be achieved by closed manipulation is that represented by fractures around joints in which there may be several small fracture fragments whose accurate replacement is essential if normal joint function is to be restored. Examples of such injuries are certain fracture-dislocations around the ankle and depressed fractures of the tibial condyles. Often the fragments cannot be manipulated at all, and certainly they cannot be manipulated accurately, through the skin.

The maintenance of reduction by external splintage may be impossible because no satisfactory form of splint is available to immobilise a small fragment or to overcome muscle pull. Small fragments requiring immobilisation are commonly adjacent to joints (as, for example, the head of the femur, fragments around the ankle, or the tibial condyles). Although it may be possible to immobilise such fragments by external splintage, this will involve immobilisation of the whole joint. Unfortunately a combination of severe injury and prolonged immobilisation may produce serious joint stiffness and, therefore, in skilled hands, better results may be obtained by internal fixation of the fracture so as to allow early mobilisation of the joint. The opportunity provided by the internal fixation of any fracture to mobilise muscles and joints early after injury, constitutes an argument for operating even when closed means of management are available, such as for example in fractures of the shafts of the femur or tibia. This advantage has to be balanced carefully against the disadvantages, of which the most serious is the risk of sepsis. Unless the surgeon is experienced, closed means are preferable.

The inability of external splintage to overcome muscle pull is illustrated by transverse fractures of the patella, after which the quadriceps muscle may pull the proximal fragment of the patella upwards into the thigh. The two fragments can be pressed together through the skin under anæsthesia, but even if the knee is then splinted in full extension, the gap between the fracture fragments opens again as soon as the quadriceps muscle contracts. Thus the restoration of normal anatomy is impossible unless the two fragments of the patella are fixed together at operation.

The maintenance of reduction may be possible by closed means but may be hazardous to the general health of the patient. Thus in fractures through the intertrochanteric region of the femur it is possible to immobilise the fracture either by applying a plaster-of-Paris spica or by the use of traction, and

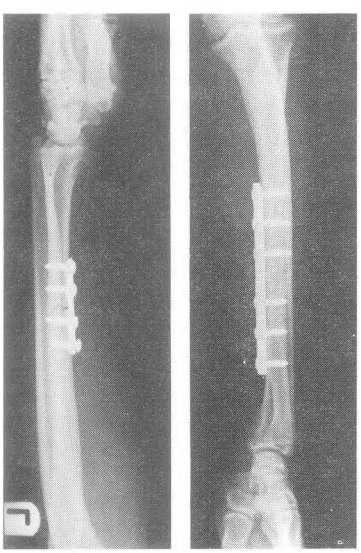
both of these techniques are employed in the young. In the elderly, the prolonged immobilisation in bed which either form of treatment requires is hazardous to the patient's general health since pressure sores, deep vein thrombosis, pulmonary embolism, and hypostatic pneumonia are all common complications of bed rest in this age group. Thus for this fracture in the elderly, operation is required to produce internal fixation of the fracture so as to allow the patient to be mobilised out of bed.

Fractures associated with damage to vital soft tissues may be best treated by internal fixation of the fracture so as, for example, to allow a ruptured artery to be repaired.

Methods of Internal Fixation.—A variety of devices is available to splint fractures internally. The fracture fragments may be wired or screwed together (figs. 363 and 389) or united by one, or rarely two, onlay plates screwed to the bone (fig. 345), or by a nail passed down the medullary canal (fig. 395). Special devices are available for certain fractures, such as the pin and plate used for fractures of the proximal femur (fig. 399).

If it is felt that a fracture fragment cannot survive usefully—a decision which is sometimes made, for example, in connection with the head of the femur after fractures of the femoral neck—the bone may be removed and replaced with a prosthesis (fig. 397).

It is argued that, if internal fixation is to be used at all, it should be as strong and as rigid as possible since: (1) this allows the joints of the limb to be mobilised early, free of external splints, and (2) there is evidence that union is promoted by rigid fixation of the fracture. Strong, rigid implants have been designed for this purpose and techniques have been developed for enhancing the quality of fixation by applying the implant in such a way as to compress the fracture site (fig. 345). In long bone shaft fractures this is done with an onlay plate, a technique known as ' compression plating '.



(*a*) (*b*)

FIG. 345.—(*a*) Inadequate internal fixation: the onlay plate is too slender, the screws are too few, too near the fracture site and one is loose. (*b*) Adequate internal fixation: the onlay plate is more rigid and is firmly held to the bone by well-placed screws.

Internal fixation is technically difficult in comminuted fractures and in fractures through cancellous bone.

Local Management of Soft Tissues

Muscles may be injured at the time of the fracture or they may be affected indirectly by adhesion formation between themselves and the fracture site. Their function must be preserved as far as possible by encouraging the patient to contract all the muscles in the injured limb statically, and regularly to move through a full range

all those joints which have not been immobilised. If the muscles are not exercised, they waste and adhere to the bone and other soft tissues, and the venous return from the limb is impaired. Muscular exercise is obviously much easier if the fracture has been neutralised by internal fixation to leave the joints free.

Immobilisation of *joints* produces gradual obliteration of the synovial space with fibrous tissue, and damages articular cartilage. This results in stiffness of the involved joint. Immobilisation of a joint may be an inevitable consequence of immobilising a fracture, but wherever possible it should be avoided. Thus plaster-of-Paris splints should not include joints if their immobilisation is not essential to the immobilisation of the fracture, and any joint which is not immobilised in plaster should be actively exercised.

If the *skin* is lacerated or dead, the fracture may be compound (p. 339). The treatment of such injuries presents special problems.

The Treatment of Compound Fractures.—A compound fracture is a surgical emergency: it must be treated urgently and with skill. Treatment aims at sterilising the fracture site, and consists of (1) wound toilet, (2) the reconstitution of skin cover, and (3) the use of antibiotics.

Wound Toilet.—It is imperative that this operation be carried out carefully, in optimal operating conditions, with a good light. If possible, a tourniquet should be applied to the limb following exsanguination so that the fracture site can be examined thoroughly. The wound in the skin is extended if necessary and the damaged soft tissues inspected. All dead tissue (which if left in the wound would provide a culture medium for bacteria) including the margins of the skin wound are then removed by sharp dissection. Dirt or foreign material is removed from the wound and the wound tissues are washed with sterile saline. At the end of this procedure all remaining bone and soft tissues should be surgically clean, and demonstrated to be viable by releasing the tourniquet to see that the tissues bleed.

Wound Closure.—Wound closure is safe only provided that the patient can be observed closely in the days following operation. If the wound is closed by suture without having been adequately sterilised, infection may develop deep to the suture line. Since the suture line is relatively inelastic, the tension deep to the wound then rises and further soft tissue damage results. The infection is finally very much more severe than if the wound had not been sutured. Therefore, if it is impossible to arrange for close examination of the patient in the early post-operative period, it is safer to pack the wound with sterile dressings and leave it to granulate for 10 to 14 days following operation. At the end of this period the wound can be inspected and either closed by the application of a split skin graft or by secondary suture. If the patient and his wound can be observed post-operatively so that at the first sign of infection the sutures can be removed, the wound should be closed by simple suture provided that this can be done without tension at the suture line. A substantial dead space may be produced by simple suture, and the deep soft tissues should then be closed with removable sutures passing through the skin, deep tissues and the dead space. If suture of the laceration is impossible without tension, the wound should be left open (or, in certain situations plastic procedures may be considered). A sutured wound of any size should be drained by closed suction.

Once the skin wound has healed satisfactorily without infection of the fracture site, the fracture itself may be treated as if it were simple. While the skin is healing the fracture should be reduced and splinted, but splintage should never (save in experienced hands or in the presence of severe associated soft tissue injuries) take the form of internal fixation since the foreign material which must necessarily be used to produce internal fixation hinders sterilisation of the fracture site by the local inflammatory response and by antibiotics. So far as possible, external splintage should

allow for inspection of the wound: external skeletal fixation using a suitable frame is ideal in this context (see External Skeletal Fixation, p. 343).

Antibiotics and Prophylaxis against Tetanus are discussed on p. 21 and p. 31.

Gas Gangrene is discussed on p. 33.

The Treatment of Non-union (p. 336)

If an obvious cause of non-union such as infection is evident, the underlying pathology should be treated. Commonly there is no obvious cause of non-union, and in this event treatment relies upon two factors: (1) absolute fixation and (2) the use of a bone graft.

Absolute fixation cannot be produced by external splintage alone, but may be achieved by internal fixation either with an intramedullary nail or with sufficiently rigid onlay plates combined if necessary with external splintage. The periosteum adjacent to the fracture is elevated and a bone graft is applied subperiosteally around the fracture site without disturbing the fibrous non-union itself. A bone graft, combined only with external splintage, is adequate if only a very little movement is permitted at the fracture site by the fibrous tissue crossing the fracture. The bone graft ideally should consist of the patient's own bone since the cells of the graft then contribute to new bone formation. Cortical bone is relatively ineffective in this context since per unit of mass it contains relatively few cells with osteogenic potential. Autogenous cancellous bone is, therefore, the material of choice and a convenient site for this is the patient's iliac crest. The matrix of the graft acts as a scaffold upon which new bone is formed and may also induce new bone formation. Subperiosteal new bone formation is probably induced by the surgical elevation of the periosteum and the rawing of the subjacent cortical bone as well as by the graft itself.

FRACTURES IN CHILDHOOD

Fractures occurring in children differ from those in adults in 6 important respects: (1) the fractures unite more readily, (2) mal-union can to some extent be corrected by growth, (3) joint stiffness is rare after immobilisation, (4) the fractures may involve the epiphyseal plate and hence interfere with growth, (5) the bone may fracture incompletely, and (6) the patient is often unable to protect himself and to give a history, a combination which gives rise to the Battered Baby Syndrome (see below). The first 3 differences make fractures in children much easier to treat than in adults, and make immobilisation by external splintage almost invariably the method of choice.

Union is almost invariable following all fractures in childhood: a fracture of the neck of the femur is an occasional exception. Not only is union invariable, but it occurs in about half the time required for an adult.

The correction of mal-union by growth seems to occur most readily when the residual angulation occurs about an axis parallel with that of the movement permitted at the adjacent joint. Thus, for example, in supracondylar fractures of the humerus backward angulation of the distal fragment (i.e. extension) can be corrected by growth but a varus deformity (i.e. adduction) cannot. Torsional deformities correct less well than angular.

Joint stiffness is rarely caused by immobilisation in a child. Following the removal of splintage, children quickly use the injured limb and usually regain a full range of movement rapidly and without physiotherapy.

Involvement of the epiphyseal plate in a fracture is not uncommon since the plate is weaker than the bone. Although epiphyseal injuries may lead to a disturbance of growth, they do so surprisingly rarely. The fracture, which often involves the metaphysis, should be accurately reduced but nothing can be done to prevent or to predict a disturbance of growth. Operative reduction should be avoided if at all possible.

FIG. 346.—A greenstick fracture of one cortex of the radius.

Incomplete fractures are called *greenstick fractures*: one cortex may fracture whilst the other bends (fig. 346). The resultant angulation may be hard to correct without completing the fracture.

The Battered Child.—The term ' battered child ' is used to describe a child who has received serious, deliberately inflicted injuries at the hands of his parents or some other adult. The true incidence of this type of injury is not known, but it is undoubtedly common. In one London hospital a case is diagnosed almost every month and for every case diagnosed there must be dozens which are unrecognised. The injuries occur because the child is unable to protect himself, and are unsuspected because he is unable to give a history.

The injured children are usually under the age of 2 years. They are taken to the general practitioner or to hospital, often after a suspicious delay, and the parents are either unable to explain the injuries or suggest some minor accident, such as falling off a sofa, which is quite inappropriate to the severity of the injuries. In more than half the cases there is a history or radiological evidence of previous injury. The commonest injuries are soft tissue bruises and abrasions, burns, fractures of the ribs and long bones, epiphyseal injuries, and sub-dural hæmatoma with or without a fractured skull.

Since this type of child-abuse is repetitive, it is vital, for the safety of the child, to make the diagnosis and take appropriate action as soon as possible. The doctor who first sees the child should be aware of the condition and ready to suspect it. Suspected cases must be admitted to hospital immediately in order to protect the child while the pædiatrician and the medical social worker undertake the investigations necessary to establish the diagnosis.

PATHOLOGICAL FRACTURES

The foregoing discussion has been concerned entirely with injuries to normal bones. Bone may be the seat of a number of pathological processes, for example neoplasm or osteoporosis, which significantly weaken it either generally or locally, and hence make it liable to fracture in the face of trivial forces. Such fractures are known as pathological fractures.

Pathological fractures due to secondary neoplasm should be treated by internal fixation, if the local state of the fracture and the general state of the

patient allow, in order to make the patient more comfortable. Following internal fixation, radiotherapy may arrest the growth of the tumour and allow union to occur.

'STRESS' FRACTURES

High, oft-repeated loads applied to the skeleton may produce lesions known as ' stress ' fractures (but for which a better term would be ' fatigue ' fractures). Typical examples occur in the tibiae of runners training on town pavements, in the femoral necks of newly-inducted infantrymen carrying out fully-laden route marches (see Intra-Capsular Fractures of the Neck of the Femur, p. 404), and in the metatarsals (see march fractures, p. 379, and fig. 376). The bone in question becomes painful and is tender. Radiologically, the fracture, which is nearly always incomplete, is invisible at first but later is revealed by a zone of increased radiodensity due to callus formation. The only treatment necessary is rest.

INJURIES TO ARTICULAR CARTILAGE

(see also Transchondral Fractures p. 298)

Pathology.—Mitosis of human adult chondrocytes does not occur following injury and therefore true healing is impossible. In this sense the chondrocyte is similar to the neurone. Experimental defects involving only the cartilage do not heal: the corresponding lesion in man is only detected as an incidental finding during the exploration of a joint and its fate is presumed to be the same as that in experimental animals. An osseo-cartilaginous defect, produced by a fracture extending through the articular surface, results in bleeding from the bony surface into the joint. The defect fills with granulation tissue arising from the bone and this is eventually converted into fibro-cartilage.

Clinical Features.—There are no specific clinical features of a cartilage injury, but because an injury producing cartilage damage usually produces an effusion or bleeding into the involved joint (hæmarthrosis), swelling and fluctuation in the involved joint usually accompany cartilage damage. In the absence of treatment, some wasting always occurs in the muscles acting on the joint during the weeks after the injury. A loose fragment may fail to gain attachment to the synovial membrane and may then mechanically block movement in the joint, a symptom known as ' locking ' of the joint.

Blood in a joint tends to remain liquid because of the presence of hæmolysins in the synovial fluid, but it eventually clots and subsequently becomes organised into fibrous tissue. This organised hæmatoma adheres to the cartilage surfaces and to the synovial membrane, so that movement in the damaged joint is mechanically limited. Immobilisation—imposed either by pain or in the course of treatment—can be shown, experimentally, to damage cartilage and to lead to the proliferation of fibrous tissue in the synovial cavity. Immobilisation and injury also result in adhesion formation between peri-articular soft tissues such as tendons, ligaments and muscles. Thus articular injuries commonly lead to stiffness in the injured joint.

Because it is rare for defects in the articular surface to be perfectly reconstituted, irregularities persist after these injuries which may eventually result in osteoarthrosis.

INJURIES TO LIGAMENTS AND TENDONS

Pathology.—Ligament injuries may be either simple sprains or ruptures. In a simple sprain some of the collagen fibres of which the ligament is composed are torn but the ligament as a whole is mechanically intact. Blood vessels in the ligament are ruptured so that local swelling and bruising occur. Nerve fibres running in the ligament may suffer a traction injury. In a rupture, the same pathological events occur but to a greater extent, so that the ligament is broken into two and all the blood vessels, collagen fibres and nerve fibres in the involved ligament are divided. Following a rupture, the joint is mechanically unstable.

Tendons rarely rupture, save in pathological circumstances, for example if they become avascular or have been subject to persistent attrition over a roughened bony edge. Traction injuries to a healthy bone/tendon/muscle unit usually produce a rupture at the tendon muscle junction or an avulsion of the tendon from the bone. Tendons, especially those in the hand, may be involved in lacerations anywhere in their length.

Clinical Features.—Injuries to ligaments and tendons heal by the formation of a hæmatoma around the injured structure which subsequently becomes organised to form fibrous tissue. At this stage the divided ends of the ligament are connected by a scar in which the collagen fibres run at random and in which the ends of the original structure can be seen histologically. Such a scar gradually becomes replaced by well-orientated collagen fibres until eventually the histological appearances of the structure have been reconstituted. Whether or not the mechanical properties of the ligament return to normal, depends upon the length of the resulting scar, upon its strength, and upon the extent to which the scar adheres to the surrounding tissues. A tendon, when divided, usually retracts because of the contraction of its parent muscle. Such retraction may produce a gap so wide as to be unbridgeable and as a consequence healing does not occur. If the tendon does not retract, vessels invade the tendon from the surrounding tissues so that the scar tissue which eventually forms, involves both these tissues and the tendon itself. Thus the tendon becomes adherent, its excursion is limited and its function impaired.

Clinically, simple sprains result in two specific physical signs—localized tenderness (usually over the bone/ligament junction) and pain when the ligament is stressed. Ligament ruptures result in three physical signs—localised tenderness, pain when the ligament is stressed and in addition, mechanical instability. The demonstration of mechanical instability is diagnostic of a ligament rupture and in particular distinguishes a rupture from a simple sprain. These physical signs are well illustrated by ligament injuries at the ankle (p. 381) and knee (p. 390). Bruising, local swelling, an effusion into the joint and, with time, muscle wasting are usual, but non-specific, concomitants of these injuries.

Damage to the nerve fibres and mechanoreceptor endings in ligaments, and especially in joint capsules, breaks the postural reflex arcs of which these fibres form the afferent limb. As a consequence the muscles acting across the joint lose their normal postural co-ordination so that in the lower limb, the joint tends to be unstable: for example at the ankle the patient complains that the foot ' gives way '.

If a tendon has been divided, the active movement for which it is responsible is lost. Passive movement remains but may be painful. Loss of active movement may not be obvious clinically if other muscles are available to take over the function of the severed tendon, e.g. the profundus flexor tendons can flex the proximal interphalangeal joints and hence replace sublimis, and all the muscles passing behind the ankle can plantarflex the foot, so masking a rupture of the Achilles' tendon.

Disturbances of Normal Healing.—Failure to heal, the analogue of non-union in bone, may occur in tendons and ligaments if the ends of the divided structure are too widely separated. With lesser degrees of separation, an elongated scar may be produced allowing abnormal movement in the involved joint (in the case of a ligament) or a loss of part of the active range of movement (in the case of a tendon). The collagen fibres in the scar may fail to become well orientated so that the scar is persistently painful when stressed.

Failure to co-apt the ends of the ruptured structure will certainly result in imperfect healing. Absence of tension in the healing tissue may also play a part in poor collagen orientation since it has been shown in tissue culture that collagen fibres tend to array themselves parallel to lines of tension.

Treatment.—If tendons have been lacerated and their ends separated, they will not heal unless they are sutured together. Suture should be carried out as soon as possible provided the sterility of the wound can be ensured. Following suture, the tendon should be immobilised by external splintage of the joints across which it acts for 3 to 6 weeks. The detailed management of tendon injuries in the hand is discussed on p. 211.

Ruptured ligaments may be treated either by immobilisation of the involved joint or by suture. Immobilisation is satisfactory provided that the ends of the ruptured ligaments are lying in contact with each other. Unfortunately in a number of ligaments (for example the medial collateral and cruciate ligaments of the knee) the ends of the ligament may retract: it is then preferable to explore the ligament and suture the ends together.

The joint should be immobilised until it is felt that the ruptured ligament will withstand the loads applied to it by protected movements—usually a period of 3 to 6 weeks. Ideally a sutured ligament should be sufficiently secure to allow such movement to occur immediately post-operatively, but in practice the sutures are usually not secure enough to permit this. In some ligaments, for example the lateral collateral ligament of the ankle, significant separation of the ends of the ligament rarely if ever occurs, and normal movements cause no significant separation of the two halves of the ligament. Such ruptures can be treated by early non-weight-bearing mobilisation without causing material elongation of the ligament as it heals.

Following a ligament injury, the muscles acting across the joint become weak and unco-ordinated and the joint stiff, sequels which are treated by appropriate active exercises.

CHAPTER 24

INJURIES TO THE UPPER LIMB

FRACTURES OF THE BONES OF THE HAND

Fracture of the Terminal Phalanx.—This bone is commonly fractured by crushing injuries. The skin and pulp are also crushed so that the skin is often lacerated and the fracture is compound. Perhaps because these injuries involve a small bone, they are often regarded as trivial. This is a mistake, for if the injury to the terminal phalanx and to the overlying soft tissues does not heal satisfactorily, the finger tip becomes permanently tender to touch and often painful in cold weather. Permanent tenderness of a finger tip is a severe disability: the patient tends to use the other fingers and to guard the affected finger so that it gradually becomes stiff. If the digit involved is the thumb, the situation is even more serious for loss of the thumb is functionally almost equivalent to loss of the arm.

Treatment, if the fracture is compound, consists of careful wound toilet and loose suturing of the skin followed by the application of a pressure dressing. The dressing should be changed at 48 hours and thereafter at regular intervals in order to inspect the skin. A broad spectrum antibiotic and tetanus toxoid are administered. If the skin dies or is lost, it may require replacement with full thickness skin on the pulp or split skin elsewhere on the digit (p. 212). If the fracture is simple, a pressure dressing should be applied until pain has subsided.

Fractures of the Middle and Proximal Phalanx

These fractures may be caused by a direct blow (when the fracture is often comminuted) or by bending or twisting the finger (when the fracture is transverse or spiral). The fracture is usually unstable, and if it is, is angulated forwards. The tendons adjacent to the fracture may adhere to the fracture as it heals and hence limit movement in the finger.

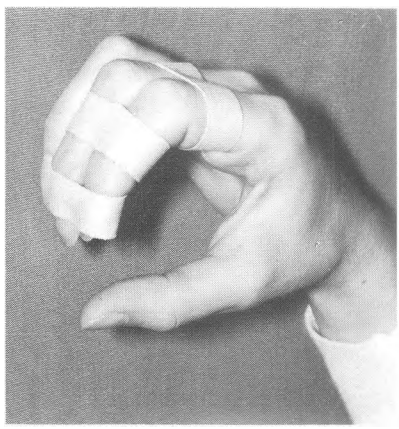


FIG. 347.—The fingers strapped together for undisplaced phalangeal fractures.

Treatment is difficult since it is not easy to maintain reduction and to immobilise the fracture without stiffening the joints of the finger. If the fracture appears to be stable and there is relatively little displacement, the fracture can be splinted satisfactorily whilst allowing movement at the joints of the finger by strapping the finger to its neighbour for 4 weeks (fig. 347). Care must be taken when this is done to ensure that there is no rotational de-

formity at the fracture site, for if there is, the finger will deviate as it flexes so as to interfere with its neighbour. If the fracture is displaced, it is reduced by traction followed by flexion of the finger over a curved aluminium splint applied to the flexor surface of the finger. The aluminium splint is incorporated into a Colles' plaster (fig. 348) and is maintained for at least 4 weeks. A Sheffield splint (fig. 349) may be used as an alternative to an

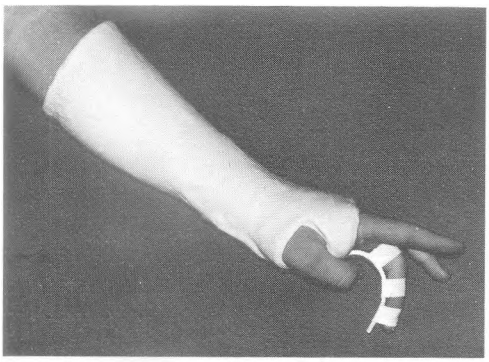


FIG. 348.—An aluminium splint for displaced phalangeal fractures.

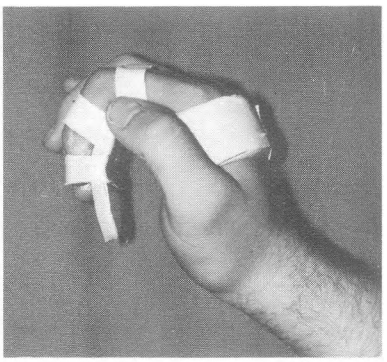


FIG. 349.—A Sheffield splint for displaced phalangeal fractures or fractures of the metacarpals needing temporary splintage to relieve pain.

aluminium splint. If the fracture is not soundly united when the splint is removed after 4 weeks, the fracture may require a further period of splintage provided by strapping the finger to its neighbour. If accurate reduction cannot be obtained by manipulation and splintage, the fracture should be reduced at operation and the reduction held by crossed Kirschner wires.

In any finger injury it is important that *only* the injured finger should be immobilised. Neighbouring fingers should be actively mobilised throughout the period of treatment.

Mallet Finger.—This injury consists of a rupture, or avulsion-fracture, of the terminal slip of the extensor tendon to the distal phalanx. If the tendon is avulsed, the bony insertion of the tendon can be seen as a small fracture fragment on the dorsal aspect of the terminal interphalangeal joint (fig. 350). The injury is caused by forced flexion of the terminal phalanx whilst the extensors of the finger are contracting. It may occur, for example, during bed-making when the hand is thrust under the mattress to tuck in the sheets: the finger tip catches in the mattress and is forcibly flexed at a time when the patient is holding the interphalangeal joints actively extended.

The patient complains of localised pain and swelling and an inability actively to extend the terminal phalanx. Passive extension is possible but when the finger is released the terminal phalanx falls back into a position of 30° of flexion under the influence of the long flexor tendons (fig. 351).

Martin Kirschner, 1879–1942. Professor of Surgery, Heidelberg Germany. Described the use of traction wires in 1909.

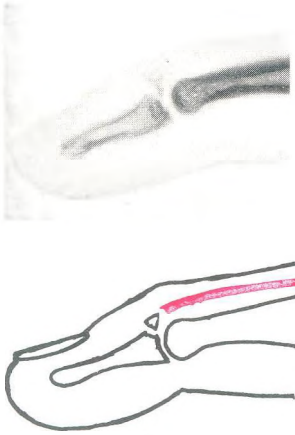


FIG. 350.—Mallet finger. Avulsion of a flake of bone to which the long extensor tendon is attached.

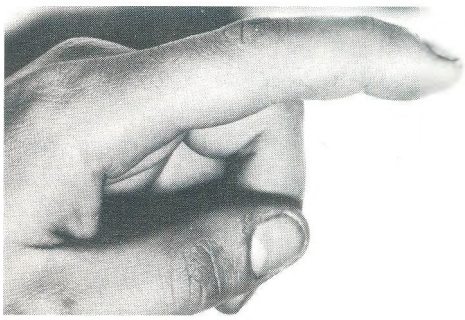


FIG. 351.—Mallet finger.

Treatment.—If a fracture fragment has been avulsed and can be seen radiologically to be rotated from its bed, the fragment should be sutured back into place. If no fragment has been avulsed, or if an avulsed fragment is not significantly displaced, the injury should be treated in a splint holding the terminal phalanx in full extension for 3 weeks (fig. 352). On this regime most tendons unite so that the patient regains active extension, but occasionally the deformity persists. The patient should be advised to accept this deformity since firstly, it often improves gradually over the year following the injury; secondly, the disability it causes is very slight, save in certain occupations such as playing the guitar; and thirdly, late treatment (by suture of the tendon) tends to stiffen the distal interphalangeal joint.

Dislocation of the Interphalangeal and Metacarpo-phalangeal Joints.—These injuries are uncommon. They can usually be reduced easily by traction on the finger followed by flexion of the joint over the fulcrum provided by the surgeon's finger on its flexor aspect. Occasionally the head of the proximal bone 'button holes' through the capsule or, in the case of the thumb, through the two slips of the flexor pollicis brevis tendon (alternatively the long flexor tendon may become interposed in the joint). Closed reduction is then impossible and the injury should be treated by operation.

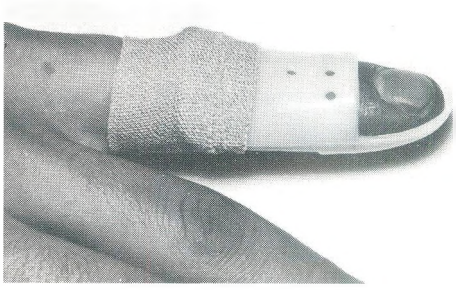


FIG. 352.—A Stack mallet finger splint.

Ruptures of the Collateral Ligaments.—Forced adduction or abduction at the interphalangeal or metacarpo-phalangeal joints may rupture the collateral ligament on the tension side of the joint. The joint momentarily subluxates but reduces spontaneously unless the ruptured collateral ligament becomes interposed between the joint surfaces.

Clinically, the joint is tender on the side of the ruptured ligament and mechanical instability can be appreciated. Instability can be confirmed by a stress X-ray. There is no abnormality in ordinary radiographs unless the bony attachment of the involved ligament has been avulsed and is radiologically visible, or the ligament is interposed in the joint space in which case persistent malalignment of the finger will be noticed.

Treatment.—The finger should be immobilised on an aluminium splint (fig. 348). If the collateral ligament is interposed in the joint surface or an avulsed bony fragment is displaced, the joint should be opened and the collateral ligament or fracture fragment sutured in its anatomical position.

Fractures of the Metacarpals.—Fractures of the shaft or neck of one or

Hugh Graham Stack, Contemporary. Orthopædic Surgeon, Harold Wood Hospital, Essex, England.

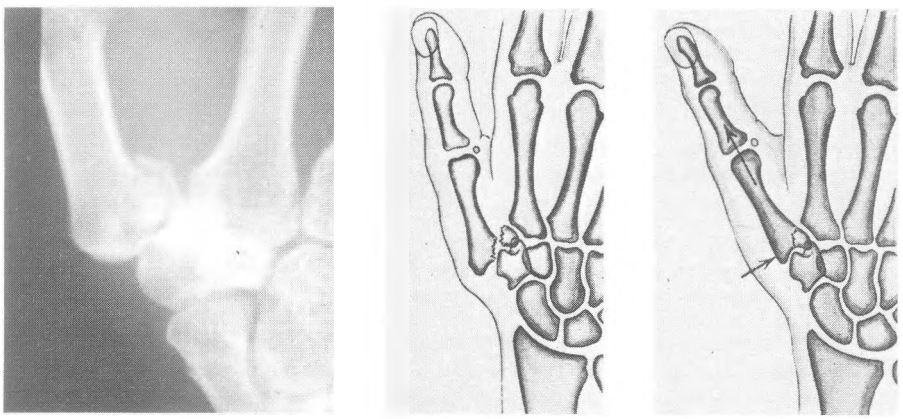
more of the medial four metacarpals are produced by the knuckles striking objects (i.e. by punching), or by blows on the dorsum of the hand. Fractures of a single shaft are splinted by the adjacent metacarpals and suffer little displacement. Fractures through the neck are usually associated with some forward angulation of the head of the bone but this deformity, even if gross, results only in recession of the knuckle and a minimal loss of extension at the metacarpo-phalangeal joint. This is of no significance if full movement at other joints and a good grip is achieved by early mobilisation. Transverse fractures of the metacarpals are sometimes slow to unite, but all other fractures unite well.

Treatment.—If pain demands temporary splintage, the hand should be rested on a Sheffield splint with the immobilised finger in a position of function, i.e. flexion not extension (fig. 349). When pain has settled, the earlier mobilisation is started the better.

It should be explained to patients with angulated fractures of the neck of the metacarpal that, if no specific treatment other than early mobilisation is undertaken, their hand will return to normal save for a ' dropped knuckle ' and perhaps a small extensor lag. If they prefer not to accept this disability, an attempt should be made to reduce the fracture by flexing the metacarpo-phalangeal joint to 90° and then pressing the proximal phalanx, and with it the distal fragment, dorsally in relation to the shaft of the metacarpal. The reduction is held by a strip of plaster wrapped round the extensor surface of the finger and incorporated proximally in a Colles' plaster (fig. 359). The plaster is allowed to set as the reduction is held by the surgeon. The disadvantages of this form of treatment are (1) that the method of holding the reduction, although the best available, is inadequate and the fracture usually slips in plaster and (2) that the finger is stiff for a longer total time than after early mobilisation.

Fractures of 3 or more metacarpal shafts require reduction and splintage since the metacarpus as a whole is unstable.

Fracture-Dislocation of the Base of the First Metacarpal (Bennett's Fracture).—This injury is caused by a blow to the point of the thumb. An oblique fracture of the base of the metacarpal occurs extending proximally to involve the articular surface. The shaft of the bone is drawn proximally by



(*a*) (*b*) (*c*)

FIG. 353.—(*a*) Bennett's fracture dislocation, (*b*) the nature of the initial displacement and (*c*) the principles of reduction.

Edward Hallaran Bennett, 1837–1907. Professor of Surgery, Trinity College, Dublin. Described this fracture in 1882.

the pull of the long muscles of the thumb, so that it subluxates from the carpo-metacarpal joint (fig. 353).

Treatment.—The fracture-dislocation is easily reduced by traction, extension of the thumb, and pressure over the dorsal aspect of the base of the first metacarpal (fig. 353). The difficulty is to maintain reduction. This can be done if the 1st metacarpal is immobilised in plaster in almost full extension at the carpo-metacarpal joint whilst direct pressure is applied over the dorsum of the base of the bone as the plaster sets, so as to press the shaft medially against the palmar fragment. Unfortunately a reduction held in this way often slips. Therefore some surgeons apply continuous traction to the thumb by means of a plaster cast applied to the forearm, in which is incorporated a loop of thick wire to take adhesive traction applied to the end of the thumb. Alternatively the fracture may be fixed internally with a screw.

Lacerations of the Hand. (These are discussed on p. 209.)

INJURIES TO THE CARPUS

Any of the bones of the carpus may be fractured but by far the commonest injury is fracture of the scaphoid.

Fractures of the Waist of the Scaphoid

The injury occurs following a fall on to the outstretched hand, typically in young adults. It is uncertain precisely how this causes fracture of the scaphoid, but it seems possible that the bone is 'bent' at its waist over the radial styloid.

The patient presents with a complaint of pain in the wrist, but function of the wrist may not be grossly impaired. On examination there is tenderness over the scaphoid in the anatomical snuff-box, a little swelling and no bruising. These physical signs often appear trivial and suggest a 'sprained wrist' rather than a fracture. Radiological examination (which should include not only an antero-posterior and lateral view of the wrist, but also two oblique views) may not display the fracture immediately after injury since no displacement occurs at the fracture site.

Treatment.—Because this fracture does not produce substantial clinical and radiological signs it is often missed and wrongly diagnosed as a 'sprained wrist'. The wrist is then treated with strapping and as a consequence the fracture may not unite. Therefore any 'sprained wrist' in which a fracture of the waist of the scaphoid cannot be excluded should be treated for 2 weeks in a scaphoid plaster (fig. 354). Two weeks after the injury, the plaster should be removed and the wrist re-X-rayed. By this time bone resorption will have occurred at the site of a scaphoid fracture so that the injury will be radiologically obvious (fig. 355). If the fracture is diagnosed at the time of the injury or at re-X-ray 2 weeks later, a scaphoid plaster

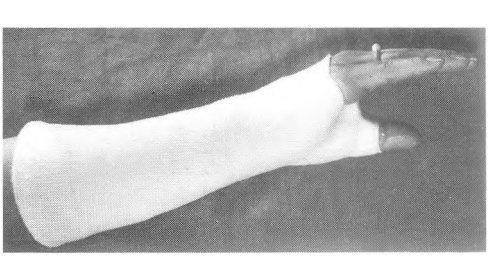


FIG. 354.— A scaphoid plaster.

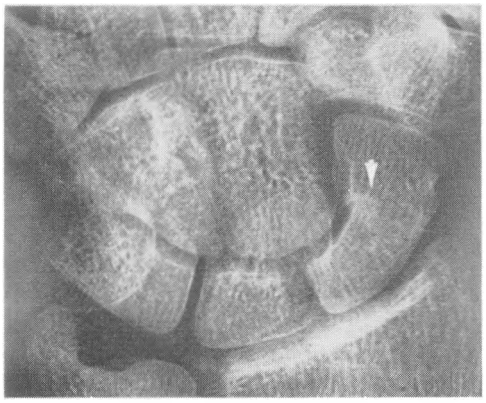


FIG. 355.—Fracture through the waist of the scaphoid.

should be applied for 8 weeks.

Non-union is not infrequent even after immobilisation for 8 weeks, because the blood supply to the proximal part of the bone often enters entirely through the distal pole. Thus when the waist of the scaphoid is fractured the proximal pole is infarcted. The fracture then fails to unite and avascular changes, similar to those seen in the femoral head (p. 404), may develop in the proximal half of the bone. These changes may lead to osteoarthrosis of the wrist (fig. 356). Non-union used to be treated by further immobilisation for periods of as long as a year but although immobilisation may eventually result in union of the fracture, it may also result in a stiff wrist. It is therefore preferable to offer the patient with an un-united fracture at 8 weeks two alternatives: to accept the un-united fracture (which will not always give rise to significant disability) or to treat the non-union by a bone graft and internal fixation with a small screw followed by a further 8 weeks immobilisation in plaster.

Fracture of the tubercle of the scaphoid occurs occasionally and is an insignificant injury. It is functionally immaterial whether the fracture unites or not, and the wrist need only be treated by such immobilisation as is necessary to relieve pain. The essential difference between this injury and fractures of the waist of the scaphoid is that in this injury no articular surface is involved, whereas after fractures through the waist of the scaphoid the mechanism of the wrist may be prejudiced by an unsound fibrous non-union.

FIG. 356.—Non-union of a fracture of the waist of the scaphoid with secondary osteoarthrosis of the wrist.

Lunate and Perilunate Dislocation of the Wrist

A fall on the palm of the outstretched, dorsiflexed hand may dislocate the whole of the carpus backwards in relation to the radius and lunate. This is a perilunate dislocation of the wrist. The dislocation may persist, spontaneously reduce, or reduce but in so doing displace the lunate forwards. This third possibility produces a dislocation of the lunate. These dislocations may be complicated by a fracture of the waist

of the scaphoid. If they are, the proximal pole of the scaphoid remains with the lunate, and the distal pole with the rest of the carpus.

Surprisingly, it is not uncommon for these injuries to be missed clinically and radiologically. Clinically the wrist and fingers are stiff and the wrist swollen and tender. If the lunate has dislocated, it may compress the median nerve to cause median nerve signs. A well-centred lateral X-ray clearly reveals the dislocated lunate in front of the carpus or the posterior displacement of the carpus, but a poor, slightly rotated lateral view may conceal the dislocation. The antero-posterior view at first sight may be passed as normal, but close inspection will show that the bones of the proximal row of the carpus (scaphoid, lunate and triquetral) are not disposed round the head of the capitate with the intervention of a regular joint space as in the normal wrist, and, if the lunate has dislocated, the lunate appears tri-angular rather than quadrangular in outline because it is rotated as it is dislocated forwards.

FRACTURES OF THE DISTAL RADIUS AND ULNA

Colles' Fracture

This is a fracture of the distal end of the radius produced by a fall on to the palm of the outstretched hand. The injury is uncommon below the age of 50. After this age, it becomes increasingly common so that overall it is one of the commonest fractures. It is particularly common in women, as are fractures of the proximal femur, and, like fractures of the proximal femur, its increasing frequency with age has been attributed to the development of osteoporosis in post-menopausal women. The association of the fracture with increasing age is probably also due to the fact that the elderly are more liable to fall than are the young.

Morbid Anatomy.—The fracture line lies approximately 2 cm. proximal to the distal articular surface of the radius. The fracture is often comminuted, especially on the dorsal surface. The distal fragment is: (1) displaced dorsally, (2) angulated dorsally, (3) driven proximally into the shaft, (4) angulated laterally and (5) supinated. There is always an associated injury to the inferior radio-ulnar joint: the articulating surfaces of these bones are displaced relative to each other and their connecting ligaments (including the triangular cartilage) are torn or the ulnar styloid is avulsed. It is uncertain whether the latter injury is due to avulsion of the ulnar styloid by the triangular cartilage or by the ulnar-collateral ligament of the wrist.

Clinical Features.—The appearance of the wrist is typical and is described as a 'dinner-fork' deformity (fig. 357). Viewed from the side the dorsal aspect of the wrist is unduly prominent; viewed from the dorsum the lateral aspect of the wrist is slightly prominent and the hand is radially deviated. The fracture site is tender. The radial styloid is no longer distal to the ulnar styloid; instead the two styloid processes are approximately at the same level, a reflection of shortening in the radius (fig. 357). Radiologically the fracture, the extent of the displacement, and the degree of comminution will be obvious (fig. 358).

Treatment.—The fracture is reduced by manipulation and immobilised

Abraham Colles, 1773–1843. Professor of Surgery, Royal College of Surgeons in Ireland (1804–1836) and Surgeon Dr. Steven's Hospital, Dublin. Described this fracture in 1814.

with a plaster back-slab which is completed to form a Colles' plaster (fig. 359) 24 hours after the injury.

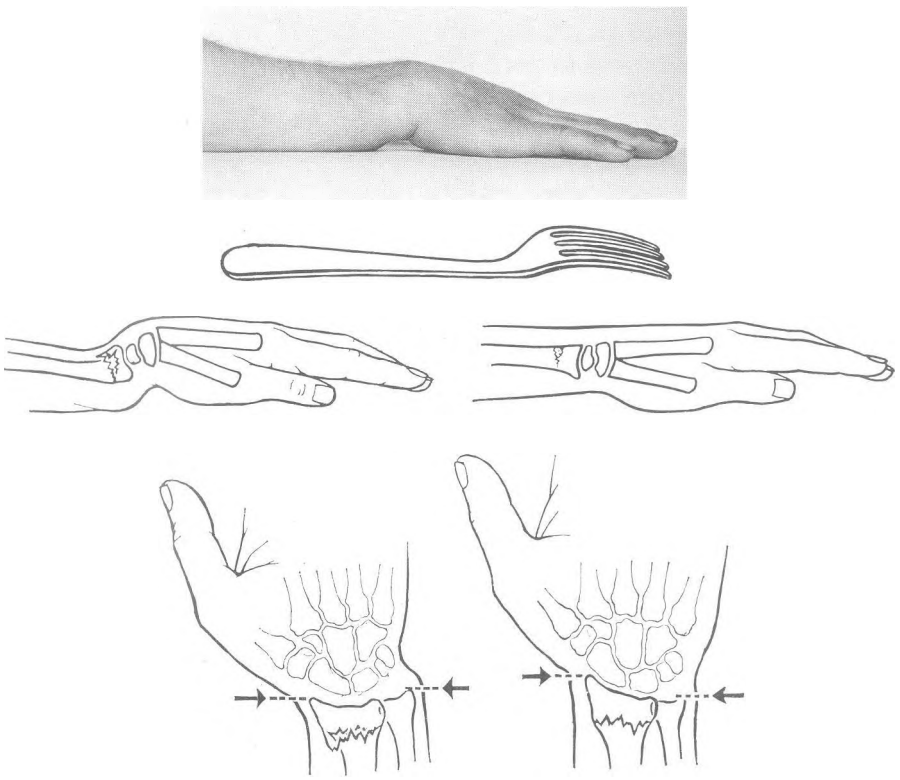


FIG. 357.—Dinner-fork deformity of Colles' fracture. Note the levels of the radial and ulnar styloid processes before and after reduction.

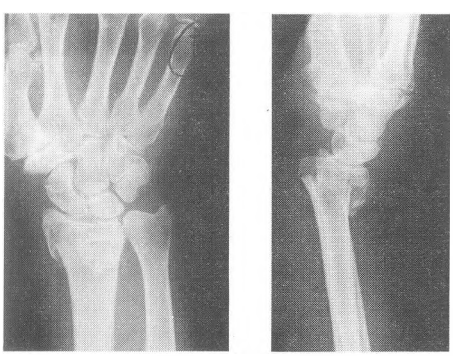
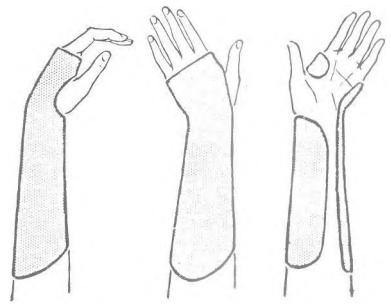


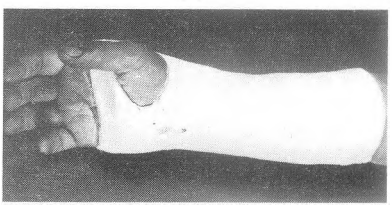
FIG. 358.—Colles' fracture.

Manipulation of a Colles' Fracture.—The patient is given a general anæsthetic or a regional block using either intravenous anæsthesia distal to a tourniquet or a brachial plexus block. An assistant grasps the upper arm whilst the surgeon grasps the injured hand as if he were ' shaking hands '. He then exerts firm traction in the long axis of the limb in order to disimpact the fragments of the radius. When this has been accomplished, he presses the distal fragment into palmar-flexion and ulnar-deviation using the thumb of his other hand. At the same time, he draws the patient's hand into pronation, ulnar-deviation and palmar-flexion. A plaster slab is then applied to the lateral, dorsal and medial aspects of the forearm, wrist and hand from the elbow to the metacarpal heads holding the wrist in palmar-flexion and ulnar-deviation (fig. 359(*a*)). Satisfactory reduction is confirmed by X-ray. The slab is converted to an encircling Colles' plaster if the circulation to the fingers is satisfactory 24 hours later.

The plaster is maintained for 6 weeks. If extreme palmar-flexion and ulnar-deviation is required to maintain the reduction, this position should be maintained only for 3 weeks, at the end of which time the plaster is removed and re-applied with the wrist more nearly neutral. It should be noted that although pronation is required to correct the supination element of the deformity, a plaster is applied which does not control the movements of pronation and supination. In this sense a Colles' plaster is inadequate for this injury: it is used only because, in the age group involved (the elderly), the injury to the inferior radio-ulnar joint results in a greater limitation of pronation and supination if these movements are restricted than if they are permitted. Loss of forearm rotation is a more serious disability than is that attributable to malunion. The fingers should be actively mobilised immediately after the injury since otherwise they may become stiff.



(*a*)



(*b*)

FIG. 359.—Colles' plaster. (*a*) Plaster slab in ulnar deviation and slight palmar-flexion applied immediately after the reduction of a Colles' fracture. (*b*) The completed plaster.

Union is almost invariable, but there is often some residual deformity because (1) even if the initial reduction is perfect, recurrent backward angulation of the distal fragment is common if the dorsal cortex of the radius is comminuted and, (2) a Colles' plaster does not control the supination element of the deformity. When the plaster is removed, the wrist is often stiff and may require remobilisation in a physiotherapy department. The inferior radio-ulnar joint is also commonly stiff and may be very difficult to remobilise. Occasionally persistent limitation of pronation and supination combined with significant displacement of the distal radius relative to the distal ulna may, in younger patients, require excision of the distal ulna (a procedure which can be regarded as an excision arthroplasty of the inferior radio-ulnar joint).

Fracture of the Radial Styloid

This injury occurs following a direct blow on the wrist (the injury used to be called 'chauffeur's fracture', when cars were started with a starting handle, since if this kicked back it often struck the wrist on its lateral aspect) or occasionally following a fall on the wrist. The fracture line runs proximally and laterally through the radial styloid roughly from a point opposite the articulation between the scaphoid and the lunate. Displacement is uncommon and the only treatment required is the application of a Colles' plaster for 6 weeks.

Displacement of the Distal Radial Epiphysis

This is an injury confined to children. The displacement is similar to the Colles' displacement. The fracture line runs partly through the epiphyseal plate itself and

partly through the metaphysis. The injury may be surprisingly difficult to reduce. Reduction should be carried out under general anæsthesia and a Colles' or above-elbow plaster applied for 6 weeks. Fortunately, a disturbance of growth is rare.

Fracture through the Distal End of the Radius in Children

This may occur at any level proximal to the epiphyseal plate. The fracture may be of the greenstick variety or may be complete. The former is often almost undisplaced and only requires support in a Colles' plaster for 3 to 4 weeks. The fracture may however be complete, with marked angulation (concave dorsally) at the fracture site. This is a deceptive injury for it is often regarded by the inexperienced as the equivalent of a Colles' fracture and treated in the same way. If it is, so that pronation and supination are not controlled by the plaster which is applied, significant re-angulation commonly occurs at the fracture site leading to mal-union and marked limitation of pronation and supination. Following reduction, the fracture should be held by a well-moulded plaster which extends above the elbow with the hand palmar-flexed, ulnar-deviated and fully supinated, the elbow itself being at right angles so that pronation and supination are controlled. Both undisplaced and displaced fractures should be X-rayed one week after reduction to ensure that angulation of the fracture has not occurred in plaster. If it has, re-manipulation is required.

Smith's Fracture

This is the reverse of Colles' fracture, the distal fragment being palmar-flexed rather than dorsiflexed. It is uncommon. The injury is conventionally attributed to a backward fall on the outstretched hand but it seems more likely that it is in fact due to a fall on the dorsum of the palmar-flexed wrist, not to a fall on the palm of the outstretched hand at all. Reduction is by reversal of the original mechanism and immobilisation in an above-elbow plaster for 6 weeks or, better, by internal fixation using an Ellis plate.

FRACTURES OF THE SHAFT OF THE RADIUS AND ULNA

Fractures of One Forearm Bone without Angulation.—A fracture of the radius or ulna, without angulation, may occur as a consequence of a blow applied directly to the forearm. The fracture requires immobilisation in an above-elbow plaster for 6 weeks in an adult, 3 weeks in a child. (See also: Fracture-Dislocation of the Forearm, p. 363).

Fractures of Both Forearm Bones

These fractures usually occur at approximately the same level and are caused by a direct blow, which in effect bends the bones at the point where the blow is received. Occasionally the bones may be fractured by torsion of the forearm: in this case the fractures are oblique and are disposed at different levels in the two bones.

Treatment.—In the adult it is imperative that an absolutely perfect reduction be achieved since otherwise a loss, often substantial, of pronation and supination will occur. In the child residual angulation of about 5° can be accepted as it will be corrected by remodelling. Residual rotation (pronation or supination) at the fracture site cannot be corrected by remodelling.

Perfect reduction is usually impossible by closed manipulation. Therefore in the adult only one attempt should be made to reduce the fractures conservatively. If this fails, the injury is best treated by operative reduction of both fractures followed by internal fixation using either intramedullary Rush nails or, better, strong plates applied with the fracture sites in com-

Robert William Smith, 1807–1873. Professor of Surgery, Trinity College, Dublin. Described the reverse Colles' fracture of the lower ends of the radius in 1847.
James Stokes Ellis, Contemporary. Professor of Orthopædic Surgery, University of Southampton, England.
Leslie V. Rush, Contemporary. Surgeon, Rush Memorial Hospital, Meridian, Mississippi, U.S.A.

pression. Following mechanically adequate internal fixation no plaster is required provided the patient undertakes only gentle activities with his arm. If plates have been applied which maintain reduction but which are not mechanically sound, they will need the support of an above-elbow plaster for at least 6 weeks. In a child, an acceptable reduction can usually be obtained conservatively but if it cannot the fractures should be treated by internal fixation using onlay plates, never Rush nails.

The Technique of Closed Manipulation.—The elbow is flexed to 90° and the surgeon applies traction to the hand against counter-traction applied by an assistant grasping the upper arm. Traction corrects angulation and disimpacts the fractures so that the bones in the distal part of the forearm can be pronated or supinated to align them with those in the proximal part. If the radial fracture is distal to the insertion of pronator teres, the proximal radius will lie in mid pronation, but if the fracture is proximal to the insertion of pronator teres, the proximal radius lies in full supination and this is therefore the position in which the surgeon places the hand. When the fracture has been reduced, an assistant applies an above-elbow plaster whilst the surgeon maintains traction. The fracture is X-rayed immediately the plaster has been applied, at 48 hours and then weekly for 3 weeks. The plaster is maintained for 6 weeks. If at any time the position of the bones is seen to be unsatisfactory, the fracture is treated by open reduction.

Fracture-dislocation of the Forearm

Monteggia fracture-dislocation consists of a fracture of the ulna and dislocation of the superior radio-ulnar joint.

The Galeazzi fracture-dislocation consists of a fracture of the radius and dislocation of the inferior radio-ulnar joint.

The presence of a fracture of one forearm bone should always raise the suspicion that the inferior or superior radio-ulnar joint has been dislocated as well. This suspicion is confirmed if angulation occurs at the fracture site, for this is only possible in a fracture of a single forearm bone if the ligaments connecting the angulated bone to its opposite number have been torn. Thus the general rule that a fracture somewhere in the shaft of a long bone demands an X-ray, not only of the bone itself but of the proximal and distal joint, is particularly well exemplified by this fracture.

Treatment is by open reduction of the fracture and if necessary of the dislocation, followed by internal fixation of the fracture and immobilisation of the arm in an above-elbow plaster.

INJURIES TO THE ELBOW

The elbow is very liable to become stiff following an injury, regardless of its exact nature. It is uncertain why this is so: commonly no specific explanation for the stiffness can be found and it is presumed that adhesions have formed within and around the capsule of the joint. It seems likely that too vigorous an attempt to move the joint in the first few weeks after an injury may promote stiffness of this kind and therefore in general it is wise to rest the elbow for a short period following injury (immobilisation may, of course, be inevitable as a consequence of the nature of the injury). Movement is then gently encouraged under close supervision in a physiotherapy

Giovanni Battista Monteggia, 1762–1815. Professor of Surgery, Milan. Described this fracture in 1814.
Riccardo Galeazzi, 1866–1952. Director of the Orthopædic Clinic, University of Milan. Described this fracture in 1934.

department, making repeated measurements of the precise range of flexion and extension that has been obtained.

Myositis Ossificans.—This is a condition, misleadingly named, which may occur following musculo-skeletal injury anywhere in the body but which is particularly common following injuries at the elbow. Why it should be common at the elbow is unknown.

Pathologically, the condition consists of ossification in a hæmatoma. This may occur following a fracture, or may follow a purely soft tissue injury in which periosteum has been avulsed. It is believed that the periosteal cells proliferate within the hæmatoma and ossify it. Since the new bone replaces the hæmatoma, the bone tends to lie between the muscles rather than to involve the muscles themselves. Thus myositis ossificans is not, as its name implies, an ossifying inflammatory disease of muscle.

FIG. 360.—Myositis ossificans anterior to the elbow: mature bone.

Clinically, at the elbow the development of the condition may first be suggested by a failure of the range of movement gradually to increase following gentle mobilisation. If exercises are then pressed strenuously, the range of movement actually decreases rather than increases and pain in the elbow becomes worse. At this stage (about 3 or 4 weeks after injury) there are no abnormal radiological signs. By 4 to 6 weeks after injury a faint cloud of new bone formation with indistinct edges can be seen in the area destined to become ossified. The new bone gradually becomes more dense and more organised until finally it takes on the radiological appearance of mature bone (fig. 365).

Treatment is difficult. When the condition is first suspected the elbow should be rested absolutely in a plaster cylinder until pain and tenderness have settled. Remobilisation should then be started extremely gently taking care to measure the range of movement at least twice a week in order to detect the earliest sign of any reduced mobility since this implies that the attempt to mobilize the joint is increasing the amount of new bone formation and thus making the condition worse, not better. Attempts have been made to remove the new bone surgically. This should never be attempted until the bone has a radiologically mature appearance, since if it is, the hæmatoma produced by the operation is liable to ossify in its turn. The removal of a block of mature new bone is often surgically difficult and may give a disappointing increase in the range of movement since by the time that the operation is possible, fibrous adhesions have formed between all the soft

tissues around the elbow. However the condition is treated, some permanent limitation of movement is the usual outcome.

Dislocation of the Elbow

This usually occurs in a posterior direction, following a fall on the outstretched hand with the elbow slightly flexed. The coronoid process of the ulna passes posteriorly below the distal end of the humerus. Spasm of the triceps muscle then locks the elbow in a position of posterior dislocation (fig. 361).

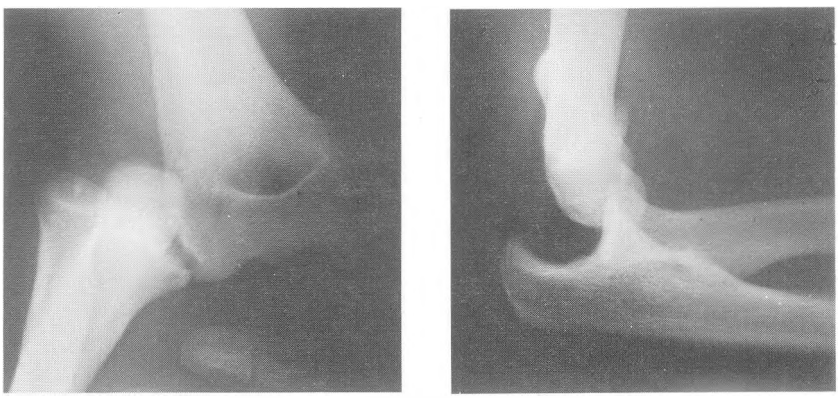


FIG. 361.—Posterior dislocation of the elbow.

Clinical Features.—The patient is unable to move the elbow from a position of slight flexion. The point of the olecranon can be felt to be abnormally posterior to the humeral epicondyles: a clinical feature which distinguishes this injury from supracondylar fracture of the humerus (fig. 362). Radiological examination reveals the dislocation and may show associated fractures either of the coronoid process, of the head of the radius, or of the distal articular surface of the humerus. The fractures are often best, or only, seen in a post-reduction radiograph, an investigation which should never be omitted.

Treatment consists of giving the patient a general anæsthetic to produce muscle relaxation. It is then easy to reduce the dislocation by applying

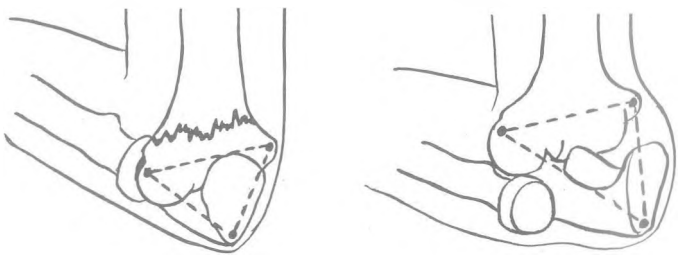


FIG. 362.—Bony points in the elbow. 1. In the normal elbow and in the elbow after a supracondylar fracture, the triangle formed by the tip of the olecranon and the two epicondyles is roughly equilateral. 2. When the elbow is dislocated the tip of the olecranon is displaced and the triangle is no longer equilateral.

traction in the long axis of the (slightly flexed) ulna. The sensation of reduction is usually obvious and once achieved the elbow can be put through a full range of movement. The reduction is stable because of the bony configuration of the elbow, but it must be remembered that this injury can only occur if the capsule and ligaments of the elbow have been ruptured. The elbow should therefore be immobilised in a plaster back slab and a sling for 3 weeks and thereafter gently mobilised.

Rupture of the Collateral Ligaments of the Elbow

Forced abduction of the forearm bones on the humerus may rupture the medial collateral ligament or avulse its bony attachment to the medial epicondyle. Adduction may produce a similar injury to the lateral collateral ligament.

Clinical Signs.—There is marked tenderness, bruising, and swelling in the region of the injured ligament. Mechanical instability of the joint may be suspected clinically, and can be proven by examining the elbow under an anæsthetic, if necessary taking a stress radiograph.

In the absence of a fracture, the arm is treated in the same way as following the reduction of a dislocation of the elbow. The treatment of an associated fracture of the medial epicondyle (an injury equivalent to a rupture of the medial collateral ligament) is discussed on p. 371.

Fractures of the Proximal Ulna

Two fractures of the proximal ulna may involve the elbow: fracture of the olecranon and fracture of the coronoid process.

Fracture of the olecranon is usually caused by a fall on to the point of the elbow, the olecranon being broken by the distal end of the humerus. The fracture line is transverse and runs through the narrowest point of the olecranon. The pathological and clinical features of the injury and its treatment are closely paralleled by transverse fractures of the patella.

Clinical Features.—It is essential to decide clinically whether or not the triceps expansion is intact over the fracture site. If it is, there will be local bruising, swelling and tenderness, but active extension of the elbow against gravity will be possible, there will be no clinically detectable gap at the fracture site, and radiologically there will be no separation of the fracture fragments. If the triceps expansion is ruptured, active extension against gravity is impossible and the proximal fragment is drawn up into the arm by the triceps muscle creating a palpable gap at the fracture site and an obvious separation of the fragments radiologically (fig. 363).

FIG. 363.—Fracture of the olecranon with separation.

Treatment.—If there is no significant separation of the fracture fragments and active extension is possible, the elbow may be treated by 6 weeks' immobilisation in a padded plaster cylinder followed by gentle

mobilisation or by internal fixation. If the fracture fragments are separated, the fracture must be reduced and immobilised by internal fixation since the fracture cannot be completely reduced even by full extension of the elbow, and immobilisation of the fully extended elbow may result in disastrous stiffness of the joint. The fracture should therefore be fixed internally by, for example, a screw placed through the tip of the olecranon (fig. 364). Following operation, the elbow should be immobilised for 3 weeks in a plaster back slab. Thereafter screw fixation is usually sufficiently secure to allow progressive mobilisation of the elbow.

Fracture of the coronoid process is uncommon and usually accompanies dislocation of the elbow. The coronoid process is usually approximated to the ulna by reducing the dislocation. If it is not, open reduction is required.

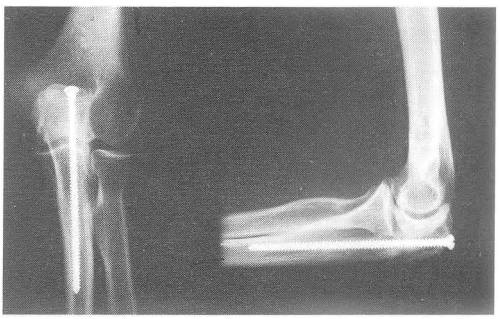


FIG. 364.—Internal fixation of a fracture of the olecranon with a screw.

Fractures of the Proximal Radius

These fractures occur as a consequence of a fall on the outstretched hand. If the thrust goes directly up the long axis of the radius, the neck of the bone may be fractured and is often impacted: a mechanism similar to that which produces fractures of the neck of the humerus. More commonly the fracture line runs vertically to split off a fragment of the head (fig. 365). The two fractures may be combined to produce a comminuted fracture of the head of the radius.

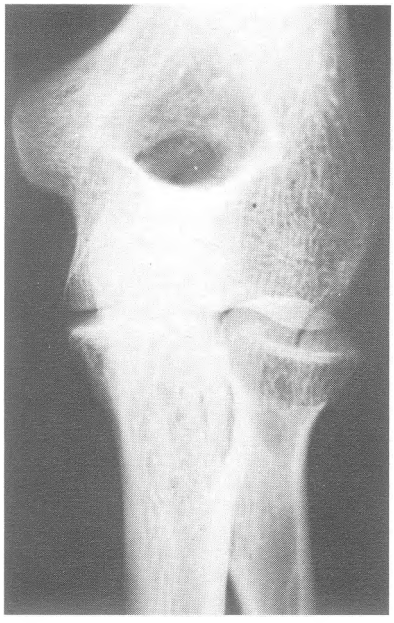


FIG. 365.—Vertical fracture of the head of the radius.

The clinical signs are often trivial and do not suggest that a fracture has occurred. There is commonly very little bruising and minimal swelling. The only significant physical signs are painful limitation of movement combined with tenderness over the head of the radius. The injury is revealed radiologically.

Treatment.—If the fracture involves the articular surface and significant displacement has occurred, the head of the radius must be excised. If it is not, the resulting deformation of the head of the radius limits movement, the fracture fragment may not unite so that it forms a loose body, and late osteoarthrosis is almost inevitable. If the

fracture line runs through the neck of the radius or runs through the head but with very little displacement, the elbow should be immobilised for 3 weeks in a plaster back slab and then gently mobilised in a physiotherapy department. If the final outcome is significant limitation of pronation and supination combined with deformation of the head of the radius, the head of the radius should be removed some months after injury.

Under no circumstances should the head of the radius be removed in a child since this results in loss of growth at the proximal end of the radius and hence in progressive derangement of the elbow and radio-ulnar joints.

Supracondylar Fracture of the Humerus in Children

This injury is one of the most serious fractures in childhood and poses considerable therapeutic problems.

Mechanism and Morbid Anatomy.—The injury is caused by a fall on to the outstretched hand with the elbow slightly flexed. The fracture line runs transversely through the epiphysis and distal metaphysis of the humerus (fig. 366).

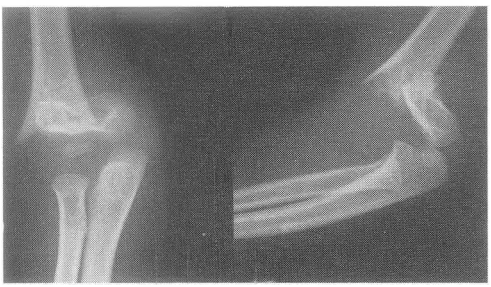


FIG. 366.—Supracondylar fracture of the humerus; there is danger to the neuro-vascular structures between the fragments in this very displaced fracture. (See fig. 343.)

This displacement of the distal fragment may be minimal, but more commonly there is substantial, complex displacement. This consists of the following elements: 1. backward displacement of the distal fragment, 2. backward angulation of the distal fragment, and 3. pronation of the distal fragment (because the hand is usually pronated at the time of the injury). Pronation produces internal rotation of the distal fragment (the two movements are really the same). As a consequence, the medial cortex of the distal fragment moves posteriorly relative to the medial cortex of the shaft of the humerus, whilst on the lateral side of the fracture the fragments often remain 'hitched' to each other. The pull of the arm muscles then draws the medial side of the distal fragment proximally, posterior to the shaft of the humerus, whilst leaving the lateral side roughly in its original anatomical position. The effect of this displacement is to adduct the distal fragment relative to the shaft of the humerus. Thus pronation results in internal rotation and adduction of the distal fragment.

Clinical Features.—The elbow swells rapidly following this injury so that gross swelling may obscure the other physical signs within 3 or 4 hours. During this time bruising is not obvious. The child is unable to move the elbow. If significant swelling has not occurred, the posterior prominence of the point of the elbow is obvious and it is possible to palpate the medial and lateral epicondyles and the point of the olecranon so as to establish that they

are normally related to each other (fig. 362). This finding distinguishes the injury clinically from a posterior dislocation of the elbow.

The possibility of an interruption of the blood supply to the forearm following this injury must be paramount in the mind of the surgeon from the moment that he first sees the child. This complication may occur at the time of the injury if the brachial artery is contused or lacerated by the anterior aspect of the proximal fragment of the humerus. In this event the radial pulse will not be palpable. More commonly the radial pulse is present at the time of the injury but is obliterated by swelling and flexion of the elbow following reduction. It is therefore imperative to examine carefully the blood supply to the hand on the injured side at repeated intervals over the first 2 or 3 days following injury.

The details of the injury may be difficult to make out radiologically, since ossification of the distal end of the humerus is complex and presents a confusing radiological picture. Further difficulty is created by the fact that the elbow cannot be extended to take an X-ray. These difficulties can to some extent be overcome by X-raying the other elbow in the same position of flexion for comparison.

Treatment.—Reduction of this fracture is difficult and if the surgeon has not already had the opportunity of reducing the fracture himself he should if possible consult an experienced surgeon urgently so that the latter has the opportunity of assisting the manipulation before gross swelling has made palpation of the normal anatomy impossible.

Technique of Manipulation of a Supracondylar Fracture

The assistant grasps the upper arm whilst the surgeon holds the injured hand as if he were 'shaking hands'. The surgeon then exerts firm steady traction for a period of 2 minutes or more in the *long axis of the forearm*: that is to say he does not attempt to extend the elbow until traction has drawn the distal fragment beyond the proximal fragment. This reduces the chance of damaging the brachial artery with the distal end of the proximal fragment when the elbow is extended in the next stage of the manipulation, or of entrapping it in the fracture site when the elbow is subsequently flexed. Following traction in the flexed position, the surgeon extends the elbow, feeling the radial pulse as he does so. When full extension has been obtained, the forearm is fully supinated to correct the pronation described above. The fact that the hand and distal fragment are fully supinated is confirmed by fully supinating the uninjured hand and externally rotating the shoulder: the two hands should now have assumed the same attitude. Next, the carrying angle at the elbow is corrected by eye. The surgeon then grasps the upper arm with his second hand placing his fingers over the biceps muscle so that his thumb rests on the olecranon. He then changes the position of the hand exerting traction, placing it so that he grasps the distal forearm with his index on the radial pulse. He then slowly flexes the elbow using the hand with which he is exerting traction to produce a combination of flexion and continuous traction in the long axis of the forearm. The thumb over the olecranon presses the olecranon (and with it the distal fragment) forwards into flexion; the fingers of this hand exert counter-traction against the hand pulling in the long axis of the forearm. Flexion is continued until a point just beyond 90° is reached. Throughout this manœuvre the radial pulse is felt and if it is obliterated by flexion (which may be due either to impingement of the brachial artery on the distal end of the proximal fragment of the humerus or to compression of the brachial artery as the swollen forearm 'folds' into it) the elbow is extended until the pulse returns. When the maximum degree of flexion has been obtained compatible with the presence of a radial pulse, a light back slab is applied over padding to hold this position and the reduction is checked radiologically.

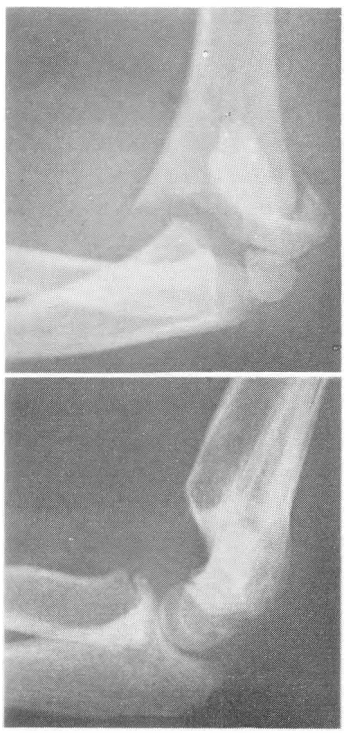


FIG. 367.—The upper radiograph shows an incompletely reduced supracondylar fracture which remodelled to the appearance seen in the lower illustration over the course of two years with full return of function.

Following reduction of the fracture, *the child is admitted to hospital* and the arm is elevated. Throughout the following 48 hours the state of the circulation in the hand is examined regularly and any suggestion that the circulation is impaired is treated instantly by removal of the plaster and increased extension of the elbow. If extension of the elbow does not return the circulation to normal, the brachial artery must be explored to ensure its patency.

The triceps muscle wrapped around the back of the fracture holds the fragments in a position of reduction provided adequate flexion has been obtained. X-rays are taken at 48 hours and again 1 week after injury to check that reduction has been maintained, but it is difficult to obtain a view in which the fracture fragment can be adequately displayed. The best view is one taken 'shooting through the elbow' at right angles to the distal end of the humerus.

The fracture unites in 3 or 4 weeks. The sling is then removed and the child's arm gently mobilised. Some loss of movement and mal-union, resulting especially in a loss of the carrying angle, are not infrequent sequels to this injury. Deformities due to incomplete reduction (other than loss of the carrying angle) are gradually reduced, but not necessarily eliminated, by bone remodelling over the years following injury (fig. 367).

Supracondylar Fracture of the Humerus in Adults

This injury, like a fracture of the olecranon, is caused by a fall on to the point of the elbow, but in this case the olecranon breaks the humerus rather than the humerus breaking the olecranon. Which of these two fractures occurs appears to depend upon the degree of flexion at the elbow at the moment of impact. The fracture line runs vertically upwards from the centre of the distal articular surface of the humerus and then diverges to reach the medial and lateral cortices, forming a 'Y' shaped fracture. There may be further comminution of the fragments.

The diagnosis is made upon the clinical evidence of injury to the elbow combined with the obvious X-ray appearances.

Treatment.—In young adults attempts should be made to reduce this fracture accurately but this is difficult. The best method of achieving accurate reduction is by open operation and internal fixation. Unfortunately, elbow stiffness is common following this procedure even if reduction has been accurately achieved. In the elderly the fracture should be ignored, the elbow rested in a plaster cylinder for 2 weeks, and then gradually mobilised regardless of displacement at the fracture site. A surprisingly good range of movement is often obtained in this way.

Fracture of the Medial Epicondyle

This is an avulsion injury of either the humeral attachment of the wrist and finger flexors, or of the medial collateral ligament of the elbow. If the fragment is only slightly displaced, the elbow should be rested for 3 weeks and then gently mobilised. Even if fibrous union occurs it is of no consequence. If the fragment is displaced and rotated, closed reduction is impossible. It is imperative to replace the fragment accurately, especially if it is displaced into the elbow joint, since, if this is not done, permanent instability of the joint may result. The fracture site should be exposed surgically and the fragment re-attached either by periosteal sutures or by a small screw.

Fracture of the Shaft of the Humerus

Mechanism.—These injuries are due either to a twisting injury to the arm or to a direct blow over the shaft of the humerus. The former injury produces a spiral fracture which may include a ' butterfly ' fragment, the latter produces a transverse fracture which may also have a ' butterfly ' fragment, but usually a smaller one.

Clinical Features.—The physical signs of tenderness, swelling, deformity and abnormal movement usually make the presence of a fracture obvious and this is confirmed radiologically.

Treatment.—Reduction of these fractures is not necessary since gravity acting on the arm exerts steady traction which tends to correct any angulation that may occur. Because the shoulder is a multi-axial joint, any angulation which may persist at the fracture site does not interfere materially with function and, surprisingly, up to 20° of angulation at the united fracture produces no clinically obvious deformity (Klenerman). It is necessary only to rest the arm in a collar-and-cuff (not a sling since a sling supports the weight of the arm and hence removes the traction force on the fracture site) and, for pain relief, to bandage the arm to a slab of plaster running from the top of the acromion, down the lateral side of the arm, under the elbow and up the medial side of the arm for 5 cm. For further security the arm, splinted by the U-slab, may be bandaged to the chest wall. Immobilisation should be maintained for 3 weeks during which the hand and, as far as possible, the elbow are kept mobile. From 3 to 6 weeks the arm is removed daily from the collar-and-cuff to mobilise the shoulder. Non-union is rare, but does occur occasionally. If it does, it should be treated by a bone graft and internal fixation, using an onlay plate.

An occasional complication of fractures of the humerus at the level of the spiral groove is damage to the radial nerve. The nerve is rarely transected, so that if there is a radial nerve palsy immediately after injury, fixed flexion of the wrist and fingers should be prevented by a combination of splintage and passive mobilisation whilst recovery is awaited. Only if there is no recovery after 2 months should the fracture site be explored. The radial nerve is dissected free and (if necessary) sutured.

INJURIES TO THE SHOULDER

Fractures of the Proximal Humerus

The humerus may fracture through the surgical neck following a fall on to

Leslie Klenerman, Contemporary. Orthopædic Surgeon, Northwick Park Hospital, Harrow, Middlesex, England.

the outstretched hand which drives the humerus upwards against the acromion and glenoid. The anatomical neck may fracture following a fall on to the point of the shoulder which drives the humerus directly against the glenoid.

Fractures through the surgical neck are common in the elderly. The fracture is often comminuted: the main fracture line runs through the surgical neck, but in addition one or both of the tuberosities may be separated to give a total of 4 fragments (the head, the 2 tuberosities, and the shaft) (fig. 368). This fracture has been divided on radiological grounds into an adduction and an abduction variety. This distinction has no therapeutic importance and in any event is probably based upon a radiological illusion. The greater tuberosity may be avulsed totally from the rest of the humerus and is then occasionally drawn proximally by the short rotators. More commonly the fracture fragments are held to each other by the soft tissues enclosing the proximal end of the humerus and are impacted into each other. All the fragments have a good blood supply so that non-union is rare.

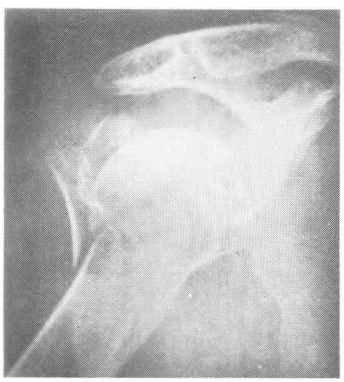


FIG. 368.—Comminuted fracture through the surgical neck of the humerus in an elderly subject.

Fractures through the anatomical neck may be combined with anterior dislocation of the head of the humerus from the shoulder joint. The head of the humerus then comes to lie as an isolated fragment anterior to the glenoid.

Clinical Features.—The patient complains of pain in the shoulder and usually cannot move the joint. Occasionally, however, in minor impacted fractures through the surgical neck, limited movement is possible. The diagnosis is made radiologically.

Treatment of fractures of the surgical neck is simple. The arm is rested in a sling until pain has subsided sufficiently to allow active mobilisation of the shoulder to be commenced. Fractures of the anatomical neck without dislocation of the shoulder should be treated in the same way. Shoulder stiffness following these injuries is usual: it commonly improves over a period of about 9 months but may be permanent.

The treatment of fractures through the anatomical neck of the humerus combined with anterior dislocation of the shoulder is difficult. The dislocated head cannot be reduced by conservative means. It is advisable to remove the head of the humerus, since it is totally avascular, and to replace it if possible with a Neer prosthesis.

If the greater tuberosity is fractured and has retracted substantially from the rest of the humerus under the pull of the short rotators, the fragments must be displayed surgically and the tuberosity attached to the rest of the humerus by suture. Post-operatively the patient's arm should be rested in a sling and active abduction deferred for at least 3 weeks.

Charles Sumner Neer, Contemporary. (*See footnote p. 252.*)

Fracture of the Outer End of the Acromium.—This injury is uncommon and occurs following direct violence to the acromium. Treatment is symptomatic.

Fracture of the Glenoid.—This fracture is uncommon. Treatment is by early mobilisation of the shoulder joint.

Dislocation of the Shoulder

This is a not uncommon injury. The head of the humerus usually dislocates forwards from the shoulder joint so as to lie beneath the coracoid process, to produce a sub-coracoid anterior dislocation of the shoulder (fig. 369).

The injury is produced by forced extension and external rotation of the abducted arm. The head of the humerus is forced against the anterior capsule of the shoulder and either ruptures the capsule or avulses the labrum from the glenoid. The latter injury, since the glenoidal labrum is avascular, rarely heals. Having torn the anterior soft tissues of the shoulder, the humerus passes over the anterior margin of the glenoid and in so doing the posterior part of the articular surface of the head may suffer a compression fracture. Once dislocated from the shoulder joint so as to lie beneath the coracoid, the humerus is locked in this position by muscle spasm.

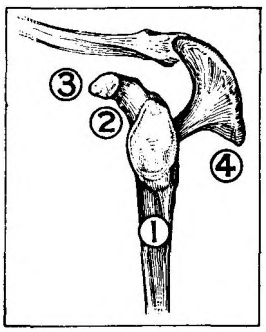


FIG. 369. — The positions taken up by the head of the humerus following a dislocation of the shoulder. 1. Sub-glenoid. 2. Sub-coracoid. 3. Subclavicular. 4. Posterior.

Rarely the humerus may dislocate posteriorly from the shoulder joint and very occasionally, particularly in women with poor musculature, may subluxate vertically downwards (fig. 369). Rarely the shoulder dislocates downwards with the arm in full abduction (Luxatio in Erecta).

The clinical features of anterior sub-coracoid dislocation are classical. The outer aspect of the shoulder is flattened and the arm appears to take origin from a point under the junction of the middle and outer thirds of the clavicle. The shoulder cannot be moved and the arm is held in a position of slight abduction (fig. 370).

Posterior dislocation of the shoulder may be difficult to diagnose clinically, since the appearance of the shoulder is essentially normal.

X-ray.—Radiologically an anterior dislocation of the shoulder may be diagnosed on an antero-posterior view because the head of the humerus can be seen to be lying beneath the coracoid (fig. 371). A posterior dislocation is less easily diagnosed since the head of the humerus does not move appreciably in a medial direction. Thus in an antero-posterior projection the appearances of the shoulder are not grossly abnormal. An axillary view, however, will display the head of the humerus lying either anterior or posterior to the glenoid.

Treatment.—Reduction of a dislocated shoulder is easy if the patient is given a general anæsthetic producing muscle relaxation. If the surgeon

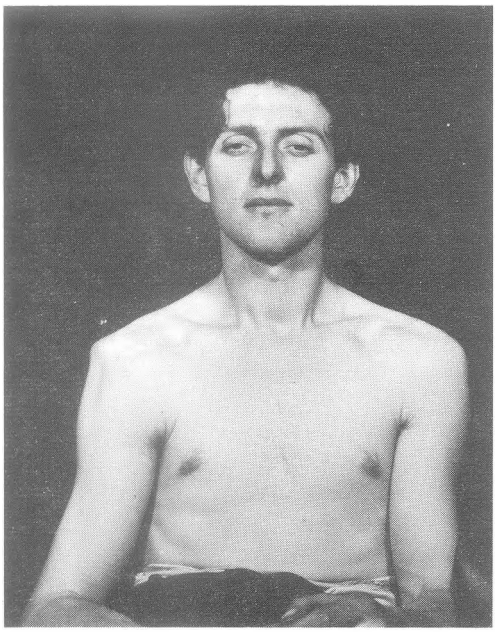
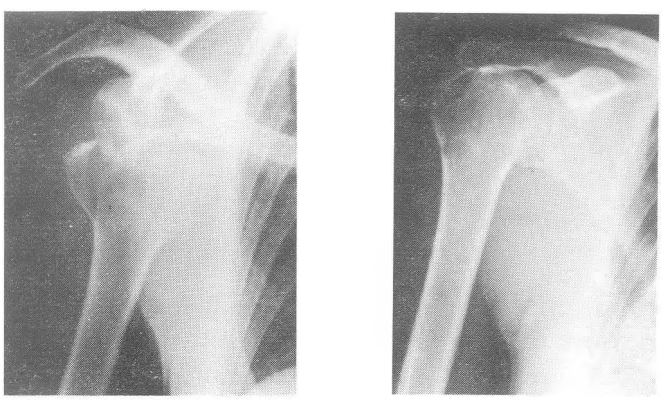


FIG. 370.—Sub-coracoid dislocation of the right shoulder.

is skilled, it is possible to reduce a dislocated shoulder without anæsthesia and even without analgesia, but if the surgeon is unskilled this will simply frighten and hurt the patient and possibly damage the shoulder joint. Thus in general it is desirable to anæsthetise the patient with muscle relaxation. When this is done reduction of a dislocated shoulder can be accomplished simply by pressure in an appropriate direction over the head of the humerus. There are however two classical methods by which an anterior dislocation can be reduced and it is occasionally necessary to resort to one of these manœuvres even in a patient whose muscles are relaxed. The more satisfactory of the two is the method described by Kocher.



(*a*) (*b*)

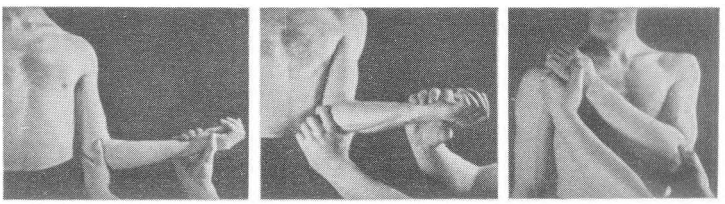
FIG. 371.—(*a*) Sub-coracoid dislocation of the shoulder with fracture of the greater tuberosity of the humerus. (*b*) Satisfactory reduction.

Technique of Reduction of an Anterior Dislocation of the Shoulder (Kocher's Method)

The patient lies on his back. If the left arm is injured, the surgeon stands on the patient's left side and grasps the injured elbow with his left hand. Traction is exerted in the long axis of the humerus for approximately 2 minutes and then, using his right hand, the surgeon gradually externally rotates the patient's humerus by flexing

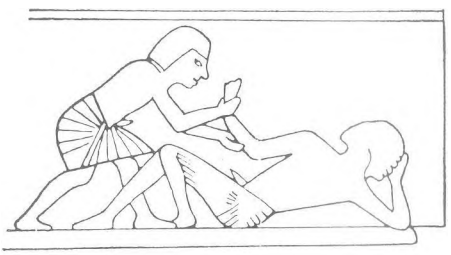
Emil Theodor Kocher, 1841–1917. Professor of Surgery, Berne. Described this method of reducing a dislocated shoulder in 1870.

the elbow and using the forearm as a lever. When full external rotation has been obtained, traction is maintained and the elbow is brought across the patient's body so as fully to adduct the shoulder. The patient's hand is then brought across to his opposite shoulder so as fully to rotate the shoulder internally (fig. 372). At some point during this manœuvre an obvious reduction is felt and the contour of the shoulder returns to normal. Reduction is confirmed by putting the shoulder through a full range of movement under anæsthesia.



(*a*)

FIG. 372 (*a*).—Kocher's method for the reduction of a dislocated shoulder.



(*b*)

FIG. 372 (*b*).—Kocher's method is 3,000 years old. Detail sketch from wall painting in the Metropolitan Museum, New York, from Ramesside Tomb. (*M. K. Hussein, F.R.C.S., Cairo from Jnl. of Bone & Joint Surgery. By permission of E. & S. Livingstone.*)

The arm is then immobilised in a sling and bandaged to the side so as to maintain full adduction and internal rotation of the shoulder for a period of 3 weeks. At the end of 3 weeks the sling is removed and progressive mobilisation of the shoulder commenced, avoiding only external rotation in abduction. (The position in which the shoulder dislocates.)

Recurrent Dislocation of the Shoulder

If correctly treated, the majority of dislocations of the shoulder heal to give no further disability. Occasionally the shoulder becomes unstable and recurrently dislocates after trivial violence. Finally the patient may acquire the knack of actively dislocating and reducing the shoulder for himself. If the patient cannot reduce the dislocation for himself, and indeed even if he can, this instability may cause significant disability and require treatment.

Treatment.—Recurrent dislocation of the shoulder is treated by operation. An anterior approach is made to the shoulder. The original lesion in the capsule or glenoidal labrum is displayed and repaired. The subscapularis muscle is then overlapped in front of the joint so as to reinforce the anterior aspect of the joint and thereafter to limit external rotation. Alternatively the coracoid process, with the coraco-brachialis and biceps muscles attached to it, is transposed to the anterior lip of the glenoid so as to form a bone block which prevents further anterior dislocation of the shoulder.

Recurrent posterior and downward dislocation of the shoulder occurs occasionally, and is treated (but with difficulty) by operation.

Acromio-Clavicular Dislocation

This injury is due to traction on the arm or to a violent downward blow on the point of the shoulder. Dislocation of the acromio-clavicular joint can only occur if not only the capsular ligament of the acromio-clavicular joint, but also the coraco-clavicular ligament, ruptures. If both these structures are torn, the scapula falls under the weight of the arm whilst the clavicle is drawn upwards by the pull of the sternomastoid muscle.

Clinical Features.—An obvious prominence is present at the outer end of the clavicle where this bone is displaced upwards relative to the acromion. The acromio-clavicular joint is tender.

An antero-posterior X-ray should be taken of both acromio-clavicular joints so as to compare one with the other. The presence of a step is diagnostic of an acromio-clavicular dislocation (fig. 373).

FIG. 373.—Dislocation of the acromio-clavicular joint.

Treatment.—Primary treatment of the dislocation is difficult. Strapping can be applied so as to lift the point of the elbow relative to the clavicle, but this is very uncomfortable, hard to maintain, and if really effective may cause pressure sores. It is preferable to reduce the dislocation by passing a screw through the clavicle to engage in the coracoid and thus draw the coracoid up to the clavicle. The screw should be removed about 3 months following the injury since it will inevitably work loose as the scapula moves on the clavicle.

Since (1) the disability from this injury even if untreated is slight (although in women the prominence of the clavicle may be thought to be unsightly), (2) conservative treatment is usually ineffective and (3) operative treatment leaves a scar, many surgeons prefer only to rest the arm in a sling and then mobilise the shoulder when pain has settled. Osteo-arthrosis in the acromio-clavicular joint is an occasional late sequel which can be treated by excision of the outer end of the clavicle.

Fracture of the Clavicle

If this occurs lateral to the attachment of the coraco-clavicular ligament, displacement is rare since the two fragments of the clavicle are both fixed to the scapula. In the absence of displacement no specific treatment is required. If displacement occurs, a rupture of the coraco-clavicular ligament must accompany the fracture, and the injury should then be treated in an exactly similar way to an acromio-clavicular dislocation.

Fractures of the shaft of the clavicle medial to the point of attachment

of the coraco-clavicular ligament are common, especially in children and young adults. The injury is caused by direct violence and in children the fracture is often of the greenstick variety. In adults there is usually more angulation and displacement than in children. Significant displacement so that the bone ends are not in contact is rare, because the soft tissues attached to the clavicle tend to hold the bone ends together.

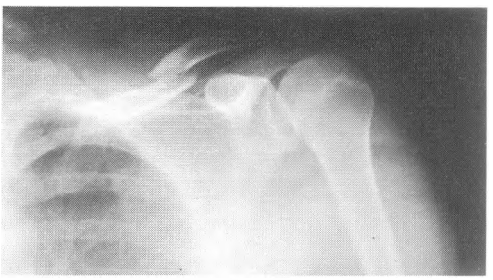


FIG. 374.—Fracture of the shaft of the clavicle with moderate angulation.

On clinical examination there is tenderness and often obvious angulation of the clavicle at the fracture site. The diagnosis is usually obvious radiologically (fig. 374).

Treatment.—No attempt should be made to reduce the fracture, since it is impossible to hold the reduction even if it is achieved. The time-honoured method of immobilising this bone is to apply clavicle rings or a figure-of-eight bandage to the two shoulders and a sling for the injured limb (fig. 375). Clavicle rings or a figure-of-eight bandage are invariably uncomfortable and probably do not contribute materially to immobilisation of the fracture. Many surgeons therefore rely only on a sling for the injured limb, combined with analgesics.

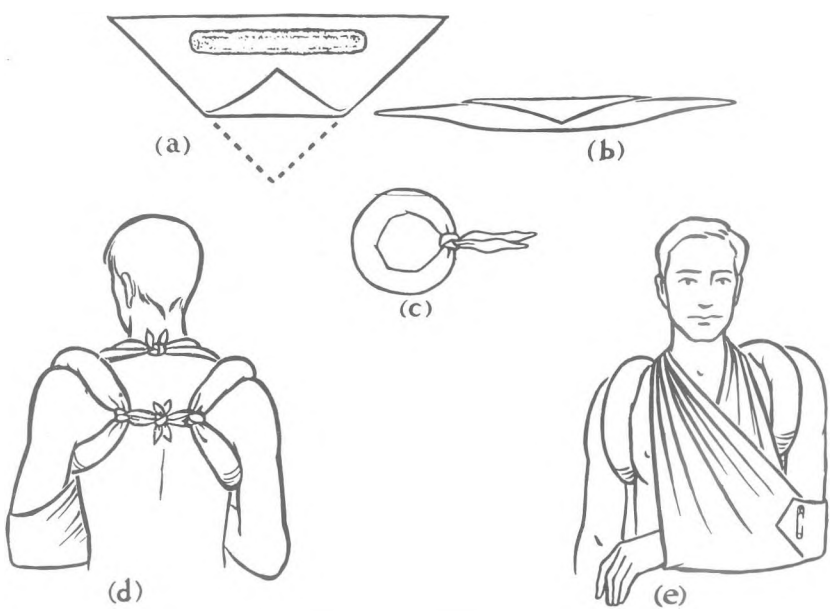


FIG. 375.—The 3 slings method for the treatment of fracture of the medial two-thirds of the shaft of the clavicle. Two of the slings have been fashioned to form clavicle rings.

The fracture almost always unites, but non-union may occur when there is substantial displacement at the fracture site. In these fractures a case can be made for immediate internal fixation of the fracture using a Kirschner wire introduced into the bone from its lateral end.

Established non-union can be treated by bone grafting if the non-union gives rise to pain. Fortunately, most ununited fractures of the clavicle are painless. Since it is difficult to achieve union even with a bone graft, non-union should not be treated unless it gives rise to symptoms.

Dislocation of the Sterno-clavicular Joint.—This is a rare injury in which the clavicle is displaced backwards relative to the manubrium. The clavicle may occasionally obstruct the trachea, making reduction urgent.

Fracture of the Body of the Scapula. Comminuted, stellate fractures occur in the body of the scapula following direct violence. They should be treated symptomatically by resting the arm in a sling until the pain has diminished enough to allow mobilisation of the shoulder.

CHAPTER 25

INJURIES TO THE LOWER LIMB

INJURIES TO THE FOOT

Fractures of the Metatarsals

Fractures of the shafts of the metatarsals are caused by crushing injuries, for example by weights falling on the feet or the wheels of vehicles running over them. When these fractures accompany severe crushing of the foot, the broken bone may be the least significant part of the injury: the soft tissues are also crushed so that a not-infrequent end result is a foot which is thickened, stiff and painful, even though the fractures have united satisfactorily.

If only one or two metatarsals have fractured, reduction is not required unless some part of the shaft, in a grossly displaced fracture, is projecting into the sole of the foot. Splintage is also unnecessary and early mobilisation with non-weight-bearing exercises is to be encouraged. Return to activity is achieved by taking weight on the heel as soon as the patient feels he can, which may be in 2 to 3 weeks. Full function is possible in 8 to 12 weeks.

If more than two bones are broken, the fractures are usually unstable and the foot must be immobilised in a plaster boot for 4 to 6 weeks. Active mobilisation of the foot is required thereafter.

Avulsion of the styloid process of the fifth metatarsal is a common, minor injury which causes unnecessary inconvenience if treated in a walking plaster. The styloid process is avulsed by the pull of the peroneus brevis contracting during forced inversion of the foot. The fragment is frequently detached by 2 mm or more, but even if bony union does not occur, a fibrous non-union is always symptomless. The foot should not be immobilised and it is unnecessary to apply anything more than adhesive strapping to support the foot and control swelling.

Fracture of the neck of the second or third metatarsal may occur not as a result of a single violent episode, but as a result of fatigue (*march fracture*). The fracture usually follows excessive walking—hence the term march fracture. It is predisposed to by a short first metatarsal which causes undue stress to fall on the necks of the second and third metatarsals during the toe-off phase of gait. Sudden pain, localised over the dorsal aspect of the bone, is characteristic. An immediate X-ray will often fail to reveal the crack, but if repeated in 3 weeks callus will be obvious (fig. 376). The only treatment needed is strapping and restricted activity. Many patients are probably treated as ' foot strain ', and more or less ignored with good results.

Fracture of the Talus

Fractures of the talus are uncommon but may occur as a result of a fall from a height. The neck of the bone is shorn through by the sharp anterior articular surface of the tibia, and the body of the talus may then be extruded from the ankle joint to be palpable under the skin. As in the case of the os calcis, considerable swelling rapidly develops and obscures the diagnosis, which is often established only after radiography. Infarction of the body of the talus is not uncommon and leads to non-union and osteo-arthrosis of the ankle (compare Fractures of the Neck of the Femur (p. 404) and of the Scaphoid (p. 357).

Treatment.—If the body of the talus is displaced, closed reduction, using skeletal traction through the os calcis, is sometimes successful, but usually operative reduction is necessary. Following reduction, the ankle is immobilised for 6 weeks in a plaster boot. Weight-bearing is avoided for 3 months in the hope of preventing deformation of the dome if it is avascular.

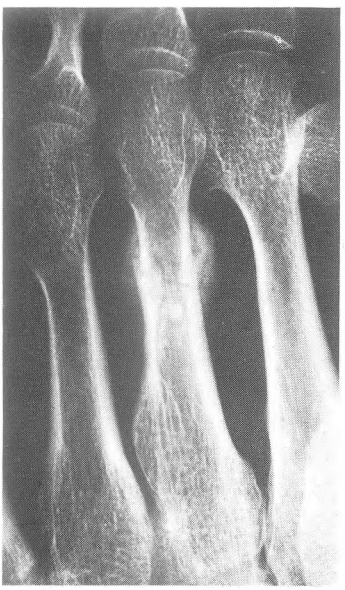


FIG. 376.—March fracture of the third metatarsal.

Fracture of the Os Calcis

Fractures of the os calcis are caused by falls from a height. The bone is shattered like an eggshell and the fracture usually involves the sub-talar joint (fig. 377).

The degree of displacement varies according to the violence. In some cases there is little or no displacement; in others the whole os calcis is flattened and widened. The Achilles' tendon tends to pull the posterior part of the bone upwards and the heel is always everted.

This injury, especially if it involves the sub-talar joint, is likely to leave the foot stiff regardless of treatment. As a consequence, walking on sloping of uneven surfaces is difficult and often painful. Though the foot is often painful for months, pain nearly always subsides eventually. Stiffness on the other hand is usually permanent, but tolerable to the patient.

Treatment.—Elaborate mechanical methods used to be employed in an attempt to restore the normal anatomy, but even when anatomically successful, such treatment does not improve the function of the foot. The best results are achieved by accepting the deformity and concentrating on early

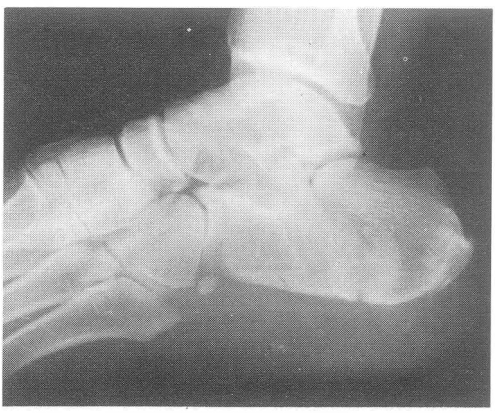


FIG. 377.—Fracture of the os calcis involving the sub-talar joint.

Achilles (*see footnote p. 331*).

non-weight-bearing mobilisation combined with elevation and compression by wool and crepe to control swelling. Weight-bearing is started in 8 to 12 weeks.

INJURIES TO THE ANKLE

Mechanism and Morbid Anatomy.—The ankle is usually injured by indirect violence: the foot being inverted, everted, externally rotated or internally rotated on the tibia. Rarely the ankle may be injured by a force which shears the talus transversely relative to the tibia, or which drives it vertically up into the tibia. Each of these mechanisms of injury produces a characteristic fracture pattern, so that these injuries can be classified, although with some over-simplification, on the basis of the mechanism.

Although the mechanism is described as if the foot moved (e.g. into inversion) on a fixed tibia, in practice these injuries nearly always occur with the foot fixed, for example by a hole in the ground or in a ski, whilst the tibia continues to move, driven by the momentum of the body weight. The foot is inherently more stable in eversion than in inversion and hence injuries due to inversion are more common than those due to eversion. Because during normal walking the feet are placed in a little external rotation relative to the line of travel, a sudden arrest of the weight-bearing foot whilst the tibia continues to move forward has the effect of externally rotating the foot on the tibia. (The student can demonstrate this for himself by walking, stopping his foot dead as it is placed on the ground, and noting the direction in which his tibia moves as he tends to fall forward.) Thus inversion and external rotation are the two most common mechanisms of injury.

The important factor in ankle injuries is the stability of the tibio-fibular mortice. If the mortice is stable, that is to say if no abnormal motion is possible between the tibia and the talus, the injury is relatively trivial. If abnormal motion is possible, the injury is potentially serious since persistent instability at an anatomically abnormal mortice causes pain and ultimately osteo-arthrosis of the ankle. Thus if the mortice is stable, the aim of treatment is to protect the ankle until healing has occurred; but if the mortice has been disrupted it must be reconstructed (reduced) and held (immobilised) until bone and soft tissue healing is complete.

External Rotation.—External rotation of the talus damages the ankle progressively, causing 1st, 2nd and 3rd degree external rotation or Pott's fractures.

1st Degree External Rotation Injury.—External rotation of the talus applies a twisting force to the lateral malleolus which suffers a spiral fracture starting distally at the level of the inferior tibio-fibular joint (fig. 378). The fracture fragment, which includes the malleolus, has a spike which lies posterior to a second spike projecting from the fibular shaft. The malleolar spike is attached to the tibia by the posterior inferior tibio-fibular ligament. Thus the lateral malleolus is attached to the tibia and the mortice is stable.

2nd Degree External Rotation Injury.—Further force either ruptures the posterior inferior tibio-fibular ligament or avulses its tibial attachment to produce the so-called ' third malleolus '. The talus is now free to turn in the mortice and as it does so the deltoid ligament avulses the tip of the medial malleolus to produce a low transverse medial malleolar fracture (rarely the ligament itself ruptures) (fig. 378). The mortice is now unstable.

Percivall Pott, 1714–1788. Surgeon, St. Bartholomew's Hospital, London. Described this fracture-dislocation of the ankle in 1765.

3rd Degree External Rotation Injury.—As the body-weight continues to move the tibia forward on the fixed foot, the talus, once the mortice has been broken, is free to slide beneath the posterior margin of the tibia. As it does so it may fracture the

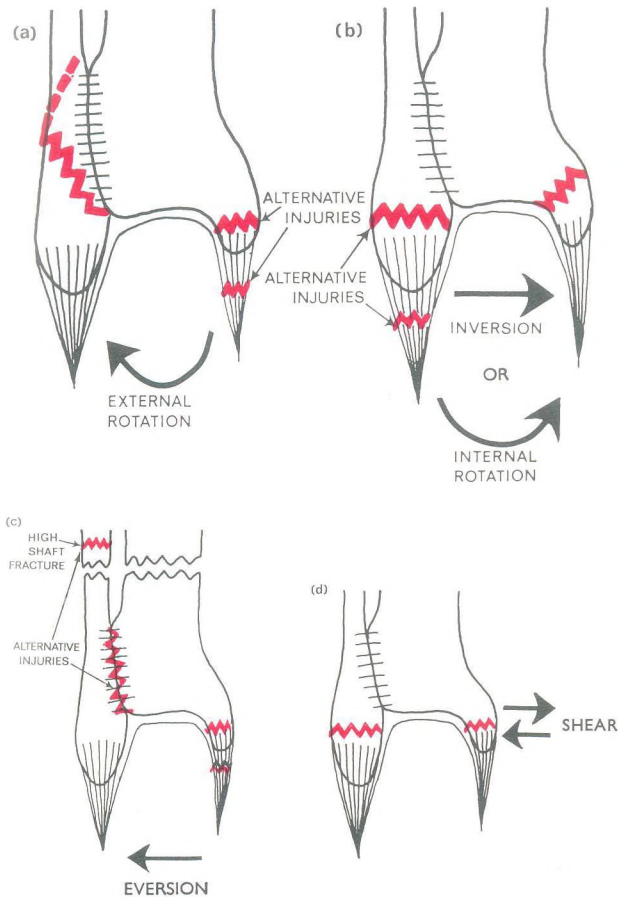


FIG. 378.—Varieties of ankle fracture.

margin producing a second variety of ' third malleolus '. Once the dome of the talus has passed posterior to the tibia, contraction of the calf muscles pulls the talus proximally and locks it in this position. Not only is the mortice now unstable but the ankle is dislocated as well (fig. 379).

Inversion (Adduction) Injuries.—In the plantar-flexed ankle, the talus is held in the mortice by the ligaments of the ankle. Thus if the ankle is forcibly inverted when plantar-flexed the lateral ligament may be sprained or ruptured (fig. 378). A rupture starts at the anterior fasciculus and extends backwards through the lateral ligament and medially through the anterior capsule, allowing the talus to tilt more and more in the mortice as it does so. Sprains and ruptures of the lateral ligament are the commonest ankle injuries and are usually loosely diagnosed as a ' sprained ankle '.

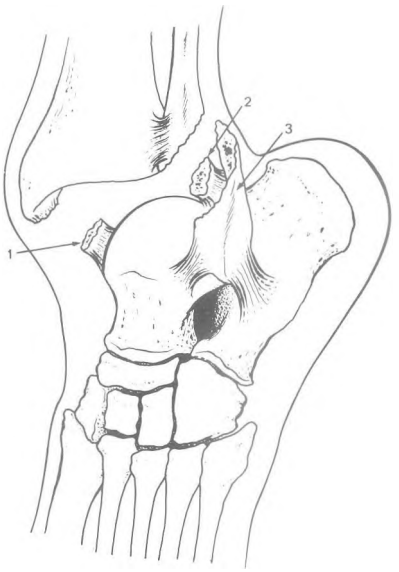
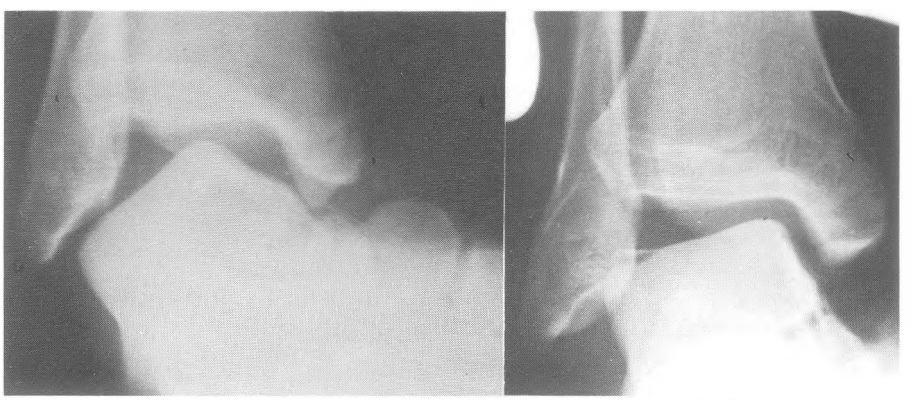


FIG. 379.—The morbid anatomy of a third degree external rotation injury of the ankle. Note that the medial malleolus (1), the third malleolus (2) and the lateral malleolus (3) are all attached directly or indirectly by ligaments to the talus or os calcis and hence that they move with the foot. (For clarity, the talus is shown in this figure as lying distal to the tibia. In fact, it lies behind the lower end of the tibia.)

Ruptures cannot be differentiated from sprains clinically but the distinction can be made by stress radiography (fig. 380).



(*a*) (*b*)

FIG. 380.—Inversion stress X-rays of two ankles with ruptures of the lateral ligament. (*a*) An AP view showing gross talar tilt indicating a complete rupture of the lateral ligament. (*b*) Less marked talar tilt, but note the transchondral fracture of the dome of the talus where the talus strikes the fibula. A routine AP X-ray of both these ankles revealed no abnormality.

The neutral or dorsiflexed talus is a tight fit in the bony mortice because the dome of the talus is broader anteriorly than posteriorly. Inversion of the foot and hence of the talus in this position therefore stresses the bones of the mortice as well as the ligaments. The medial malleolus may be sheared from the tibia—producing a fracture line running upwards from the root of the malleolus—and the lateral malleolus may then suffer a low transverse avulsion fracture at the level of the attachment of the lateral ligament (fig. 378).

The talus is now unstable in the mortice and further force may cause it to dislocate posteriorly as in third degree external rotation injuries.

Eversion (Abduction) Injuries.—Eversion of the talus levers the fibular malleolus outwards. If the inferior tibio-fibular ligament holds, the fibula may fracture high up in the calf since the shaft of the fibula is forced inwards as the long arm of a lever whose fulcrum is at the ankle. If the ligament ruptures, the mortice springs open as the fibula moves away from the tibia (fig. 378). This condition is described as a *diastasis of the inferior tibio-fibular joint*. The diagnosis may be suspected if the talus seems on an antero-posterior X-ray to be abnormally far from the medial malleolus, and is confirmed by stress radiography (i.e. by an antero-posterior radiograph taken under anæsthesia whilst the heel and hence the talus is stressed laterally relative to the tibia). If the ligament has ruptured, the mortice is unstable.

Continued eversion force may cause an avulsion fracture of the medial malleolus or rarely a rupture of the deltoid ligament as in external rotation injuries.

Internal Rotation.—This mechanism is rare and produces injuries similar to those produced by inversion.

Transverse Shear Injuries.—These are rare. They produce transverse fractures of the lateral and medial malleoli at the level of the top of the dome of the talus. Obviously the mortice is completely unstable.

Vertical Compression Injuries.—These are also rare but are occasionally caused by falls from a height. The talus is driven up into the distal tibia which suffers a comminuted fracture.

To deduce the mechanism of an ankle injury, the key is the radiological appearance of the fibular fracture (fig. 378). To summarise, this is as follows:

External rotation —spiral at the level of the inferior tibio-fibular joint.
Inversion —low transverse.
Internal rotation (rare) —low transverse.
Eversson —high in the shaft.
Transverse shear (rare) —high transverse.

Treatment

Ligament Injuries.—1. *The Lateral Ligament*.—Sprains of the lateral ligament should be treated by compression bandaging, early mobilisation within the limits of pain and swelling, and co-ordination exercises. Ruptures (which in everyday clinical practice are as common as simple sprains but are often not diagnosed as such since the diagnosis requires stress radiography) should be treated by the same regime save that for the first 3 weeks the patient should not bear weight on the injured foot.

2. *The Inferior Tibio-Fibular Ligaments (Inferior Tibio-Fibular Diastasis)*. —Rupture of these ligaments is best treated by internal fixation using a screw passed through the fibula into the tibia. Plaster-of-Paris is applied for 6 weeks post-operatively.

Fractures.—If the fracture or fractures are undisplaced, the possibility arises that the mortice is stable. If there is only one fracture on X-ray and no tenderness over a ligament clinically (for example an ankle with an isolated spiral fracture of the lateral malleolus and no tenderness over the deltoid ligament), the ankle can be regarded as stable and treated by compression bandaging and limited weight-bearing or by weight-bearing in plaster for 6 weeks.

If the injury is one which may be associated with instability, e.g. a fracture of both the lateral and medial malleoli, it is wise to examine the ankle using stress radiography or image intensification. If the talus can be made to move abnormally in the mortice by displacing the fractures, the mortice is unstable and should be treated as such. If not, the ankle is immobilized in plaster for 6 weeks during which time weight-bearing is limited.

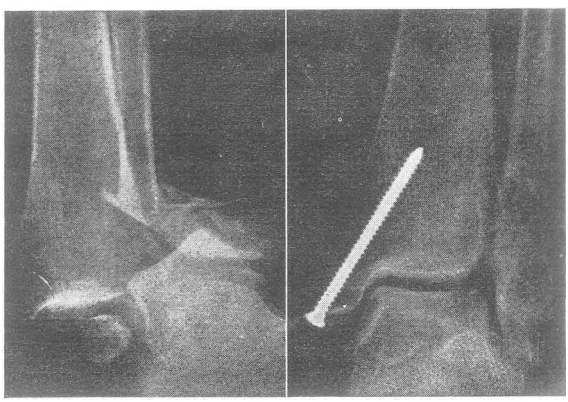


FIG. 381.—Third degree external rotation injury treated by internal fixation of the medial malleolus with a screw.

If the mortice is obviously unstable (fig. 381) or can be demonstrated to be so, most surgeons reduce the fracture and employ internal fixation. If a perfect reduction is achieved by manipulation, an above-knee plaster with the knee flexed to 30° (to control rotation) may be applied for 6 weeks as an alternative to internal fixation. During this time the patient does not bear weight on the foot and maintenance of the reduction is checked by weekly X-rays for 3 weeks. For a second 6 weeks weight-bearing is increased with or without a below-knee plaster. The danger of the first form of management (internal fixation) is that the fracture may be infected, of the second that the fracture may displace in plaster.

After a fracture-dislocation, most patients return to light work in 12 weeks and to heavy work in 6 to 9 months.

The Technique of Closed Reduction of a Fracture-Dislocation of the Ankle

As judged from the pre-reduction X-rays of a severely displaced fracture, it may appear unlikely that a satisfactory reduction could ever be obtained without operation. This impression is often misleading, reduction being possible because all the fragments are attached to the bones of the foot (fig. 379). The essential feature of the

reduction is to concentrate on restoring the alignment of the foot to the tibia, rather than on reducing the separate fragments by local manipulation of the individual fractures.

Third Degree External Rotation Injury.—The manipulation consists of reversing the direction of the forces which caused the original injury (a general principle in the reduction of all fractures) and since the injury is caused by external rotation of the foot followed by backward and then proximal displacement of the talus, reduction is produced by traction in the long axis of the limb so as to draw the talus clear of the distal end of the tibia, followed by forward displacement of the talus on the tibia, and then internal rotation and medial displacement of the foot. The talus cannot be 'over-reduced' anteriorly because the tissues posterior to the ankle will not permit it, nor into excessive internal rotation because the fibular 'spikes' interlock, nor medially because the dome of the talus strikes the root of the medial malleolus.

The anæsthetised patient lies supine with the knees flexed over the end of the operating table. The surgeon sits at the foot of the table and grasps the injured heel with his left hand (if the right ankle is injured) placing his thenar eminence behind the lateral malleolus (fig. 382). Steadying the tibia with the right hand, the surgeon then exerts traction on the injured foot and draws it forwards so as to bring the talus beneath the articular surface of the tibia. He then internally rotates the foot and displaces it medially until firm resistance is encountered. Having appreciated the sensation of reduction, a well-padded below-knee plaster is applied whilst an approximate reduction is maintained. The surgeon then grasps the foot and tibia as before, but now through the plaster, and repeats the manœuvre leading to reduction, taking care to hold the foot plantigrade as the plaster sets. If the ankle is very unstable, external rotation should be controlled by extending the plaster to the thigh with the knee flexed to 90° for 3 weeks. The plaster should be split and the patient admitted to hospital for 24 hours so that the circulation to the toes can be observed.

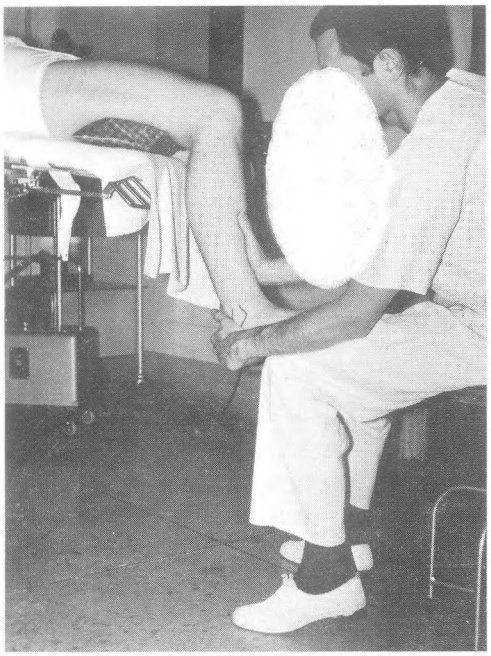


FIG. 382.—The technique of reduction of a third degree external rotation injury.

The Technique of Internal Fixation of Fractures of the Ankle.—A number of implants are available for the treatment of these fractures the details of which are beyond the scope of this text. The essentials, however, are: (1) to operate either before swelling has developed or after it has subsided, i.e. after 10 days of elevation and immobilization; (2) to employ a scrupulously aseptic technique; (3) to concentrate particularly upon secure fixation and accurate reduction of the medial malleolus (fig. 381) since the medial side is the key to the long term health of the ankle; and (4) to reduce the 'third malleolus' (see 3rd degree external rotation injuries, p. 382), if this is displaced and constitutes more than one-third of the articular surface of the tibia as seen in a lateral X-ray. After internal fixation the ankle may or may not require external splintage in plaster but weight-bearing should certainly be avoided for 9 weeks.

Rupture of the Achilles' Tendon

The Achilles' tendon is the commonest tendon to rupture, and since the injury is frequently diagnosed incorrectly, special note should be made of its

clinical features. It is an injury most common in middle-aged men and, though it can occur during games and severe exertion, it often happens as a result of a trivial stumble, the sensation of rupture being mistaken for a blow on the back of the leg. Such ruptures apparently occur because the tendon is ischæmic and weaker than normal. The rupture usually takes place about 3 cm. above the insertion of the tendon into the os calcis. A gap is easily palpable, and sometimes visible, at the site of the rupture (fig. 383). The foot can be dorsiflexed to a greater extent than the normal foot. The power of plantar-flexion is reduced so that the patient cannot stand on tiptoe on the injured foot, but it is to be noted that it is never completely absent. This is a most important point, because the surgeon can be led to imagine that the tear must be incomplete by the fact that some power of plantar-flexion exists. This power is, of course, exerted by the long flexors of the toes, the tibialis posterior, and the peronei. Very few cases of suspected ' partial ' rupture of the Achilles' tendon are in fact incomplete and it is dangerous to make this diagnosis because suture of the tendon is much more difficult if the operation is delayed.

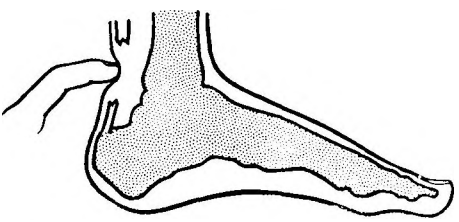


FIG. 383.—Palpable gap in the diagnosis of a rupture of the Achilles' tendon.

The diagnosis of ' rupture of the plantaris tendon ' is occasionally made in patients younger than those typically sustaining the classical Achilles' tendon rupture. It is probable that ' plantaris rupture ' is an imaginary condition, and that the clinical picture is that of a partial tear of the calf muscle fibres at a much higher level than the true Achilles' rupture.

Treatment.—Early suture of the Achilles' tendon followed by immobilisation of the ankle in a plaster cast for 6 weeks gives excellent results, but late suture is usually disappointing because the proximal end has retracted and lost its elasticity, so that the gap sometimes cannot be closed even by plantar-flexion of the foot and flexion of the knee (in which position the gastrocnemius and soleus muscles are relaxed).

FRACTURES OF THE SHAFT OF THE TIBIA AND FIBULA

Fracture of the Shaft of the Tibia Alone

This fracture is usually due to torsion causing a spiral fracture which may appear to be oblique on X-ray. Diagnosis is easy, as the bone is subcutaneous so that deformity and localised tenderness are readily detected.

Treatment.—When the fibula remains intact, the displacement of a fractured tibia is relatively slight, and as angulation is rarely more than 5°, it is usually sufficient to apply an above-knee plaster cast without any attempt at reduction. Though it might appear that fractures of the tibia when the fibula is intact would be unlikely to give rise to difficulty in treatment, in fact delayed union is not uncommon. It is probable that this is because the intact fibula acts as a ' strut ' to prevent the fragments of the tibia coming into close contact.

Fracture of the Shaft of the Fibula Alone

This fracture is usually due to direct violence or to eversion injuries at the ankle (p. 384). The procedure of ' springing ' the fibula assists diagnosis, and consists in compressing the fibula against the tibia, local pain being referred to the site of a fracture. A radiograph confirms the diagnosis, and treatment, if any is required, need be nothing more rigorous than the application of an adhesive elastic strapping.

Fractures of the Tibia and Fibula

These fractures occur as a result of either direct or of indirect violence. Road traffic accidents are the commonest cause of fractures due to direct violence: the bumper of a motor car strikes the leg and fractures the tibia and fibula by a mixture of impact and bending. The fractures occur at about the same level in the two bones, are transverse, oblique or (commonly) comminuted, and are frequently compound from without—the impact tearing the skin as well as breaking the bone.

Indirect violence may be applied to the tibia and fibula either in the form of torsion (the body turning during a fall on the fixed foot), or by bending (the foot and lower tibia being fixed while the body falls sideways, bending the tibia as it does so). The former injury is common in ski-ing, the latter in football. Both injuries may be compound, often trivially so, from within.

Fractures of the tibia are more commonly compound than those of any other bone and skin closure is particularly difficult because the bone is subcutaneous. When the skin has healed, compound fractures may be treated as if they had been simple in the first place. The treatment of compound fractures is discussed on p. 347.

Treatment.—Fractures of the bones of the leg can be treated in 3 ways:

(1) By plaster after manipulative reduction. This form of treatment is used for fractures in patients under the age of 16, and for fractures in older patients, in whom a stable reduction can be obtained. It is the most widely used and the safest form of treatment.

(2) By skeletal traction through the lower end of the tibia or the os calcis with the leg in a special frame or a plaster cast. Traction may render formal manipulation unnecessary. This form of treatment is used if a stable reduction cannot be maintained by plaster alone. A variant of this method, in which pins are passed through the tibia both above and below the fracture and are then fixed to an external metal frame, can be used to immobilise the fracture rather than to apply traction (fig. 344). It is particularly useful in the treatment of compound fractures.

(3) By internal fixation with plates and screws or an intramedullary nail. This form of treatment should only be used in expert hands. It is particularly indicated in multiple fractures and the elderly. It is contraindicated in compound fractures (p. 347). It has the advantage that the joints can be mobilised early, but the disadvantage that the fracture may be infected at operation.

The Technique of Closed Reduction of a Fractured Tibia and Fibula

Several methods are available, of which the following is the simplest if the surgeon is single-handed. The patient is anæsthetised to produce muscle relaxation and is placed supine with his knees flexed over the end of the operating table. The surgeon is seated facing the injured leg and the height of the table is adjusted until the foot just clears the surgeon's knee. An inelastic bandage is tied over plaster wool as a 'halter' round the ankle and looped under the surgeon's foot (fig. 384). The surgeon can thus exert traction on the fracture with his foot whilst leaving both his hands free to 'hitch' the bone ends. When this has been accomplished a well padded plaster is applied from below the knee to above the ankle and an X-ray is taken to check reduction—the bone ends should overlap by at least half their diameter and longitudinal alignment should be correct to within 10°; rotatory alignment is checked by inspection of the leg. If reduction is satisfactory, the bandage round the ankle is removed and the foot of the plaster is completed with the foot plantigrade. The knee is then placed in 5° of flexion and the plaster is applied up to the mid-thigh. Finally the plaster is split and the patient is admitted to hospital for elevation of the limb and observation of the circulation. Up to 10° of angulation at the fracture site can be corrected at any time in the next few days by cutting the plaster circumferentially at the level of the fracture, wedging it open on the concave side of the angulation, and then completing the plaster.

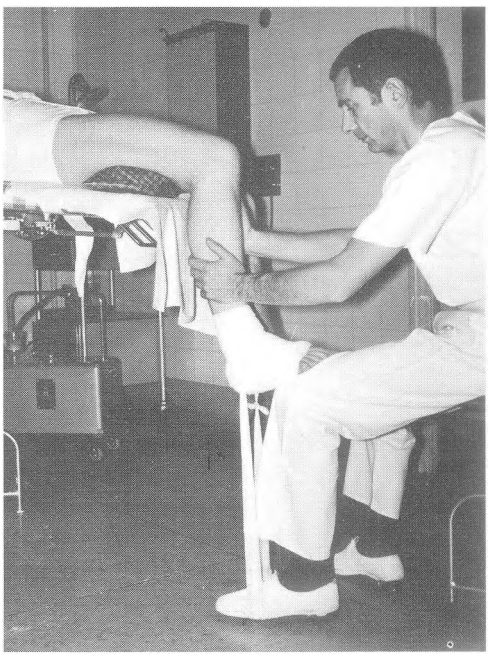


FIG. 384.—The technique of closed reduction of a fractured tibia and fibula.

The tibia usually takes about 12 weeks to unite. It is rarely fit to take weight without support under 16 weeks but if the fracture is stable, weight bearing may be allowed shortly after injury in a plaster cast, with the aid of internal fixation, or in an appliance (a 'cast-brace') composed of a cast around the shin to which is attached a brace which transmits some weight from the shoe directly to the proximal fragment.

Should the fracture be ununited at 3 months, further immobilisation in an above-knee plaster cast, preferably with weight-bearing, is required. If union has still not occurred after a further 6 weeks, immobilisation can be continued, but many surgeons prefer to reduce the duration of treatment by bone-grafting the tibia, with or without the addition of internal fixation (p. 346).

INJURIES TO THE KNEE

No joint sustains so wide a range of injuries as does the knee. This is because it is a commonly injured, major weight-bearing joint whose stability depends almost entirely upon soft tissues (ligaments and muscles). Its ligaments are both extra- and intra-articular and alone amongst the major

joints it contains menisci. Injuries to the knee may be divided into those which affect the ligaments, the menisci, the extensor apparatus, and the tibial and femoral condyles.

The Ligaments

The Mechanism.—A blow on the lateral side of the knee when the patient is bearing weight on the leg has the effect of abducting the tibia on the femur and stresses the medial collateral ligament. If the violence is relatively minor, some of the fibres of the ligament give way, but the ligament as a whole remains intact. Such an injury is a simple sprain. Should the violence be more severe, the ligament may be ruptured, usually at its femoral attachment. Because the medial meniscus is attached to the medial collateral ligament, peripheral detachment of the medial meniscus is commonly associated with this injury. Further forced abduction of the tibia stresses the cruciate ligaments, one or both of which may be sprained or ruptured, and extends the rupture of the medial collateral ligament anteriorly and posteriorly through the capsule. If the medial collateral and cruciate ligaments are ruptured, the tibia becomes grossly unstable on the femur in abduction so that the knee can be subluxated.

The reverse mechanism (i.e. adduction of the tibia on the femur) is far less common (because the knee is more commonly struck on its lateral than on its medial aspect) and results in an injury first to the lateral collateral and then to the cruciate ligaments.

The posterior cruciate ligament may be injured if the anterior aspect of the tibia is struck when the knee is flexed, an injury which not uncommonly occurs to the front-seat passenger in a motor car if he is thrown violently forward against the dashboard.

Isolated anterior cruciate ruptures may occur if the knee is forcibly hyperextended.

Clinical Features.—It is important to obtain an exact description of the injury and of the ensuing events, since this often helps to distinguish one soft tissue lesion from another. A description of the injury itself will suggest the mechanism and hence, as indicated above, the pathology. If bleeding has occurred into the joint (a hæmarthrosis), the knee swells within 1 or 2 hours and this suggests the presence of serious intra-articular damage. Should the knee swell, but only over the course of 12 or 24 hours, the swelling is likely to be due to an effusion. This implies the existence of a less severe injury. As fluid collects in the joint, the knee becomes increasingly painful and movement becomes more difficult. When a moderately large effusion has developed, only a few degrees of movement are possible and the knee is held in about 10° of flexion (a situation which must be distinguished clinically from locking of the knee (p. 393)).

Physical Signs.—Soon after the injury an effusion or hæmarthrosis can be demonstrated. Localised tenderness can be elicited over the collateral ligaments if these are injured. No clearly localised tenderness is demonstrable if a cruciate ligament is injured. It is vital that the student should have an

accurate understanding of the surface anatomy of the knee, since the diagnosis of soft tissue injuries and the distinction of meniscus injuries from ligament injuries depends upon the accurate localisation of tenderness in relation to the underlying structures (fig. 385).

If a ligament has been sprained rather than ruptured, stressing the ligament will not demonstrate abnormal movement but will provoke pain in the injured ligament. Thus, for example, if the tibia is abducted on the femur, pain will be elicited from a simple sprain of the medial collateral ligament. If the ligament has ruptured, the knee as a whole may be too painful to allow the ligament to be stressed. The knee should then be examined under anæsthesia: the tibia in nearly full extension is abducted and then adducted on the femur and any abnormal movement which occurs is compared with that in the uninjured knee. Excessive movement suggests the presence of a rupture of the appropriate collateral ligament and may be recorded by taking a stress X-ray of the knee. If the tibia is flexed to 90° on the femur, the anterior cruciate ligament resists forward glide of the tibia on the femur: excessive forward glide therefore indicates a rupture of the anterior cruciate ligament. Excessive posterior glide indicates a rupture of the posterior cruciate ligament.

If a ligament injury, especially a rupture, is missed soon after injury and hence goes untreated, the pain will settle, but the knee will not become sign- and symptom-free. The patient complains that the knee feels secure and it may be recurrently painful and swollen. If the joint is examined some months after injury, mechanical instability and often an effusion may be demonstrated and marked quadriceps wasting will be seen.

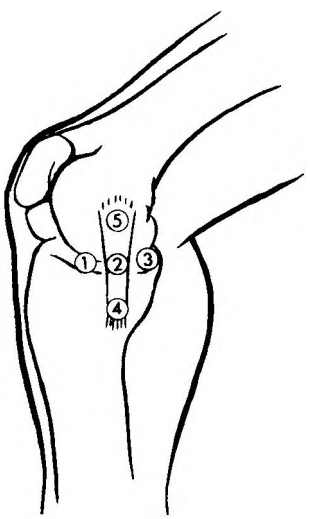


FIG. 385.—Sites of localised tenderness in knee injuries: 1, 2 and 3 on the joint line suggest tears of the medial meniscus; 4 and 5 suggest sprains or ruptures of the tibial or femoral attachments of the medial collateral ligament.

Radiological Appearances.—At the time of injury, there are usually no abnormal radiological features. The cruciate ligaments, when stressed, may sometimes avulse their tibial attachment rather than rupture in their length and the lateral collateral ligament may avulse its fibular attachment: the resultant fractures can be seen radiologically, especially if the bone fragment is significantly displaced. Rarely, there may be slight persistent separation of the medial femoral and tibial condyles if the medial collateral ligament has ruptured and become interposed in the joint space. Some weeks following injury, new bone formation may be seen at the femoral attachment of a collateral ligament if the periostium has been avulsed at the time of injury (a condition known as Pellegrini-Stieda's disease).

Treatment of ruptures of these ligaments is controversial, both immediately after injury and later should instability persist.

The cruciate ligaments when ruptured never lie with their ends in apposition. The collateral ligaments also commonly recoil. Therefore if simple

Augusto Pellegrini, Contemporary. Surgeon, Ospedale Mellini, Chiari, Italy.
Alfred Stieda, 1869–1945. Professor of Surgery, Konigsberg, which was in Germany until 1945 but is now Kaliningrad in the U.S.S.R. Pellegrini described traumatic calcification of the collateral tibial ligament in 1905; Stieda published his account of the condition in 1908.

immobilisation is employed, the ligaments may not heal because their ends are not apposed. For this reason many surgeons explore a rupture of any ligament of the knee immediately after injury so that the ends of the ligaments can be sutured together. This operation is technically difficult in the case of the cruciate ligaments, but relatively simple for the collateral ligaments. Following operation the knee is immobilised in a few degrees of flexion in a plaster cast extending from the groin to the ankle for 3 to 6 weeks. Alternatively the knee may be immobilised, without operation, in the hope that the ruptured ligaments will then unite spontaneously. Whilst in plaster, quadriceps exercises are begun and as soon as the plaster is removed the knee is actively mobilised and the thigh muscles exercised. Weight-bearing should be deferred for 3 weeks from the time of injury.

Simple sprains of the collateral ligaments require only limitation of normal activities combined with mobilisation and quadriceps exercises for 3 to 6 weeks.

Chronic Ligament Injuries.—If a rupture of a ligament of the knee fails to heal normally, the knee may no longer be mechanically stable. It is then liable to ' give way ', especially when stressed in sport, to swell and to be intermittently painful. The instability often results in repeated injuries so that the condition tends to become worse as time goes by. This is a very serious state of affairs for professional sportsmen.

As a result the subject of knee injuries and their sequelæ in sportsmen has almost become a subspeciality in orthopædics and represents a large part of sports medicine.

Because of the complexity of the ligaments stabilising the tibio-femoral and patello-femoral joints, because the same symptoms (i.e. ' giving way ', swelling and pain) can be caused by meniscal injuries or loose bodies, because more than one lesion may coexist in the knee, and because the exact nature of the instability which occurs when the patient runs (for example abnormal, uncontrolled rotation of the femur on the tibia in flexion) may be difficult to demonstrate on the examination couch, these lesions are hard to diagnose and even harder to treat. Experts in the field are not in full agreement but, in general, clinical and radiological examination is used to define the nature of the lesion and the resultant instability. This is then controlled, if possible, by a brace or by surgery. The latter may involve an attempt to re-suture ruptured ligaments but since this is often difficult or impossible, surgery usually takes the form of the transfer of tendons in the hope of restoring the function of the ligaments by substitution. The use of prosthetic ligaments has been tried but so far without great success.

The Menisci[1] (The Semi-Lunar Cartilages)

Mechanism.—The menisci are commonly torn in sports injuries or when rising from a kneeling position. Tears of degenerate menisci occur in the osteoarthrosic knee. The mechanism is rotation of the tibia on the femur in the flexed, weight-bearing knee. This sequence of events comes about, for

[1] Meniscus (Greek) = a crescent.

example, if a games player stands on one leg with the knee slightly bent and then twists his body. The medial meniscus tends to be damaged if the femur is internally rotated on the tibia, the lateral meniscus if the femur is externally rotated. Medial meniscus injuries are more common than lateral meniscus injuries in a ratio of about 20 to 1 and both are rare in women.

Morbid Anatomy.—Tears may occur in the anterior horn, the posterior horn or as bucket-handle tears in the body of the meniscus (fig. 386). Following an initial tear, progressive damage to the meniscus is common, so that eventually the original anatomy of the tear may not be discernible. The menisci are avascular and once torn do not heal.

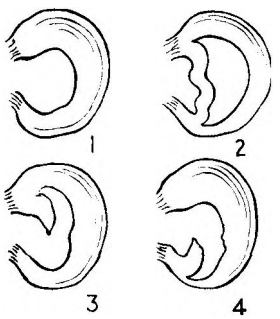


FIG. 386.—Tears of the meniscus. 1. Normal. 2. Bucket-handle. 3. Posterior Horn Tear. 4. Anterior Horn Tear.

The lateral, and commonly the medial, meniscus may occasionally be congenitally discoid in shape rather than semi-lunar. Discoid menisci are more prone to tear than the normal meniscus and, even if untorn, occasionally give rise to very loud ' clunks ' in the knee.

Clinical Features.—Patients suffering meniscus injuries may not necessarily attend a doctor at the time of the original injury. Physical examination of the knee is often not diagnostic and the diagnosis rests largely upon the history. In the history the important features are an accurate description of the original injury, a complaint of locking and unlocking, and the lateralisation of any pain or clicks.

The mechanism of the injury has already been described. Following injury the knee swells overnight as an effusion collects, and usually comes to be held in about 10° of flexion, from which position movement is painful. As the effusion settles, the range of movement gradually increases until after about 6 weeks it is nearly full. However, at the time of the injury the torn portion of the meniscus may become interposed between the femoral and tibial condyles so as mechanically to prevent either full extension or (much more rarely) full flexion of the knee. (Such a mechanical block to flexion or extension is known as ' locking '.) The patient may notice this at the time of the injury but more commonly it is masked by the more obvious events of pain, swelling and the consequent limitation of movement. The displaced meniscus sometimes returns to its original position (so as to unlock the joint) as the effusion settles and thus the original episode of locking may never be noticed by the patient. Usually, however, it becomes obvious that the knee is locked as the effusion subsides.

Following a return to apparent normality, the knee may still be prone to two sequelæ: recurrent episodes of locking and ' giving way '. These events are attributable to displacement of the torn segment of the meniscus between the articulating femoral and tibial condyles. Such displacements may suddenly prevent either full extension or full flexion of the knee (' locking '). A sudden inability fully to extend the joint is always obvious to the patient

and is accompanied by pain in the appropriate compartment of the joint. The patient may not notice the contrast in the loss of the last few degrees of flexion. Sometimes the knee unlocks spontaneously or it may have to be manipulated by a physician or by the patient himself in order to regain a full range of movement. The history of sudden unlocking with a click located in one or other joint compartment is almost diagnostic of a torn meniscus (but it may be due to the presence of a loose body in the knee). 'Giving way' denotes a condition in which the knee suddenly flexes under the patient so that he falls to the ground, or, more commonly, the knee may feel as if it is about to give way (i.e. may feel unstable) without actually collapsing. This symptom may be caused by a torn meniscus or may be due to damage to the mechano-receptors in the capsule of the knee upon whose normal activity the co-ordination of the thigh muscles depends. Quadriceps weakness may produce the same symptom. (Since the stability of the knee is dependent upon the accurate, co-ordinated contraction of the thigh muscles, any injury producing muscular weakness and inco-ordination may cause a feeling of instability in the joint.)

Physical Signs.—Soon after injury the meniscus is tender. As with ligament injuries, the demonstration of such tenderness depends upon an accurate knowledge of the surface anatomy of the knee. Tenderness over the anterior or posterior horns of either meniscus cannot easily be confused with tenderness over any other structure, but tenderness over the central part of the meniscus cannot be distinguished from tenderness over a collateral ligament (fig. 385). Wasting of the quadriceps muscle is invariable within 2 weeks of injury. If the knee is locked, a full range of movement cannot be obtained and gentle attempts to force a full range will produce a sensation of elastic resistance and pain localised to the appropriate compartment of the joint. If a meniscus is torn, there will always be an effusion (albeit sometimes a small one) in the knee. Thus, the absence of an effusion excludes a meniscus injury. On the other hand effusions occur in other conditions so that their presence is not diagnostic of a meniscus injury. McMurray's sign (the demonstration of a painful click if the flexed tibia is rotated upon the femur) is sometimes positive. The distinction between a medial and a lateral meniscus injury depends entirely upon the localisation of tenderness and lateralising features in the history.

In summary.—It is often difficult to make a confident clinical diagnosis of a torn meniscus unless the knee is locked. The significant features in the history are recurrent locking, effusions and medial or lateral pain. On examination the diagnosis is suggested by an effusion, appropriately localised tenderness (and a block to full movement if the knee is locked).

There are no abnormal radiological features in routine X-rays but meniscal tears can be demonstrated by arthrography or by arthroscopy.

Treatment.—If a definite diagnosis of a torn meniscus is made, the meniscus should be excised since it will not heal spontaneously. Commonly, the diagnosis cannot be made with certainty and surgery should then be deferred. The patient is treated with quadriceps exercises so as to build up his thigh muscles

Thomas Porter McMurray, 1887–1949. Professor of Orthopædic Surgery, Liverpool, England.

and the knee is watched. Should symptoms suggestive of a meniscus injury persist, it may be inferred that the meniscus is torn, and the knee is investigated by arthrography or arthroscopy in an attempt to make a precise diagnosis before exploring the joint.

If the knee is locked at the time when it is first examined, the patient may rapidly be made more comfortable by manipulation of the knee (or menisectomy). No special manœuvre is needed: the patient is given a general anæsthetic producing muscle relaxation. The knee usually then falls straight and can be put through a full range of movement. Occasionally a click is encountered as the knee is put through a full range when the cartilage is displaced from between the condyles.

Menisectomy

The leg is exsanguinated using an Esmarch's bandage and an inflatable tourniquet. The cartilage is exposed by an oblique incision running from the appropriate femoral condyle downwards towards the tibial tubercle. The torn cartilage is steadied by a blunt hook and its anterior attachment to the tibia is divided. The cartilage is then grasped by forceps and pulled forwards, dividing its attachment to the coronary ligament. When the cartilage has been freed as far posteriorly as it can be displayed, it is dislocated into the inter-condylar notch and the posterior horn divided. The synovial membrane and capsule are sutured with plain catgut and the skin sutured with interrupted nylon sutures. A Robert Jones bandage is applied before the tourniquet is removed so as to limit bleeding into the joint. Post-operatively quadriceps exercises are started immediately.

Cysts of the Menisci.—A cyst of a meniscus is due to myxomatous degeneration occurring in the substance of the cartilage itself. The lateral cartilage is affected perhaps 20 times more commonly than the medial.

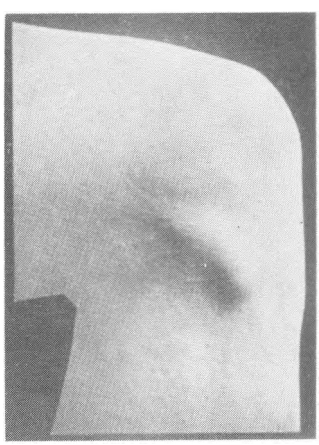


FIG. 387.—Cyst of the right lateral meniscus.

Clinically a tense swelling, often more obvious when the knee is flexed, appears over the joint line (fig. 387). The cyst sometimes appears suddenly. Pain follows exertion but may occur at rest. It is commonly described as being like toothache.

Treatment.—The cartilage together with the cyst should be removed if symptoms are persistent, but like a ganglion in any other site it can be ignored and often disappears spontaneously after a number of years. Local removal is unsatisfactory as recurrence is inevitable.

The Extensor Apparatus

The extensor apparatus of the knee comprises the quadriceps muscle, the tendinous insertion of the quadriceps muscle into the patella, the patella retinaculæ, the patella itself, the ligamentum patellæ and the insertion of the ligamentum patellæ into the tibial tubercle. This apparatus constitutes a single functional unit and provides the active extension of the knee which is

Johann Friedrich August von Esmarch, 1823–1908. Professor of Surgery, Kiel, Germany. Introduced this bandage in 1869 for use on the battlefield.

Sir Robert Jones, 1858–1933. Orthopædic Surgeon, Royal Southern Hospital, Liverpool, England. He was the nephew of Hugh Owen Thomas (see footnote p. 402) and did much to spread the knowledge of his uncle's work.

vital to normal gait. The apparatus may be injured anywhere in its length either by direct or by indirect violence (i.e. by a direct blow or by forced flexion of the knee when the quadriceps muscle is contracting).

Contusion of the quadriceps muscle results from direct violence. Localised pain follows attempts at contraction of the muscle and an extravasation of blood occurs within the muscle sheath. The extravasated blood may appear subcutaneously some distance from the site of the contusion, for example near the patella. Rest and pressure dressings are required for the first few days and thereafter the knee is progressively mobilised.

Contusion of the deep fibres of the quadriceps muscle may be associated with avulsion of the periosteum from the femur. The resulting hæmatoma may ossify to produce a radiologically demonstrable and occasionally palpable bony swelling on the anterior aspect of the femur (fig. 388). (This pathological process is sometimes referred to, wrongly, as myositis ossificans (p. 364).)

FIG. 388.—An ossifying hæmatoma overlying the femur.

Rupture of the muscle belly of the rectus femoris occurs at about mid-thigh level in young athletes as a consequence of very forceful, but resisted active extension of the knee. Extension of the knee is still possible after the pain of the rupture has subsided. When all local œdema has disappeared, the proximal end of the muscle stands out as a visible lump when the patient contracts the thigh. Distal to this lump there is a palable gap in the muscle. No surgical treatment is possible (since the muscle will not hold sutures), nor is surgical treatment necessary because the injury causes no permanent disability.

Rupture of the tendinous insertion of the quadriceps muscle into the patella is an uncommon injury which usually occurs in middle-aged and elderly patients, often after a trivial indirect injury such as stumbling. It may, however, occur in athletes. It is probable that the underlying pathology in the middle-aged is similar to that in ruptures of the Achilles' tendon, namely ischæmic necrosis of the tendon. The gap is easily visible and palpable a few weeks after injury (fig. 389) but is less obvious immediately after injury when the space is filled with hæmatoma, the tissues are swollen and the quadriceps inhibited. Soon after the episode the diagnosis may therefore be missed. The tendon should be repaired with mattress sutures.

Fractures of the patella may be due to indirect violence, i.e. to forced flexion of the knee when the quadriceps are contracting (in which case the fracture runs transversely across the patella) or to direct violence, for example

a blow on the anterior aspect of the flexed knee (in which case the fracture is usually comminuted, although sometimes only one or two fragments are broken from the main body of the bone). The latter fractures are described as 'stellate' and are sometimes compound.

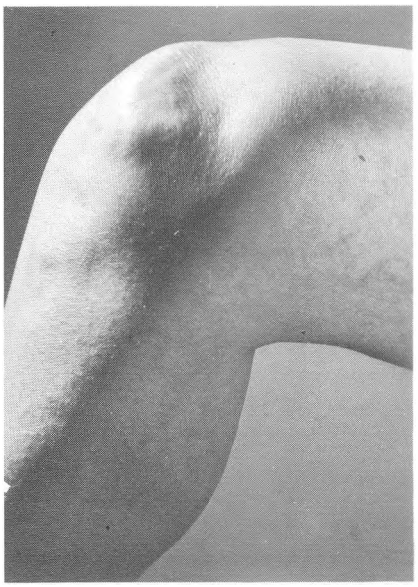


FIG. 389.—Rupture of the insertion of the quadriceps muscle into the patella.

Transverse fractures of the patella may be isolated injuries: the two fragments of the patella do not then become displaced since they are held in their normal position by the pre-patella expansion of the quadriceps tendon and the patella retinaculæ. Should force continue to be exerted on the extensor apparatus after the patella has fractured, the fracture may extend as a tear into these soft tissues. The quadriceps muscle then draws the proximal fragment of the patella proximally so that a substantial gap opens at the fracture site (compare Fractured Olecranon, p.366).

Undisplaced transverse fractures may be suspected by the demonstration of local tenderness and with care can be visualised radiologically (their demonstration is not easy since in an antero-posterior view the shadow of the patella is obscured by that of the distal end of the femur). Active extension of the knee is possible (but painful) without an extensor lag. Treatment is by immobilisation of the knee in a plaster cylinder for 6 weeks combined with quadriceps exercises, followed by mobilisation in a physiotherapy department.

Fractures with separation are obvious clinically because the gap is palpable. They are also obvious radiologically. Active extension of the knee is impossible. Treatment is by open reduction and internal fixation using a screw or wire (fig. 390). Should the fracture line traverse the patella so as to produce one large and one very small fragment it is often best to excise the small fragment rather than to attempt reconstruction.

Comminuted fractures of the patella cannot be accurately reduced and they therefore invariably result in significant irregularity of the posterior, articular surface of the bone. Such irregularity predisposes to osteoarthrosis of the patello-femoral joint. These fractures should therefore be treated by removal of all the fragments of the patella (i.e. by patellectomy) followed by reconstruction of the soft tissue components of the extensor apparatus. Following this operation, powerful, active extension of the knee is possible but there may be some loss of flexion. Surprisingly, the appearance of the knee is nearly normal. Vertical fractures separating one or two

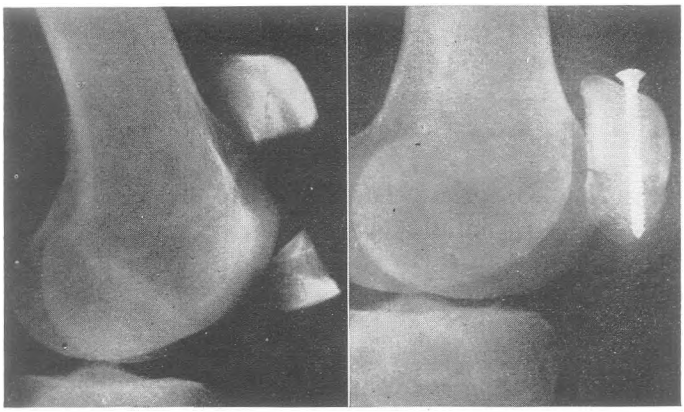


FIG. 390.—Transverse fracture of the patella fixed internally with a screw.

small fragments from the lateral or medial border of the patella can be treated symptomatically if the extensor mechanism is intact. (Radiologically, these fractures may be confused with congenitally bi-partite patellæ.)

Ruptures of the ligamentum patellæ occur, although rarely, in young athletes following extreme violence. The diagnosis is obvious, since the patella is drawn upwards to create a considerable, palpable gap. The knee is bruised, swollen and tender. Active extension is impossible. Treatment is by suture of the ruptured ligament followed by immobilisation of the leg in a plaster cylinder for 6 weeks.

Avulsion of the tibial tubercle occurs very rarely in adults. Minor avulsion injuries are not uncommon in adolescents, a condition known as Osgood-Schlatter's Disease (see p. 297 and fig. 302). A similar condition occurs, more rarely, at the distal pole of the patella and is known as Sinding-Larsen's disease (p. 297).

Dislocation of the patella over the lateral femoral condyle may occur following a direct blow on the medial side of the patella or without significant violence if some predisposing anatomical abnormality exists in the knee. Following direct violence, the medial patella retinaculum is ruptured and the patella passes over the lateral femoral condyle to lie on its lateral aspect. Once in this position, the patella remains there unless replaced by the patient himself or by a physician, and thus the knee is locked. The anatomical abnormalities which predispose to this condition are a small patella, a high patella, an unusually low lateral femoral condyle, marked knocked knee (so that the extensor mechanism tends to bowstring across the lateral aspect of the knee), or marked internal rotation of the legs (producing 'kissing patellæ'). Minor degrees of misalignment in which the patella does not actually dislocate may be responsible for some cases of Chondromalacia Patellæ (p. 248).

Once the patella has dislocated it is prone to do so repeatedly, and spontaneously. This condition is known as '*Recurrent Dislocation of the Patella*' and results in repeated locking or giving way of the knee. It is attributable

to chronic elongation or rupture of the medial patella retinaculum so that the transverse, distal fibres of the vastus medialis muscle no longer stabilise the patella in a medial direction when the quadriceps muscle contracts. The clinical features of the initial dislocation are obvious. Recurrent dislocation is also usually obvious, but occasionally if the dislocations reduce spontaneously the patient is not aware that the patella is dislocating and merely complains that his knee repeatedly locks or gives way.

The treatment of an initial dislocation is reduction of the patella, followed by immobilisation of the knee in a plaster-of-Paris cylinder for 3 weeks to give the medial patella retinaculum a chance to heal. Following removal of the plaster the knee is mobilised and particular attention is given to developing the quadriceps muscle.

Recurrent dislocation of the patella requires operative treatment. Numerous procedures have been described, but the most commonly employed is medial transposition of the tibial tubercle combined with release of the lateral patellar retinaculum and ' double-breasting ' of the medial retinaculum.

Fractures of the Tibial and Femoral Condyles

These injuries may be due to indirect violence (by a mechanism similar to that which produces ruptures of the collateral ligaments) or to direct violence:

1. If the tibia is abducted or adducted on the femur, either the appropriate collateral ligament ruptures in tension or a condyle on the opposite side of the knee collapses in compression. The result is a *depressed fracture of the tibial (or rarely femoral) condyle* (fig. 391). An associated tear of the meniscus is not infrequent.

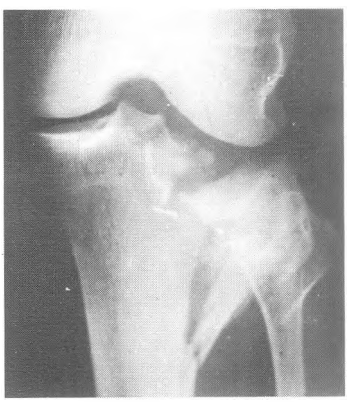


FIG. 391.—Fracture of the lateral tibial condyle with severe displacement. The neck of the fibula is also fractured.

2. Fractures due to direct violence are commonly produced by motor cars running into pedestrians. The bumper strikes the leg in the region of the knee and produces a *comminuted fracture* at the site of impact. The articular surfaces are frequently disrupted to produce a grossly irregular surface.

3. A direct blow applied to the anterior aspect of the flexed knee (i.e. to the patella) may drive the patella backwards into the femur, splitting the two femoral condyles from the femoral shaft so as to produce a ' *Y* ' *shaped fracture of the femoral condyles.* This fracture may also be produced by falls from a height on to the heels.

4. Finally a transverse *supracondylar fracture of the femur* may occur without involvement of the knee joint.

The diagnosis is suspected clinically by obvious deformity, bruising and swelling of the knee. The details of the injury are made out radiologically.

The treatment of fractures of the condyles is difficult. In young adults an attempt should be made to reconstruct the joint surfaces, but if comminution is extreme, this is often impossible. If reconstruction seems possible, it should be attempted by open operation with internal fixation of the fracture fragments. In more elderly patients or in young adults with minimally depressed fractures good results can be obtained conservatively either by treating the knee with early mobilisation on skeletal traction, or alternatively by immobilising the knee for 3 to 6 weeks in a plaster cylinder followed by active mobilisation in a physiotherapy department. Whatever form of treatment is employed, weight-bearing should be avoided for 3 months. Osteo-arthrosis is a common late complication.

Supracondylar fractures of the femur present a special problem, since the gastrocnemius muscle tends to flex the distal fragment, producing posterior angulation at the fracture site. This is very difficult to control conservatively, although it is possible to do so, using two Steinmann pins, one through the tibial tubercle to provide traction in the long axis of the femur and one through the distal fracture fragment to provide forward traction at right angles to the long axis of the femur. Preferably the fracture should be treated by internal fixation of the fragments using a blade-plate.

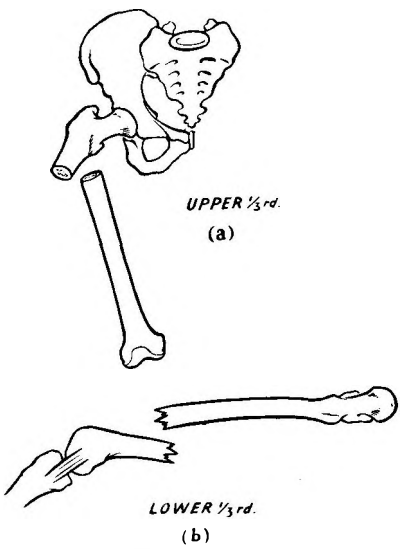


FIG. 392.—Characteristic deformities following fractures in the upper and lower thirds of the shaft of the femur.

FRACTURES OF THE SHAFT OF THE FEMUR

Fractures of the shaft of the femur occur in infants and young adults. In the latter the injury is usually due to severe violence so that associated injuries are common.

Displacement depends upon the direction of violence, muscular contraction and gravity. In the upper third of the shaft, the proximal fragment is flexed by the iliopsoas muscle, abducted by the gluteal muscles, and everted by the external rotators. The lower fragment is adducted by the adductor muscles, drawn proximally by the hamstrings and quadriceps, and everted by the weight of the limb. In middle-third and lower-third fractures, the deformity is one of backward angulation of the distal fragment and shortening (fig. 392).

The diagnosis is usually obvious clinically and is confirmed radiologically.

There is no obvious bleeding in a simple fracture of the shaft of the femur, but bleeding into the thigh—often in excess of a litre—is invariable in adults and such blood loss must be replaced.

Fritz Steinmann, 1872–1932. Surgeon, Berne, Switzerland. Described a pin for skeletal traction in 1907.

Treatment.—As in any other fracture, the aims of treatment are: (1) reduction (i.e. restoration of alignment and of length), (2) immobilisation of the fracture and (3) the prevention of knee stiffness.

1. *Restoration of alignment* is essential in the femur because mal-alignment throws an abnormal strain upon the knee joint, so that osteo-arthrosis is prone to develop in later life. Residual posterior angulation results in an extensor lag and hence in instability of the knee.

2. *Restoration of adequate length* is essential unless the patient is to wear an ugly raised shoe. Severe shortening tends to occur at the fracture site because (1) it is often produced by the initial injury, (2) the powerful thigh muscles pull the distal fragment proximally and (3) substantial bleeding at the fracture site is invariable and tends to ' balloon ' the thigh to produce an increase in girth and decrease in length. Full restoration of length is not necessary: up to 2 cm of shortening is acceptable as this amount is easily concealed by pelvic tilt.

3. *Immobilisation of the fracture site* can be achieved by traction and counter-traction, by internal fixation, or by plaster-of-Paris fixation (but in the adult this involves a very heavy, extensive plaster and is ineffective in overcoming muscle pull).

4. *Severe knee stiffness* is a common complication of fractures of the shaft of the femur, especially in the distal half, as a result of scarring in the thigh muscles and adhesion formation in the joint. Delayed union is especially prone to result in permanent knee stiffness. Distraction seems to cause non-union and it is preferable to have a femur which is a little short but united with a fully mobile knee, than a femur which is of full length but in which union is delayed and knee movement is limited.

Conservative Treatment (Traction and Counter-traction).—Because of the muscular contraction by the powerful muscles of the thigh, an untreated fracture of the femur may easily develop 10 cm of shortening (' over-riding '). The maintenance of length against this constant tendency to shorten is the special problem presented in this fracture. It is, therefore, essential to have a clear understanding of the principles of traction and counter-traction.

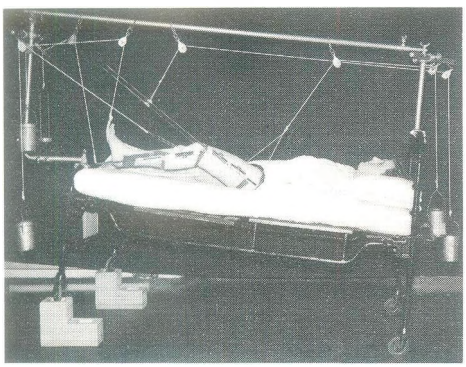
In most fractures, other than in those of the shaft of the femur, it is common practice to overcome shortening by a manipulation under anæsthesia and, if the bone ends can be successfully ' hitched ' against each other, to maintain this length merely by encasing the limb in plaster. This is quite impossible in the femur because, even if the fragments could be ' hitched ', the soft bulk of the thigh would allow them to slip again soon after the plaster was applied. If the fracture is oblique or comminuted, it is impossible to obtain stable end-to-end contact in the first place because the thigh muscles are so powerful. Thus the reduction has to be maintained by the continuous application of traction.

In applying traction to the distal fragment so as to hold the thigh to an acceptable length, it is always necessary to use counter-traction to the proximal fragment to prevent the trunk and pelvis following the traction force and again allowing the thigh to shorten.

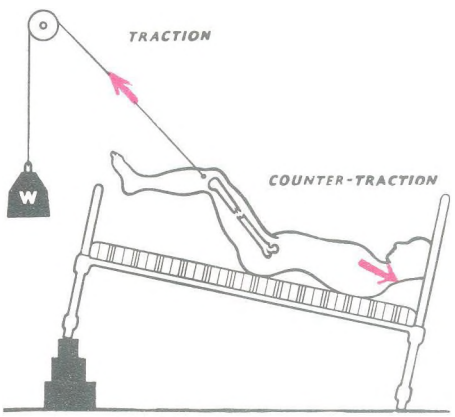
There are two entirely different methods of applying traction and counter-traction: (1) Sliding traction and (2) Fixed traction. The latter is now rarely used.

1. **Sliding Traction** (*syn.* Weight Traction, Balanced Traction).—There are many modifications of detail in applying sliding traction, but the essential principles are:

(*a*) Traction is applied to the distal fragment by weights and pulleys attached to the limb either by skin strapping or by skeletal traction. Skeletal traction is to be preferred in the adult as it is more comfortable and can be more powerful. It is secured by driving a 4 mm stainless steel Steinmann or, better, Denham pin through the upper end of the tibia. Skin traction is perfectly adequate in children.



(*a*)



(*b*)

FIG. 393.—The principal elements in sliding traction: traction and counter-traction.

(*b*) Counter-traction is provided by using the weight of the body. This is brought into action by elevating the foot of the bed so that the patient's trunk and pelvis tend to slide away from the source of traction. With a traction force of 9 kg. the foot of the bed is raised about 22 cm (fig. 393).

(*c*) The normal forward bowing of the shaft of the femur is maintained by support for the back of the thigh. Because of the tendency for the thigh to sag under the influence of gravity, the original deformity of backward angulation tends to persist, and must be controlled by slings or supports under the thigh.

Further apparatus permits knee movement, holds the foot in dorsiflexion (to prevent equinus deformity), and counterpoises the whole apparatus so that it 'floats' with the patient as he moves in bed.

Sliding traction is attractive because it is comfortable, and does not expose the patient to the risk of an infected fracture. The main danger of the method is that bone contact may be lost ('dis-traction') through excessive traction.

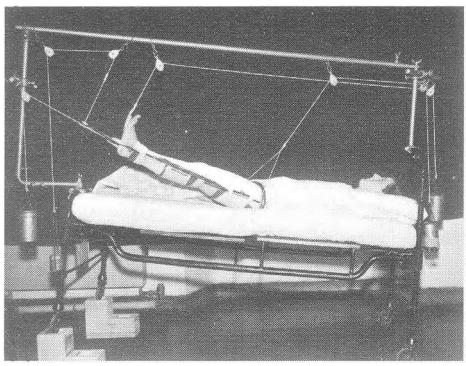
2. **Fixed traction** depends entirely on the use of the Thomas splint. Its modern application hardly differs from the original method used by H. O. Thomas nearly 100 years ago.

In this method of traction, cords are attached to the distal fragment, either by skin extensions or by a skeletal pin, and after passing the Thomas splint over the limb so that the padded leather ring takes a purchase against the ischial tuberosity, traction is applied by tying the cords to the foot of the splint. Counter-traction is thus exerted by pressure of the ring against the soft tissues and the ischial tuberosity (fig. 394).

Backward angulation of the shaft of the femur is controlled by slings passed between the side-bars of the splint behind the thigh. Fixation of the fracture is obtained by enclosing the limb and the splint in a bandage.

It is evident that this method can exert only a relatively slight traction force,

Robin Arthur Denham, Contemporary. Orthopaedic Surgeon, Royal Portsmouth Hospital, Portsmouth, Hants, England. The Denham pin is similar to the Steinmann save that it is threaded to obtain a better grip on the bone.
Hugh Owen Thomas, 1834–1891. Surgeon, Liverpool, England. Though he never held a hospital appointment, preferring to attend patients in their homes, he is regarded as the founder of orthopædic surgery. He introduced his splint in 1875.



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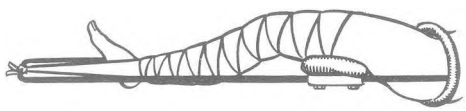


(*b*)

FIG. 394.—Fixed traction applied with a Thomas splint and adhesive strapping to the skin.

because the skin of the perineum would not be able to withstand a counter-traction force as great as that used in sliding traction. The great art of the method is therefore to hold the fracture with minimum force. To do this, the fracture must be capable of reduction under anæsthesia and the Thomas splint with fixed traction is then used merely to retain a reduction already achieved. This method has been modified by Bonnin, who adds skeletal sliding traction to fixed traction in order, not so much to treat the fracture (which is controlled by the fixed traction), but to draw the ring of the Thomas splint away from the groin.

Fixed traction provides more effective immobilisation of the fracture than does sliding traction and hence is said to result in a higher incidence of union. On the other hand, early knee stiffness is more marked after treatment by this method, although at the end of one year there is little difference in the range of knee movement obtained by either method.

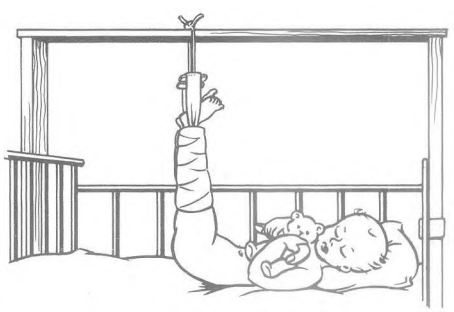


Fig. 395.—A 'gallows' splint.

Union.—A transverse or comminuted fracture takes about 12 weeks to unite if treated by traction. During this time the muscles of the leg must be exercised, at first statically but latterly with knee movement. Lateral mobility of the patella must be maintained manually from the moment of injury.

When the fracture is judged to be united, the traction is removed and the patient allowed to mobilise the leg in bed. If the fracture site remains comfortable, the patient is allowed up on crutches and gradually returns to full weight bearing.

Josiah Grant Bonnin, Contemporary. Formerly Orthopædic Surgeon, Central Middlesex Hospital, London.

A gallows splint (fig. 395) *is useful for children* below the age of 5 years. Traction is applied by means of strapping, and the legs are slung up to the crosspiece, so that the pelvis is just lifted from the mattress (a position which is very convenient for nursing purposes). The child's weight acts as counter-traction. Alternatively, children of this age can be treated in a plaster spica.

Operative Treatment: Internal Fixation.—Conservative treatment has the following disadvantages: (1) it involves prolonged treatment in hospital, (2) knee stiffness is common, (3) reduction may be impossible if muscle is interposed between the fracture fragments and (4) it requires constant intelligent attention to detail in order to ensure that the reduction is not lost and that pressure sores do not develop, e.g. over the Achilles' tendon. These disadvantages can be circumvented by internal fixation using a nail and plate in the subtrochanteric region of the shaft or an intramedullary (Küntscher) nail in the second and third quarters of the shaft (fig. 396). The nail can be inserted either by exposing the fracture site, or 'blindly' using X-ray control.

FIG. 396.—Küntscher intramedullary nail in the femur.

Provided the fracture is not too comminuted, no external splintage is required post-operatively and the patient can get up on crutches, progress to weight-bearing and start knee flexion within a few days of operation. These advantages have to be set against the occasional disaster of a surgically induced infection.

On balance, there is little to choose between conservative and operative treatment unless reduction cannot be achieved conservatively in a mid-shaft fracture, or the fracture is in the proximal shaft of an adult, for then the abduction and flexion of the proximal fragment is sometimes impossible to control conservatively. Operative treatment is never necessary in children.

INJURIES TO THE HIP

Fractures of the Proximal Femur

Since the days of Sir Astley Cooper these fractures have been divided into *intra-capsular* and *extra-capsular*. Intra-capsular fractures occur in that portion of the neck of the femur which lies within the capsule of the hip. Extra-capsular fractures occur in the remainder of the neck and through the trochanteric mass.

Intra-capsular Fractures of the Neck of the Femur

Pathology.—These fractures are anatomically sub-divided into subcapital and (more distally) transcervical. It is possible however that they all consist of a fracture starting superiorly at the junction of the head and neck (i.e. 'subcapital'), which propagates downwards through the head–neck junction

Gerhard B. G. Küntscher, 1900–1972. Medical Director, Hafen Hospital, Hamburg, Germany.
Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London.

until it encounters the medial trabecular system, and which may then be deflected distally by this column of strong bone so that it emerges inferiorly somewhere in the neck (i.e. to be ' transcervical '). Two features of this fracture make treatment difficult: (1) the blood supply to the proximal fragment is often impaired and (2) the proximal fragment can be neither manipulated nor immobilised by conservative means.

The head of the adult femur receives its *blood supply* via the femoral neck. Most of these vessels lie in the neck itself, entering it at about the level of the attachment of the capsule. All these vessels must be ruptured by an intra-capsular fracture of the neck. A few vessels lie subsynovially on the surface of the postero-superior neck and enter the bone subcapitally. These vessels are not always ruptured when the neck fractures, especially if the fracture is relatively distal. Thus the more proximal the fracture the greater is the chance of serious vascular damage. Similarly the greater the displacement which occurs at the fracture, the greater will be the vascular damage. A small, inconstant blood supply reaches the head through the ligamentum teres. This vessel may not be patent in the arteriosclerotic elderly and in such patients the vessels in the neck are more fragile. Thus the danger of an impaired blood supply in the proximal fragment increases:

(1) the more proximal is the fracture;
(2) the more displaced is the fracture;
(3) the more elderly is the patient.

A poor blood supply may result in (1) non-union and (2) avascular necrosis of the femoral head (see ' Complications ', p. 407).

These fractures are very much more common in elderly women in whom they are associated with seemingly trivial domestic stumbles. The fact that these fractures seem to be produced by very slight violence is perhaps due to the fact that osteoporosis has weakened the femoral neck. There is now however evidence that the fracture is due to fatigue in these patients, not to a fall; i.e. that the patient falls because the bone fractures, not that the bone fractures because the patient falls.

Rarely *fatigue fractures* may occur in the femoral neck of young adults exposed to unusual physical activites such as route marches with a full military pack.

In young adults the fracture may occur following severe violence applied in the long axis of the femur so as to shear the femoral neck. Such fractures may be associated with dislocation of the hip.

Displacement is usual at the fracture site and takes the form of external rotation and adduction of the distal on the proximal fragment. Occasionally subcapital fractures are impacted: no rotation then occurs and the fracture appears to be abducted, rather than adducted.

Clinical Features.—In unimpacted fractures the leg is short and lies in external rotation; there is tenderness over the fracture site in the groin; attempted movement of the hip is painful; and the patient is unable to lift the leg off the examination couch. In impacted subcapital fractures there is tenderness over the fracture site but otherwise there may be no abnormal

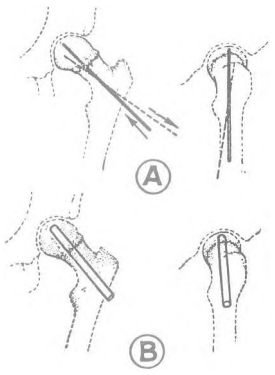


FIG. 397.—(*a*) Insertion of guide wire. The wire in the satisfactory position is retained. (*b*) The nail is inserted and the wire withdrawn. (*A.P. and lateral views.*)

physical signs. These fractures are therefore easily missed clinically.

An exact diagnosis requires X-ray examination. Even on X-ray, impacted fractures with minimal displacement may be missed.

Treatment is invariably operative because the proximal fragment cannot be controlled conservatively. In patients under 65 years of age, and in impacted fractures in older patients, the fracture is fixed internally using three pins, a tri-flanged nail (such as those designed by Smith-Petersen or Watson Jones), a blade-plate (see Extra-capsular Fractures, p. 408), or a sliding blade-plate in which the nail can slide through the plate so that, if collapse occurs at the fracture site as it unites, the nail can back out without fixation being lost nor the head being penetrated (fig. 399(*c*)).

The Technique of Internal Fixation.—A triflanged nail (with or without a plate) is inserted under X-ray control (fig. 397).

The patient is placed on a special operating table incorporating traction to the feet and counter-traction via a post in the perineum. The fracture can usually be reduced simply by placing the leg in abduction and internal rotation combined with traction. If the reduction is satisfactory (as checked by X-rays of the neck of the femur in two planes), a lateral incision is made over the great trochanter and a guide wire 2 mm. in diameter is inserted into the femoral neck. X-rays are taken, and if the direction of the wire is unsatisfactory, it is repositioned. The nail is provided with a central cannula which enables it to be slid over the guide wire and hammered into the head. A plate may then be bolted to the nail and screwed to the proximal shaft. The wound is closed and the patient is returned to bed without external splintage.

After operation the patient can be allowed out of bed in a few days, but weight-bearing is not permitted until some evidence of union is suggested in the X-ray—which may take 3 to 4 months.

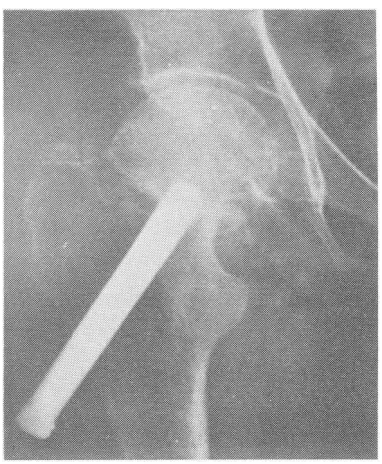
In unimpacted fractures in patients over 65 (or in younger patients with a shortened expectation of life) the chance of non-union or of avascular necrosis is substantial, and the ability of the patient to withstand a second operation (see ' Complications ') is slight. Thus these fractures are treated by replacement of the head and neck with a prosthesis (fig. 398).

FIG. 398.—Replacement of the femoral head and neck with a Thompson prosthesis.

Marius Nygaard Smith-Petersen, 1886–1953. Professor of Orthopædic Surgery, Harvard University, Boston, Mass., U.S.A. Introduced the tri-flanged nail in 1931.
Sir Reginald Watson Jones, 1902–1972. Orthopædic Surgeon, The London Hospital.
Frederick Roeck Thompson, Contemporary. Professor of Orthopædic Surgery, New York Polyclinic Medical School, New York, U.S.A.

Complications

Non-union occurs in approximately 30 to 40% of all intra-capsular fractures, partly because adequate immobilisation is difficult to achieve even by internal fixation and partly because the proximal fragment frequently has a poor blood supply. Non-union can be treated by:



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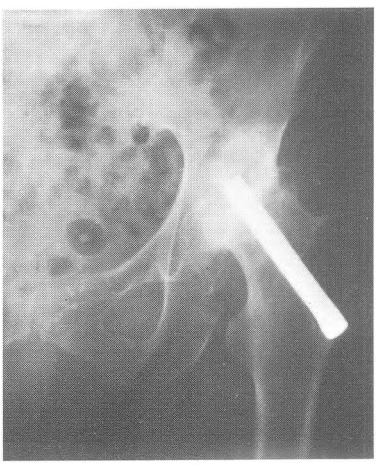

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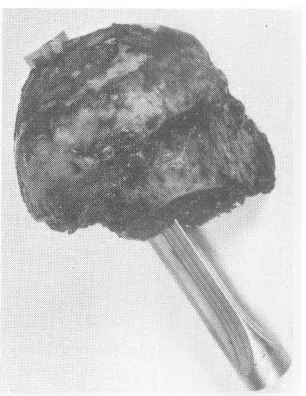

(*c*)

FIG. 399.—(*a*) Avascular necrosis of the head of the femur following an intra-capsular fracture. Little collapse has occurred in the head itself but collapse has occurred at the fracture site resulting in extrusion of the Smith-Petersen nail. Note that the femoral head is more dense than the surrounding bone. (*b*) Avascular necrosis. Here the head of the femur has collapsed. The pin has protruded from the femoral head rather than from the femoral shaft and damaged the acetabulum. (*c*) The operative specimen.

1. *Total Replacement of the hip.*—This is the treatment for the elderly.

2. *Subtrochanteric Osteotomy* with displacement of the shaft under the head. In this way the shearing stresses produced by weight-bearing are taken off the fracture and union may occur between the proximal fragment and the shaft. This method is only suitable for young adults and forms an alternative to bone grafting.

3. *Insertion of a fibular bone graft* into the neck across the fracture site and, at the same time, renewal of the internal fixation to ensure that it is adequate.

This method is indicated if the head is viable with an intact blood supply, and the patient is young.

Avascular Necrosis.—In this condition part or the whole of the femoral head, having become avascular, gradually collapses. The commonest cause of infarction of the femoral head is a femoral neck fracture but the condition also occurs in alcoholics, patients receiving steroid therapy, after irradiation, and spontaneously. For some unexplained reason the incidence of the spontaneous variety of avascular necrosis shows a wide geographical variation. Infarction itself is symptomless but weeks later infarction is suggested by a relative increase in radio-density of the infarcted bone, and months later its presence is proven by collapse of the infarct (fig. 399). At this stage the condition is termed avascular necrosis.

This process is painful, and because the femoral head becomes irregular in outline it predisposes to osteo-arthrosis. If pain cannot be relieved by a stick and a raise to the shoe, the femoral head is replaced with a prosthesis. The acetabulum should also be replaced if it has become secondarily damaged, for example as in fig. 399 (*b*) and (*c*). Avascular necrosis and non-union commonly co-exist.

Extra-capsular Fractures of the Proximal Femur

These fractures are divided anatomically into basal cervical, and pertrochanteric. In the latter the fracture is always comminuted and the main fracture line runs somewhere through the trochanteric mass. Both anatomical varieties have the same prognosis and are treated in the same way.

In contrast to intra-capsular fractures, all the bone involved in extra-capsular fractures—especially in those fractures involving the cancellous bone of the trochanters—has a good blood supply so that non-union and avascular necrosis of the femoral head are almost unknown. The proximal fragment can be controlled conservatively and hence operative treatment, although frequently used, is not mandatory.

Pertrochanteric fractures in the elderly are probably due to a sideways fall producing a blow over the greater trochanter. In the young they occur following violent trauma—for example in road traffic accidents. Basal cervical fractures are probably due to the same mechanism as that producing other cervical fractures.

Clinically the diagnosis is made, as in the case of the intra-capsular fracture, by the combination of external rotation and shortening of the leg, tenderness over the fracture, and the patient's inability to raise the leg from the examination couch. In pertrochanteric fractures the leg tends to be more externally rotated than in fractures through the neck.

Treatment.—It is a relatively simple matter to treat a pertrochanteric fracture without operation, merely by applying traction to the limb and confining the patient to bed for 3 months until the fracture is clinically united. The traction will restore length, and the deformities of adduction and external rotation are corrected by abducting and internally rotating the leg. This method of treatment is perfectly acceptable for young adults. Most of these

fractures, however, occur in the elderly and in this age group 3 months of bed rest would frequently result in *bed sores*, *pneumonia*, *venous thrombosis*, *pulmonary embolism and death*. Hence in this age group the fracture is invariably fixed internally so that the patient need not stay in bed.

A simple nail (such as the Smith-Petersen) is inadequate since it gains no purchase on the distal fragment. Accordingly a 'pin and plate' is used in which the 'plate' is screwed to the shaft and the 'pin' is driven into the neck of the femur (fig. 400).

The implant is inserted with X-ray control as described for intracapsular fractures. The patient is allowed out of bed, to walk non-weight-bearing on crutches, within a few days of operation.

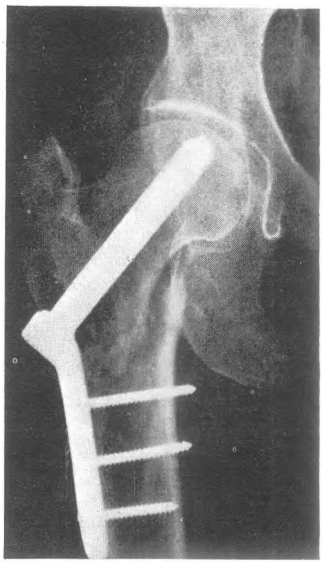

FIG. 400. — Inter-trochanter fracture treated by a pin and plate.

Avulsion of the Lesser Trochanter.—This injury is sometimes caused by sudden contraction of the ilio-psoas muscle, and is common at about puberty. The condition is unlikely to be diagnosed without the assistance of X-rays. Treatment consists in immobilising the limb for 4 weeks in slight flexion.

DISLOCATION OF THE HIP

This injury is relatively uncommon because of the depth of the acetabular cavity and the strong support afforded to the joint by its ligaments and muscles.

The injury usually occurs when the hip is flexed, especially if it is also adducted, because in this position the head of the femur is covered posteriorly by capsule rather than bone. Thus a force applied in the long axis of the femoral shaft may dislocate the head over the posterior lip of the acetabulum. Such a force is commonly provided either by a weight falling on the back of a person in a stooping position, as may happen if a coal-miner is struck by a 'fall of roof', or, more commonly, in car and motor cycle accidents in which, for example, a front seat passenger in a car is thrown forwards so that the knee strikes the dashboard.

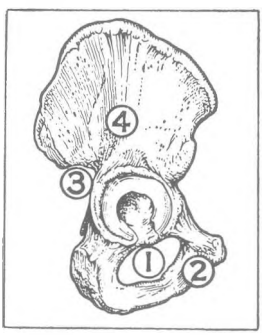

FIG. 401. — The positions taken up by the head of the femur following dislocation of the hip. 1. Obturator. 2. Pubic. 3. Sciatic. 4. Dorsal.

Posterior Dislocation of the Hip

In the usual posterior dislocation, the head of the femur escapes into the sciatic notch (where rarely the sciatic nerve may be damaged), and then passes up on to the dorsum of the ilium (fig. 401). The leg is flexed, adducted, and internally rotated, so that the sole of the foot rests upon the opposite instep. Pain is sometimes referred along the sciatic

nerve, which may be involved by direct injury. X-rays should always be taken to confirm the dislocation and to detect any associated fracture.

Reduction is usually easy provided that the injury is recent, and the anæsthetist obtains full muscle relaxation. The patient is placed on a mattress on the floor, and the iliac crests are steadied by an assistant. The surgeon stands over the limb and flexes the knee and thigh, bringing the head of the bone beneath (i.e. posterior to) the acetabulum. The femur is then adducted and pulled vertically upwards so as to draw the head forward from its posterior position. The essence of the reduction lies in the vertical lifting of the femur. Usually reduction will occur with this vertical traction alone but sometimes the hip must be internally rotated while upward traction is being exerted before the head of the femur suddenly snaps into its socket.

Usually the reduction is quite obvious when it occurs, and if the acetabulum is intact the reduction is stable—especially so when the hip is extended, abducted and internally rotated. Reduction is maintained by skeletal traction through the tibial tubercle for 3 to 6 weeks and during this time the hip is mobilised.

About 10% of dislocations are followed by avascular necrosis of the femoral head (p. 408). This complication cannot be predicted at the time of injury and therefore the patient should not bear weight on the injured limb until avascular necrosis can be excluded, that is to say if possible for 6 months after injury.

Two fractures may be associated with posterior dislocation: fracture of the femoral neck (rarely) and fracture of the posterior lip of the acetabulum. The first fracture must be treated by operation since in no other way can the fracture and the dislocation be reduced. Avascular necrosis is almost invariable. The second fracture is usually reduced by the same manœuvre as that which reduces the dislocation. The reduction however is unstable and its accuracy cannot be guaranteed. Therefore unless the fragment is very small, it is best to treat the injury by open reduction and internal fixation of the fracture with a screw. Damage to the sciatic nerve is a rare but serious complication of this fracture.

Anterior Dislocation of the Hip.—This is rare. The femoral head may lie either in the obturator or pubic position (fig. 401): in both cases the limb is in a position of flexion, abduction, and external rotation.

Central Dislocation of the Hip

This may be caused by a blow on the greater trochanter, driving the head of the femur through the floor of the acetabulum. This injury amounts to a comminuted fracture of the acetabulum and may be accompanied by other fractures of the pelvis.

Treatment is difficult and unsatisfactory. The femoral head can often be pulled out to its anatomical position by traction on the leg (sometimes combined with a side pull applied via a wire or nail in the greater trochanter). Unfortunately the weight-bearing part of the acetabulum rarely reduces with the head of the femur and, even if it does, the floor of the acetabulum does not accompany the reduction. The fracture can be reduced by operation but this is difficult. Therefore probably the best form of management is to accept the displacement and mobilise the hip with tibial skeletal traction as

soon as pain allows. Osteo-arthrosis usually develops eventually, regardless of treatment.

THE PELVIS

Fractures of the pelvis comprise fractures of the iliac crests and of the true pelvis.

Fractures of the Iliac Crests.—These are not of any significance because displacement is slight, as the iliacus muscle on the inner side, and the gluteal muscles on the outer side, support the bone. The patient is confined to bed till symptoms have settled, which usually takes 2 to 3 weeks.

Fractures of the Pelvic Ring

These fractures are due to a blow applied either directly to the pelvis, commonly so as to crush it, or indirectly through the thigh.

The pelvis is a rigid osseo-ligamentous ring. If it is broken in only one place, no significant displacement can occur at the fracture site and the only treatment which is needed is for the patient to rest until the pain has settled. This usually takes about 6 weeks and by this time the fracture has invariably united. If a single fracture in the pelvis runs through the acetabulum, the outcome may be far less satisfactory, for the slightest displacement leads to incongruity in the hip and hence pre-disposes to osteo-arthrosis (p. 233).

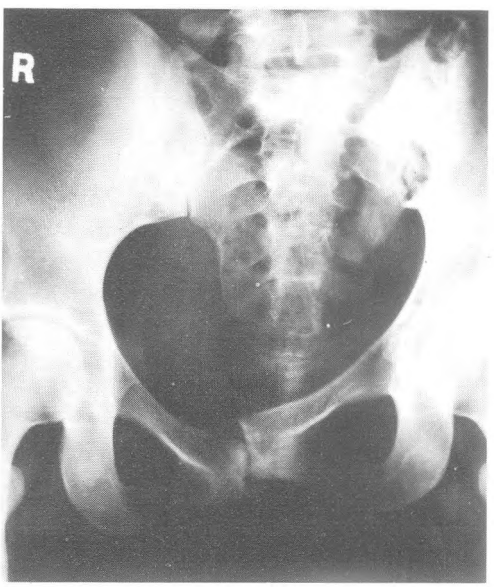

FIG. 402.—Fracture of the pelvis. Since the two halves of the pelvis are displaced relative to each other at the symphysis pubis, there must be a fracture elsewhere in the pubic ring. In this case the fracture, in the left ala of the sacrum, could easily be missed.

Fractures through two places in the ring constitute a far more serious injury since (1) the pelvis is no longer a stable weight-bearing structure, (2) the separated fragment may be displaced and (3) the fracture may be difficult or impossible to reduce. The fractures may traverse the pubic rami on both sides leaving a free central fragment, or they may traverse the rami, the acetabulum or pubic symphysis and the sacro-iliac joint (fig. 402). Dislocation of the sacro-iliac joint almost invariably results in persistent back pain.

The diagnosis of a fractured pelvis is suspected on the history, and the findings of localised tenderness and pain on stressing the pelvis. It is proved radiologically.

Other Injuries.—The most important aspect of fractures of the pelvis is the liability to severe, internal and therefore concealed hæmorrhage, and to injury of the viscera—the male urethra commonly, the bladder in both sexes occasionally, and the rectum and vagina rarely. Systemic signs of blood loss should be sought and blood transfused if necessary. After anterior fractures the patient should be instructed not to attempt to pass urine until an exploratory catheterisation can be made under fully aseptic conditions (Chap. 57). Blood escaping from the external urinary meatus in the male immediately suggests rupture of the urethra (Chap. 59).

A rectal examination is made, and in rare cases a fragment of jagged bone may be encountered, in which case laparotomy and a defunctioning colostomy are necessary. Wounds of the vagina are rare and can usually be sutured.

Having methodically excluded or dealt with visceral lesions, attention can be directed towards the fracture, although if the patient is seriously ill from visceral complications, no special measures are needed for the fracture.

The treatment of unstable, displaced fractures of the pelvic ring is difficult. When separation of the symphysis occurs, the pelvis can be compressed by suspending it in a pelvic sling from an overhead beam. In some cases, nursing the patient between sandbags or on his side will close a separated symphysis without any external apparatus. Skeletal traction through the tibia may be necessary if a free lateral fragment is proximally displaced or the pelvis may be immobilized by skeletal transfixion pins passed into each fragment and connected to each other with an external fixation frame. If the absence of displacement makes such measures unnecessary, the patient is treated by bed rest, maintaining as full a range of movement as possible at the hips and spine, for at least 8 weeks.

The Coccyx

The coccyx may be fractured as a result of kicks or falls. Pain, which is often severe, occurs on walking, sitting or with actions which involve contraction of the levator ani, such as defæcation or coughing. Rectal examination reveals local tenderness and occasionally deformity. No specific treatment is needed and the symptoms settle spontaneously in about 6 weeks.

If symptoms persist for months, in spite of adequate rest and symptomatic treatment by sitting on soft cushions or anæsthetic injections, excision of the coccyx may have to be considered. There should be no haste in making this decision as persistent symptoms are often psychological in origin. In some cases these symptoms occur without fracture or even injury. This condition, known as *coccydynia*, is of unknown ætiology. The treatment of the pain is the same as that for pain occurring after fracture.

CHAPTER 26

THE HEAD (SCALP, SKULL AND BRAIN)

THE SCALP

THE **scalp** consists of four layers—skin, subcutaneous tissue, epicranial aponeurosis or galea, into which is inserted the occipito-frontalis muscle, and the subaponeurotic areolar layer. The scalp is well supplied with blood-vessels, and the walls of the arteries are adherent to the fibrous tissue in the subcutaneous layer. Therefore scalp wounds bleed freely, as the muscular coat of a divided artery cannot retract readily when the vessel is severed.

Wounds of the scalp.—The great vascularity of the scalp indicates that wounds heal well, and areas which at first sight seem necrotic may in due course prove to be viable and heal. If scalp is lost due to trauma or surgery, repair of a relatively small resultant defect requires extensive flaps or free grafts. For these reasons any excision or 'debridement' of the edges of scalp wounds should be reduced to the minimum and only portions grossly contaminated or necrotic should be removed (Chaps 1 and 2). While all coverings of the brain are important (e.g. dura and skull), the scalp is the one layer which is essential, and must, therefore, be preserved.

Scalp wounds should be managed by shaving the hair over an area at least 3 cm clear of the wound, which is then cleaned, avoiding soapy solutions if there is any suspicion of an underlying fracture and dural tear. Bleeding from the wound edges is best controlled by simple pressure, and it is rarely necessary to apply artery forceps to the galea. It is difficult and potentially dangerous to probe wounds to determine the presence of depressed fractures and foreign bodies, which can only be detected with certainty by radiographs. Therefore, after shaving, cleansing and application of a temporary dry dressing, radiographs should be done (see p. 429 for indication for X-ray of skull) before wounds are closed in two layers, galea to galea and skin to skin, with interrupted black silk sutures. When the wound edges and layers are much disorganised it is reasonable to close the wound loosely in one layer, avoiding any tension; in these circumstances extending the incision in an S-shaped manner may ease the closure (fig. 403).

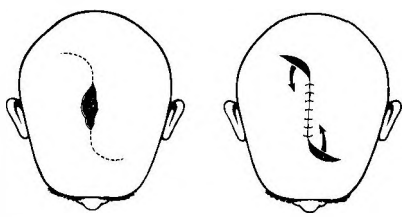

FIG. 403.—Letter S scalp plastic incision.

Hæmatoma of the scalp may be subcutaneous. Subaponeurotic hæmatomas spread extensively beneath the epicranial aponeurosis and are only limited by the attachment of the aponeurosis around the base of the calvarium. Subpericranial hæmatomas are limited by the suture lines which border the underlying bone, to which the pericranium is attached.

Infections of the scalp occur most commonly in association with wounds, particularly when lacerations are not dealt with for several days. However, the vascularity of the skin tends to promote healing, and infection usually remains localised. Rarely infection may spread widely in the loose avascular areolar tissue beneath the galea. Post-traumatic scalp infection requires identification of the organism and appropriate antibiotics after wound toilet; occasionally extensive subgaleal pus will require drainage through multiple incisions. When there is much soiling of scalp wounds, tetanus and occasionally gas gangrene are potential dangers.

Subperiosteal infection, with or without osteomyelitis, is rare and is usually the result of chronic frontal sinusitis. The associated external swelling due to subperiosteal pus and scalp œdema is termed Pott's puffy tumour (p. 447). The condition has serious implications because of the association with intracranial abscess, either extradural or subdural, and therefore any patient with signs of subperiosteal infection should be referred for further investigations.

LUMPS ON THE HEAD

General principles.—Accurate clinical diagnosis, with radiography of the skull when indicated, is an essential prerequisite before any attempt at surgical removal or biopsy. In general lumps in the midline should be approached with particular caution because they are likely to be in continuity with intracranial structures such as the meninges and sagittal venous sinus; radiography is obligatory, and surgery should be undertaken only by those able to deal with associated intracranial pathology; any lump removed must be examined histologically. The usual principles of examination of any lump are relevant and particular features include:—

(*a*) *Hardness and fixation to the skull.* This indicates that the lump is arising from or involving bone. Primary intracranial pathology includes meningioma (especially parasagittal), and meningeal sarcoma. Lesions arising from the skull include benign osteoma (exostosis), metastases, osteogenic sarcoma (sometimes arising in Paget's disease, p. 300); chronic infection with osteomyelitis may mimic a bony tumour.

(*b*) *Lumps which are firm* include sebaceous cysts, and dermoid cysts. Soft fluctuant lumps include meningocoeles (especially in the midline) which may be translucent and have a cough impulse.

Sebaceous cysts (*syn.* wens) (p. 114) are often multiple (fig. 404). The surrounding scalp becomes bald if pressure interferes with its blood supply. Infection and ulceration are described on p. 114.

Dermoid cysts are most frequent over the external angular process (fig. 508). X-ray shows bone defect with a sclerotic margin (fig. 405). Although congenital they may not appear until the patient is some years old or in adult life. Communication with an intracranial dermoid may take place by a narrow neck which passes through the inner table and which is visible on X-ray. An impulse on coughing is only detected if the communication is wide.

Treatment.—Superficial cysts are excised locally. Extensions are approached by osteoplastic craniotomy. The intracranial portion may be larger than the superficial.

Squamous and Basal-celled carcinomas (p. 59), which begin as a small ulcer which bleeds repeatedly when the hair is combed, are easily overlooked in their early

stages. Over the course of months or even years such a tumour may spread widely in the scalp and involve all layers and erode the skull. Treatment is by early biopsy and excision, and extensive rotation flaps may be needed to close the resulting scalp defect.

Papillomas are common and cause discomfort on combing the hair. Constant irritation of this nature encourages malignant changes.

Lipomas arise from fatty tissue incorporated with the areolar layer (fig. 53).

Melanomas may be unrecognised, owing to the small growth being hidden by hair. If malignant changes supervene, the appearance of secondary deposits should lead to the discovery of the tumour.

Fibrosarcoma has already been described (p. 60).

Cirsoid aneurysm is rare. Capillary nævi sometimes occur in the skin, beneath which abnormal arteries communicate directly with distended veins. The superficial temporal artery and branches are most commonly affected. The underlying bone becomes thinned by pressure which also causes the hair to fall out. Radiography may show perforations in the skull which indicate that part of the tumour is intracranial. These tumours tend to enlarge slowly with a risk of serious hæmorrhage if ulceration occurs.

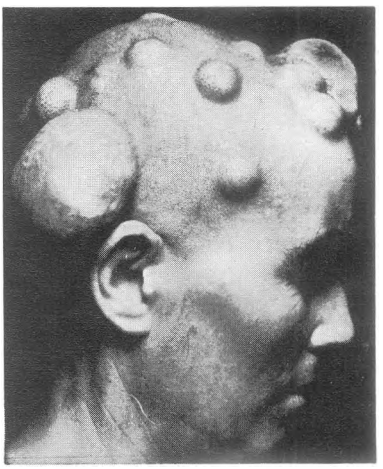

FIG. 404.—Sebaceous cysts of the scalp.

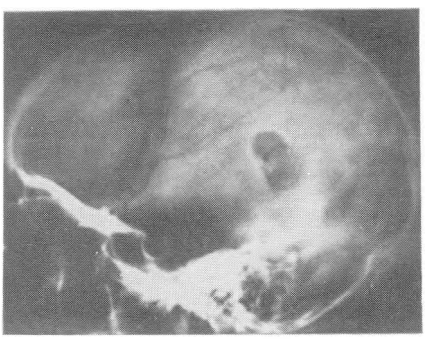

FIG. 405.—Lateral X-ray showing dermoid cyst of the diploe. The inner table has been penetrated by intracranial extension.

Treatment consists in extirpation of the tumour in the early stages (ligation of one or both external carotid arteries is advisable as a preliminary step to local excision). If its size forbids such radical treatment, then the main vessels are ligated, but this is often unsuccessful, as a large tumour on the surface usually possesses intracranial connections which maintain the blood supply.

THE SKULL

Microcephaly may be associated with agenesis of the brain and imbecility, or result later from premature synostosis in a normal child.

Oxycephaly (*syn.* steeple-head, Gk. *oxus* = sharp) is a condition in which the skull is egg-shaped, following premature obliteration of sutures. Most cases develop increased intracranial pressure.

Treatment.—*Cranioplasty* by Gigli saw cuts between burr holes and wrapping the bone edge in tantalum foil allows normal skull expansion and cerebral development to proceed.

Meningocele.—*Clinical Features.*—Protrusions of a pouch of dura mater through a congenital defect in the skull can occur at the root of the nose or over the occipital bone. Trans-sphenoidal projections protrude through the base of the skull into the naso-pharynx and have been mistaken for nasal polypi: attempted removal has resulted in meningitis. A meningocele

Leonardo Gigli, 1863–1908. Obstetrician. Director, Santa Maria Nuova Hospital, Florence, Italy. Introduced his saw for pubiotomy in 1894, and adapted it for craniotomy in 1898.

is present at birth, and forms a tense rounded swelling which is translucent and sometimes pedunculated, and which yields an impulse when the child cries or coughs. Growth of the skull may occlude the neck of a small sac; in this case a cyst remains which is non-pulsatile and unaffected by coughing.

FIG. 406.—An encephalocele. A myelocele is also present.

An encephalocele is a similar condition, but some portion of the brain is also extruded (fig. 406). Should this cerebral extrusion contain part of a ventricle, it is known as a *hydroencephalocele*. In these conditions vascular pulsation is present. The child may be stillborn, or some degree of idiocy or deformity will be associated.

Treatment.—The skin surface should be protected with padding and tulle gras to prevent ulceration and infection. If at the age of a month the child shows normal development, operation is performed under local anæsthetic the child being tied to a cross and given a feeding bottle during operation. A curved incision is fashioned in one margin of the sac, so that when the wound is sutured the incision will not overlie the bone defect. The neck of the sac is defined, transfixed, and ligated and the sac and excess skin removed. Muscle and fascia are brought together over the bone defect. Many cases have a small encephalocele at the base. This is removed with the sac, without harmful effect, as the tissue is functionless.

Skull Fractures.—See Cranio-cerebral Injuries (p. 417).

Infections of the skull.—Osteomyelitis of the skull occurs either as a result of chronic frontal or ethmoid sinusitis, mastoid infection or following inadequate treatment of a compound depressed skull fracture. Chronic sinus or mastoid infection may be associated with intracranial abscess (p. 447), irrespective of osteomyelitis, but the presence of osteomyelitis increases the risk of intracranial abscess, probably through emissary veins. Patients with osteomyelitis in association with ENT sepsis should be closely observed and investigated for intracranial infection.

Tuberculous disease of the skull is uncommon, but occasionally occurs in association with tuberculous lesions elsewhere. As with other bones, the infection commences either in the pericranium or in the medulla, i.e. the diploe. The diseased bone should be removed widely, otherwise abscesses are likely to form and erode the scalp. Antituberculous drugs are given (p. 37).

Syphilitic pericranitis is nowadays a rare affection. Localised swellings occur which are slightly tender and fixed to the bone. Under suitable treatment disappearance is usual, although a small bony swelling occasionally remains.

NEW-GROWTH OF THE SKULL

Innocent tumours are rare. An *ivory* osteoma occasionally arises in the region of an air sinus (p. 255).

Malignant new-growths resemble those of other bones. *Pericranial sarcoma* forms a tumour, the consistency of which depends upon its vascu-

larity and rate of growth. Thus it may be pulsatile or of an almost bony hardness. *Osteoclastoma* occasionally develops in the diploe.

The commonest malignant tumour is secondary carcinoma, usually derived from primaries in the breast, thyroid, prostate, or suprarenal.

Hypernephromas produce rapidly growing vascular tumours, which pulsate when the outer table is eroded. Secondary deposits usually produce a single clear area on the skull X-ray with irregular margin (fig. 407). Deposits from carcinoma of the breast are often multiple.

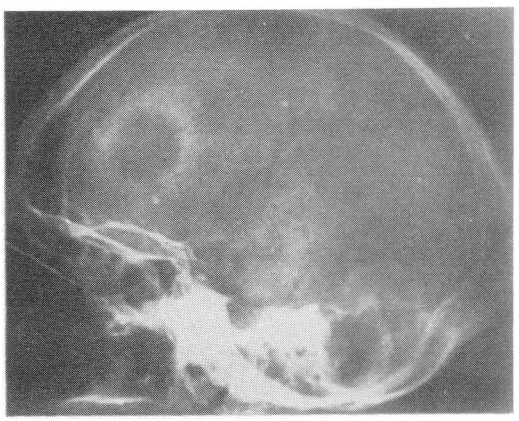

FIG. 407.—Lateral X-ray showing irregular clear area produced by a secondary deposit from carcinoma of the breast.

CRANIO-CEREBRAL INJURIES

Introduction.—In most countries the primary and continuing care of patients who have sustained a head injury is the responsibility of general and orthopædic surgeons rather than neurosurgeons. Despite the expansion of neurosurgical services this situation is likely to continue and therefore the general surgeon requires a knowledge of principles and practice in this field. Since the majority of patients do not require any neurosurgery, the major steps in the care of such patients are medical, diagnostic and nursing and it is these steps which will determine the outcome far more frequently than any surgical manœuvres. Indeed the avoidance of unnecessary cranial surgery is an important principle, because such surgery may be harmful. At the same time the general surgeon must be aware of the indications for specialised intracranial investigation which usually requires transferring the patient to a neurosurgical department. Finally he must be able to decide when there is a clear indication for urgent surgery without specialised investigation, either because the rate of deterioration does not allow any further delay, or because the facilities for intracranial investigation do not exist.

INJURIES OF THE BRAIN

Mechanism and Pathology.—At the moment of impact a diffuse neuronal lesion is inflicted on the brain which is responsible for the immediate clinical picture of brain injury. Secondary changes of œdema or intracranial hæmorrhage take time to develop. The rise in pressure resulting from these causes leads to a deterioration in the patient's level of consciousness a few hours after injury; the clinical picture in the early stages results from the neuronal lesion alone. All degrees of brain injury resulting in loss

of consciousness, concussion, contusion, or laceration of the brain are produced by one mechanism, namely displacement and distortion of the cerebral tissues occurring at the moment of impact.

The main factors which determine the severity of cerebral injury are:—

1. *Distortion of the brain.*—The brain has ' mobility ' in several respects. In life it is a soft structure which is readily distorted and therefore one part of the brain may move in relation to another. The mobility due to the consistency of cerebral tissue is accentuated by the presence of the fluid (CSF) and vascular compartments. This creates ' shearing ' forces which will produce widespread damage to neurones, nerve fibres, supporting tissues (glia) and blood vessels. These effects may be diffuse and therefore far removed from the site of the blow to the head (figs. 408, 409). The clinical effect of

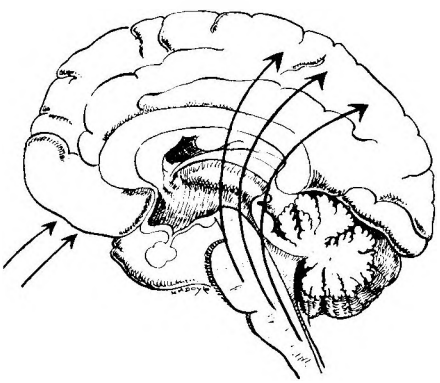

FIG. 408.—Lines of force acting on the hypothalamus and brain stem as the result of posterior displacement of the hemisphere.

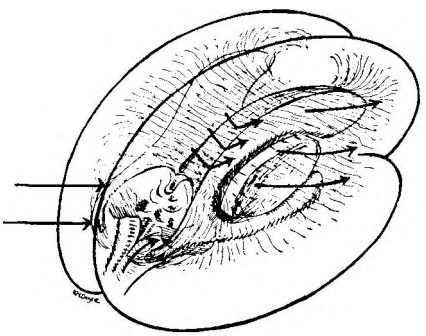

FIG. 409.—Lines of force acting on the corpus callosum and one peduncle as the result of anterior displacement of one hemisphere.

this distortion may be the initial loss of consciousness, and, if the deterioration produces significant neuronal damage, persisting loss of consciousness and/or focal neurological deficit. Changes in level of consciousness are due to distortion and neuronal damage in the lower central structures (e.g. mid brain) which may be momentarily distorted in a vertical or lateral direction. Such distortion may produce transitory damage or persisting damage in the brain stem reticular formation, the groups of cells which are responsible for normal consciousness probably through the activation of cortical mechanisms. This explains the persisting loss of consciousness in patients who have little external evidence of trauma over the skull vault.

2. *Mobility of the brain in relation to the skull and membranes.*—The dura mater is more or less firmly attached to the skull, and merges into the falx and tentorium. Between the brain and the dura is a space which varies in depth, and consists of the true subdural space and deep to that the subarachnoid space containing the CSF. The brain is not, therefore, held firmly by the skull, falx and tentorium and considerable movement between the brain and the dura may occur following ' acceleration ' or ' deceleration ' injuries, which

increase the damage to the brain. With age the brain shrinks, and the mobility increases. A secondary effect of this movement is rupture of veins which cross the subdural space and which drain blood from the cerebral hemispheres into the dural venous sinuses, especially the superior sagittal sinus (fig. 410).

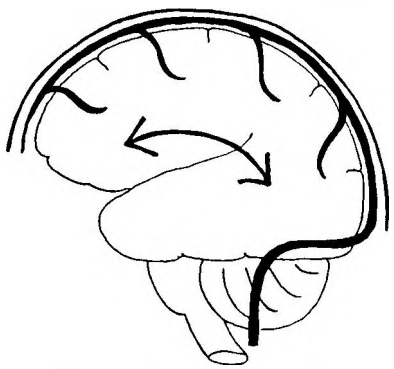

FIG 410.—Suspension of the brain in the slings formed by the superior cerebral veins.

3. *The configuration of the interior of the skull.*—The degree of brain damage due to distortion and movement of the brain will be affected by the internal configuration of the skull, falx and tentorium. Where the skull is 'smooth' (convexity) brain damage is usually less severe than where it is 'rough', 'sharp' or the cavity relatively confined. Thus the temporal lobe will be damaged by the sharp sphenoid ridge and confined middle cranial fossa; the frontal pole by the rough anterior cranial fossa floor and the confined anterior corner of the anterior fossa; the occipital pole by the meeting of falx, tentorium and convexity; the corpus callosum by the 'sharp' edge of the falx; the cerebral peduncles by the 'sharp' edge of the tentorium (figs. 411, 412).

4. *Deceleration and acceleration.*—The majority of severe cranio-cerebral injuries due to traffic accidents are the result of rapid deceleration when the moving head strikes an immovable object (e.g. the road). This produces the features of distortion aggravated by the brain's mobility (see above). The converse is the 'acceleration' injury when the stationary skull is struck by a moving object (e.g. assault). The skull will rapidly accelerate and there-

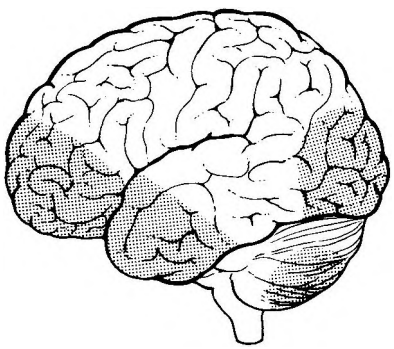

FIG. 411.—Sites of polar damage ('hatched' areas).

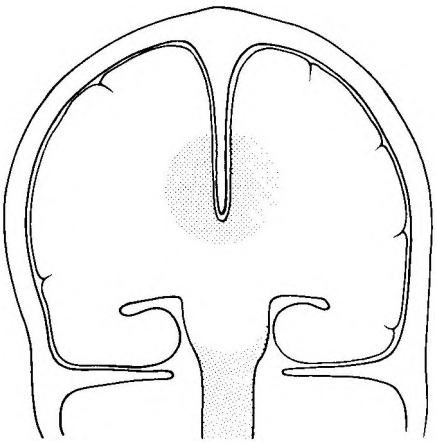

FIG. 412.—Sites of falxine and tentorial damage.

fore distort the stationary brain. The complexity and extent of the brain damage may be increased if there is loss of consciousness, the subject falls to the ground and the brain suffers a severe ' deceleration ' injury.

5. *The pre-existing state of the brain.*—The ageing brain has fewer reserves than the younger brain and therefore the lasting effects of an injury are likely to be greater in the elderly.

CEREBRAL CONCUSSION, CONTUSION AND LACERATION

The older clinico-pathological terms which were used to describe brain injury, concussion, contusion, and laceration of the brain merely indicate minor and major degrees of injury and do not imply any individual difference in mechanism.

In cerebral concussion the stretch is slight. There is a brief temporary physiological paralysis of function without organic structural damage, which results in a transient loss of consciousness, followed by complete recovery.

Cerebral contusion indicates a more severe degree of damage with pathological changes which might be diffuse or relatively localised. Changes include bruising and swelling of cortical gyri, localised or generalised œdema, shearing damage to nerve cells and axons either superficially or more deeply between grey matter and white matter, and hæmorrhage due to tearing of small blood vessels within the brain substance. In patients who have survived for long periods after such injuries, autopsy may ultimately reveal diffuse degenerative changes in grey and white matter.

In cerebral laceration the internal changes are the same as those seen in contusion, but the brain surface is torn, with effusion of blood into the cerebrospinal fluid owing to laceration against bony ridges and the edges of dural septa during brain displacement.

THE ACUTE COMPRESSING INTRACRANIAL HÆMATOMAS

These are 1. Acute Extradural Hæmatoma, 2. Acute Subdural Hæmatoma and 3. Acute Intracerebral Hæmatoma.

Acute Extradural Hæmatoma.—Extradural hæmorrhage may arise from branches of the *internal maxillary* or *anterior meningeal* vessels torn by fractures of the anterior fossa. In such cases a deterioration in level of conciousness occurs in association with an antero-basal fracture, but usually there are no accompanying localising signs.

The majority of cases of middle meningeal hæmorrhage are due to an injury associated with fracture of the temporal bone, bleeding from this fracture line causes an extracranial extravasation producing boggy swelling deep to the temporal muscle, therefore all cases of even minor concussion must be under observation for 24 hours if there is any bruising in the temporal fossa. Focal compression produced by a clot is usually accompanied by the appearance of new localising signs produced by the local pressure.

The classical syndrome of extradural hæmorrhage results from injuries of the anterior or posterior branches of the *middle meningeal artery*. In such cases the injuring force, which comes from a lateral direction, is often rela-

tively trivial, such as a blow from a golf or cricket ball which strikes the thin bone of the temporal plate, inflicting a fracture which drives the dura inwards and in the case of anterior branch bleeding, snaps the meningeal artery at a point where it leaves the bony canal in the pterion and crosses to gain attachment to the dura mater. In the elastic skull of children the injury may occur without fracture, but in all adults, comprising 90 per cent. of cases, X-ray reveals a fracture line crossing the middle meningeal groove.

The detection of this fracture is an absolute indication for the admission of the patient for observation.

From the torn vessel blood passes in three directions:

(1) Outwards through the fracture to form a boggy swelling under the temporal muscle, the finding of which is an additional indication for the *admission for observation of a conscious patient.*

(2) Downwards into the middle fossa.

(3) Upwards over the motor cortex.

Clinical Features.—Usually a laterally directed blow of small magnitude causes a short initial period of concussion, followed by a characteristic **lucid interval**, during which the hæmorrhage is collecting intracranially and also forming a swelling under the temporal muscle.

An unconscious footballer was carried off the field but later recovered sufficiently to finish the match; headache and drowsiness supervened so that he retired to bed early and was later found dead from middle meningeal hæmorrhage. Similar tragedies have occurred at school from cricket or on the links from golf balls.

When syndromes of hæmatoma formation develop in patients already deeply unconscious as the result of cerebral contusion a lucid period is absent and the collection of the hæmorrhage can only be recognised by determining the alteration in the level of consciousness and the appearance of the new localising signs.

The next change is in the level of consciousness. The patient becomes confused and irritable, and at this stage is in danger of being arrested if found wandering and smelling of alcohol; therefore, beware of the patient brought to Casualty in a confused state with a bruise in the temporal region. Persons alleged to be found drunk have been locked in cells only to be found dead in the morning, the unconsciousness and stertorous breathing of compression being mistaken for a drunken stupor. Confusion changes to drowsiness, and at this stage pressure of the clot on the motor cortex produces cerebral anoxia, causing twitching, followed by paralysis of the face, then the arm, then the leg, on the opposite side of the body as the clot spreads upwards over the motor cortex. At the same time inward displacement of the temporal lobe causes the inner portion of the lobe to press against the third nerve above the edge of the tentorium (fig. 413(B)), causing restriction, rapidly followed by dilatation of the pupil on the side of the hæmorrhage. If the pressure is not relieved, displacement of the brain stem at the tentorial opening forces the opposite crus against the rim of the tentorium, producing

further hemiplegia, which this time occurs on the side of the hæmorrhage (fig. 413(A)).

The development of the extradural compressing mass depends upon the ease with which the dura can be stripped away from the inner surface of the skull. With advancing age the dura becomes more adherent to the skull and, with the 'elasticity' of the skull, is one factor in the greater incidence of extradural hæmatoma in children and young adults. During the 'lucid' interval the hæmatoma gradually enlarges and it is not until it has reached a size sufficient to cause a severe rise in intracranial pressure and critical distortion of the mid brain at the tentorial hiatus, that the conscious level deteriorates, often rapidly (fig. 413).

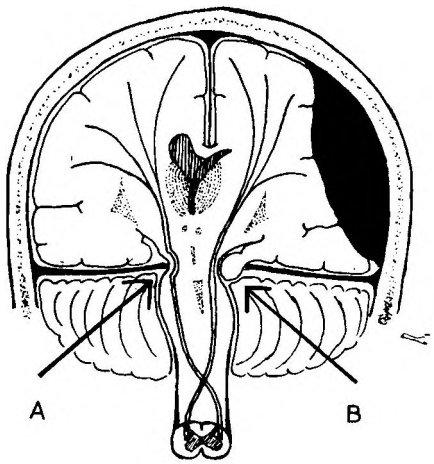

FIG. 413.—(A) Impaction of the opposite crus against the opposite rim of the tentorial opening. (B) Displacement of the inner edge of the temporal lobe (uncus) descending into the tentorial opening (mid-brain cone). *(G. B. Northcroft, F.R.C.S., London.)*

Finally, impaction of a mid-brain cone produces decerebrate rigidity and fixed dilatation of *both* pupils, at which stage the case is probably too late to remedy by operation.

Posterior branch bleeding is less than one fifth as common. Here the clot is situated at a distance from the motor cortex, the surface bruising is situated farther back, or there may be a steady arterial hæmorrhage from the ear when the fracture line involves the middle ear. The level of consciousness deteriorates, and the patient passes into a stage of cerebral irritation or stupor with a dilated pupil from pressure inwards on the third nerve, but there may be no motor paralysis for several days, or the paralysis appears on the same side of the body as the hæmorrhage as the result of brain shift and impaction of the contralateral crus. The site of operation is determined by the site of the fracture, surface bruising, and dilatation of the pupil.

Extradural hæmorrhage of venous origin is produced by fractures which injure the major sinuses. Injury of the superior longitudinal sinus produces a massive subgaleal hæmatoma, together with evidence of clot compression, causing deterioration of consciousness with generalised epilepsy if the clot lies over the frontal areas, or unilateral or bilateral leg weakness with extensor plantar responses if the clot is over the upper end of one or both motor cortices.

Acute Subdural Hæmatoma.—Following severe head injury there is commonly a thin layer of clot over the brain in the subdural space. This does not usually constitute a significant compressing lesion which is more likely to be produced by the underlying cerebral œdema and contusion. A significant subdural hæmatoma is produced either by rupture of a large cortical vein as it crosses the potential subdural space to reach the fixed dural venous sinuses (fig. 414) or by laceration of the cortex and subsequent venous or arterial hæmorrhage.

The *presentation* and *management* of acute subdural hæmatoma may be similar to that of acute *extradural* hæmatoma, but tend to differ in certain respects.

1. There is often a severe primary brain damage (e.g. laceration) and therefore initial and persisting loss of consciousness. A truly lucid interval is unusual.

2. Deterioration tends to occur sooner than in extradural hæmatoma.

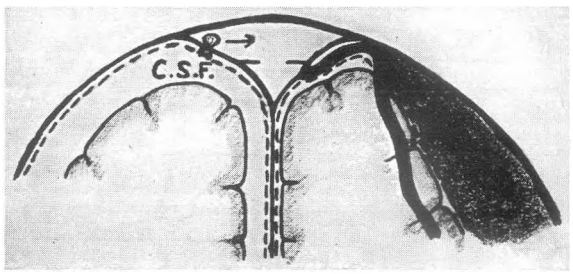

FIG. 414.—Position of a subdural hæmatoma produced by tearing of the superior cerebral veins at the level of the arachnoid. The diagram also shows the partial or occasionally complete septum which divides the superior sinus. Cerebrospinal fluid drains into the upper compartment through the arachnoid granulations. The superior cerebral veins drain into the lower. (*G. B. Northcroft, F.R.C.S., London.*)

3. The hæmatoma may be ' coup ' or ' contre-coup ' (p. 425).

4. The hæmatoma is often extensive and may cover an entire hemisphere extending both subfrontally and subtemporally.

5. Effective surgical action is unlikely without special investigation (CT scan or arteriogram, p. 435).

6. Effective surgery involves an extensive craniotomy; even then the results are poor due to the primary and often widespread brain damage and brain œdema.

When the presentation is less dramatic and delayed by several days (so-called sub-acute subdural hæmatoma) it is very difficult to determine the part played by the hæmatoma as opposed to the underlying brain damage and especially increasing brain œdema. Without the CT scan surgical opinions are frequently without foundation, and surgery ineffective.

7. Very rarely a large acute subdural hæmatoma may occur in the elderly following a trivial injury or shaking of the brain with rupture of a ' bridging ' vein.

Acute Intracerebral Hæmatoma.—This is the least common of the significant and remediable compressing intracranial hæmatomas. It is rare for a major intracerebral vessel to rupture as a result of trauma and produce an expanding hæmatoma which is the sole cause of cerebral compression. When there is a totally lucid interval before such an event it is more likely that hæmorrhage has occurred from a pre-existing vascular lesion such as an aneurysm or arteriovenous malformation. If time permits arteriography before surgery is advisable.

Usually traumatic intracerebral hæmatomas are associated with cerebral laceration, contusion, oedema and necrosis, the compressing lesion being formed by all these elements. Therefore in these circumstances the benefit of removing the clot is unpredictable and often disappointing. Occasionally the removal of such traumatic masses in the anterior part of the temporal lobe or in the frontal pole (fig. 411) may be effective, but pre-operative diagnosis with CT scan or arteriogram is essential.

Early Secondary Pathological Changes. A. Intracranial Factors

Brain œdema.—The brain often reacts to any insult, be it ischæmic, neoplastic inflammatory or traumatic, by swelling due to *œdema*. The accumulation of fluid is both extracellular and intracellular. Macroscopically

œdema may be localised, at least initially to the part of the brain most damaged, but it may extend rapidly throughout one or even both cerebral hemispheres, causing a severe rise in intracranial pressure and, at an earlier stage, features of a relatively localised space occupying or compressing lesion which may mimic a compressing hæmatoma.

Brain necrosis.—As a result of the initial localised damage, the consequent œdema, rise in intracranial pressure, hæmorrhage within the damaged brain tissue and ischæmia, parts of the cerebral hemispheres may become necrotic and present surgically as soft almost liquid masses. These changes are irreversible and occur particularly in the temporal and frontal lobes, and occasionally surgical evacuation may lower the intracranial pressure sufficiently for survival.

Hæmatoma.—The surgically remediable intracranial hæmatomas may be extradural (p. 420), subdural or intracerebral (p. 422). They may be the result of arterial or venous bleeding, and therefore vary in their time of presentation in relation to the injury. The deeper the hæmatoma, the more likely it is to be associated with primary brain damage, œdema and necrosis and therefore the results of surgical evacuation are poorer. It is rare to have an intracerebral hæmatoma due solely to the rupture of one major vessel; usually such hæmatomas are due to the tearing of many small vessels, and it is often difficult to determine the relative part played by the hæmatoma in the clinical problem.

Vascular changes.—Consequent to rising intracranial pressure disturbance in cerebral blood flow may occur which cause further brain damage through ischæmia, and the ischæmia itself may produce an increase in brain œdema. In normal life there are auto-regulatory mechanisms which, to some extent, protect the cerebral circulation despite changes in blood pressure and intracranial pressure. When the intracranial pressure rises severely, the auto-regulatory mechanism fails and irreversible ischæmic changes occur. In addition following brain injury there may be dilatation and engorgement of the venous system which will further increase the mass of intracranial content, and therefore the intracranial pressure, increase the brain œdema by impeding the normal drainage of tissue fluid and worsen the cellular ischæmia.

Coning, or herniation of the contents of the supratentorial compartment through the tentorial hiatus, or the contents of the infratentorial compartment through the foramen magnum, may accompany a rise in pressure in the appropriate compartment (fig. 413). The pathological sequelæ of the tentorial hiatus include herniation of the medial part of the temporal lobe (the uncus) on the side of the supratentorial mass which causes pressure upon the ipsilateral third cranial nerve, and upon the mid brain. The mid brain is distorted and displaced away from the side of the mass, where the free edge of the tentorium indents or ' notches ' the cerebral peduncle, and interferes with the descending motor pathways from the hemisphere opposite to that which is being compressed. In addition the compression of the mid brain contributes to obstruction of the CSF flow in the aqueduct, which contributes further to the rise of intracranial pressure. These changes produce a

deterioration in the level of consciousness, dilation of the pupil on the side of the compressing mass, and a hemiparesis on the *same* side as the mass. This situation is called ' Kernohan's Notch '.

Coup and Contre-Coup.—These words are used to indicate the types of cranio-cerebral damage which may occur either on the side of the blow to the head (*coup*), or opposite (and often diagonally opposite) to the position of the blow (*contre-coup*). Provided it is clear where, and where alone, the head was struck, this knowledge can provide a useful guide to pathology when used in conjunction with the lateralising signs of a compressing lesion (see Coning, p. 424).

COUP AND CONTRE-COUP INJURIES

Coup	*Contre-coup*
Scalp Laceration	—
Skull Fracture	—
Extradural Hæmatoma	—
Subdural Hæmatoma	Subdural Hæmatoma
Cerebral Laceration and Contusion	Cerebral Laceration and Contusion
Brain Oedema	Brain Oedema
Intracerebral Hæmatoma	Intracerebral Hæmatoma

Early Secondary Pathological Changes. B. Extracranial Factors

Respiration.—Respiratory failure or inadequacy is one of the most potent factors which can aggravate or precipitate the development of severe brain œdema and venous congestion, leading rapidly to irreversible changes. Of the two factors, the arterial pO_2 and pCO_2, a rise in pCO_2 is the more potent. It is therefore essential that pCO_2 and pO_2 are maintained at *normal* levels in any patient who has sustained a severe head injury. Although at times hyperventilation has been employed in order to reduce intracranial pressure, abnormally low pCO_2 levels may cause a reduction in cerebral blood flow and subsequent cerebral ischæmic damage, and it is therefore safer to achieve normal levels.

Systemic blood pressure and blood volume.—When the brain is at risk from ischæmic damage following injury it is obviously essential that a normal cardiac output is maintained and that blood loss from other injuries is adequately replaced. Recent work has confirmed that, when the intracranial pressure is high, protective cerebral blood flow regulatory mechanisms are impaired and a fall in cardiac output is particularly likely to result in irreversible cerebral ischæmic damage.

Fluids.—Although the onset of brain œdema is usually the result of the primary brain injury and ischæmia, the state of the vascular compartment will influence its course. Thus if hypotonic fluids are given intravenously, the plasma osmolality may be lowered, particularly if there is any delay or defect in renal excretion of the excess ' water ', and therefore by an osmotic effect fluid may be drawn into the brain tissue and brain swelling increased. With disturbance of the normal blood-brain barrier (which is poorly under-

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stood) these effects may be increased. Therefore intravenous fluids should be isotonic, and the volumes used (apart from replacement of blood loss by blood) should be dictated by normal requirements and renal function. Problems with fluid and electrolyte balance may be exacerbated by inappropriate secretion of pituitary anti-diuretic hormone as a cerebral response to the cerebral injury. This may produce dangerously low levels of plasma sodium.

Temperature.—When the body temperature rises the metabolic demands of all tissues rise and the accumulation of catabolites increases. When cells or particular groups of cells are damaged, increased metabolic demands will aggravate the cellular failure or aggravate the manifestations of poor function. This is well seen following severe head injury when a rise in body temperature will cause a further deterioration in neurological state.

Management

Primary and later neurosurgical management must include the following, which will be considered in clinical sequence:

1. Indications for admission
2. Casualty reception
3. Radiography
4. Continuing care and observations
5. Deterioration
6. Indications for surgery without special investigation (decision making)
7. Special investigations
8. Non-surgical therapy: osmotic diuretics, steroids
9. Miscellaneous early complications

1. Indications for Admission.—Following head injury patients are admitted to hospital for two main reasons: (*a*) For continuing hospital care, for those who have suffered serious injury and who are in obvious need of care and possibly surgery. (*b*) For observation in case a complication should develop, despite the patient being perfectly well at the time of admission. The first category presents little difficulty about the indication for admission, but the second presents considerable difficulty. Remediable complications (e.g. acute extradural hæmatoma) may occur following very trivial injury, but there is some evidence that they are more likely to occur after transient change in consciousness, when a skull fracture is present and when the patient complains of persisting headache. However, although the incidence of hæmatoma is very low in the absence of these features it is currently a matter of debate whether transient clouding of consciousness alone should remain an indication for admission. If the traditional view is maintained, the indications for admission for *patients who are neurologically well* can be listed as: (i) Transient loss of consciousness. (ii) Absence of any reliable witnesses. (iii) Skull fracture. (iv) Injury sustained at speed. (v) Alcohol intoxication (see p. 429). (vi) Criminal assault.

It is also debatable whether a skull radiograph should be used in deciding upon admission because a significant number of acute extradural hæmatomas

occur in the absence of a fracture. It is therefore safer to decide about admission before any evidence from a skull radiograph is available.

The duration of observation is arbitrary, but since it is exceptional for acute extradural hæmatoma to present more than 18 hours after injury, overnight admission is sufficient.

2. Casualty Reception.—The reception of the severely injured patient is a large subject which has been dealt with elsewhere (p. 6). In the following paragraphs the essential points relate to patients whose main reason for admission is a major head injury. There are certain *general principles*:

(*a*) Patients are more likely to die from airway obstruction than from any remediable intracranial lesions.

(*b*) Surgically remediable intra-thoracic and intra-abdominal lesions take precedence over any intracranial procedures.

(*c*) In normal civilian practice patients are brought rapidly to a casualty department; therefore compressing intracranial hæmatomas are unlikely to present when the patient is first seen.

(*d*) The initial clinical assessment and its recording especially of the level of consciousness is of crucial importance if any later deterioration is to be gauged accurately.

(*e*) The immediate institution of the correct care of the unconscious patient does more to lower the morbidity and mortality from major head injury than any other single measure.

In receiving and assessing a patient in casualty the essential steps in chronological order can be summarised as follows:

(i) *Protection of the airway* (see œdema).—All the usual measures for the protection of the airway in an unconscious patient should be taken immediately; removal of false teeth, positioning prone or on one side with the head low, mouth suction and insertion of a pharyngeal airway. The presence of a compound skull fracture with exuding brain tissue is not a contraindication to lowering the head. In the majority of patients these measures will suffice. However if they do not a cuffed endotracheal tube should be inserted, which is in itself a test of the need for that manœuvre.

(ii) *General assessment.*—The general assessment is often more important than the neurological because intra-thoracic and intra-abdominal injury may require immediate action. These aspects are dealt with elsewhere (p. 6) but it should be stressed that the picture of ' surgical shock ' should not be ascribed to the head injury unless the presence of another major injury has been excluded.

(iii) *Neurological assessment*

(*a*) Level of consciousness.—This is the single most important neurological observation, and one that causes greatest difficulty in accurate description and recording. Terms and gradings which require definition (e.g. ' coma', ' stupor ') should be avoided. A brief description in simple words which include the degree of alertness, ability to communicate or obey commands, and type of spontaneous or reflex limb movements is often satisfactory. The Glasgow Coma Scale (fig. 415) is a valuable method of recording consecutive

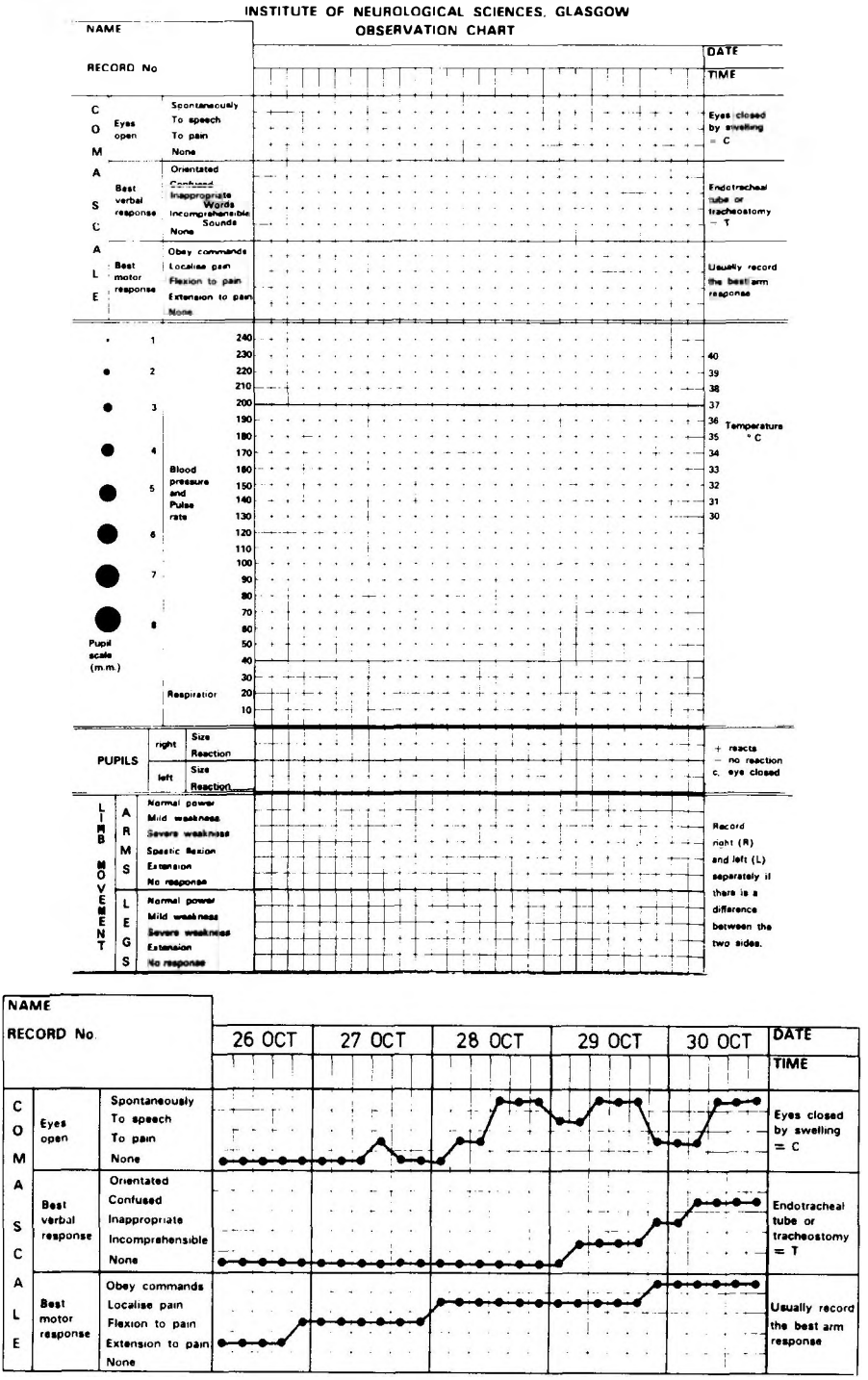


FIG. 415.—(a) The Glasgow Coma Scale; (b) details of a case.
(Institute of Neurological Sciences, Glasgow.)

observations, and of observing progressive changes but without requiring previous definition of terms used.

(*b*) Pupils.—When both pupils are small on admission and later one dilates, that provides the most accurate clinical guide to the lateralisation of a supratentorial compressing lesion (see Coning, p. 424). If however one pupil was known to be dilated immediately after the injury it is usually due to direct injury related to the orbit and neither pupil can be used later as a guide to lateralisation. Therefore the first assessment and recording of pupillary size is of extreme importance; size and equality rather than reaction to light is the more important feature, and should be recorded graphically or verbally.

(*c*) Limbs.—The types of spontaneous or reflex limb movements are an essential part of the assessment of the level of consciousness (see above). Hemiplegia is as likely to be due to a ' stroke ' prior to injury as to trauma; in the acute stage it is very rarely due to a compressing intracranial hæmatoma, but may be due to the primary cerebral damage. Alone it is never an indication for urgent surgery. Of more immediate significance is the detection of paraplegia or quadriplegia indicative of a spinal injury (p. 477).

(*d*) Cranium.—The position and nature of the external trauma should be noted accurately, the wound over an obvious fracture should be cleaned with saline and surrounding hair removed for a distance of at least 5 cm, and a head dressing applied. The presence of CSF rhinorrhoea, or otorrhoea is noted, and special note made of any mandibular or facial fracture which might lead to further difficulties with the airway.

(*e*) History of injury.—With the severely injured the initial assessment and resuscitation is usually done before a history of the injury can be obtained. However, that information is important, particularly if a preceding medical condition has led to the injury. Information from witnesses, ambulance attendants and relatives should be sought. *Alcohol* poses particular problems, which are often greater in those who have been less severely injured, and it makes accurate assessment of neurological lesions more difficult, and may be misleading when deterioration in conscious level due to an intracranial hæmatoma occurs.

3. Radiography.—The purpose of *skull radiographs* on admission are:—

(i) The demonstration of a skull fracture whose position may provide a valuable guide to the *site* of an extradural hæmatoma which might develop later.

(ii) The presence of a skull fracture provides some indication of the severity of the injury.

(iii) The demonstration of a depressed fracture which may require surgery.

(iv) The demonstration of a calcified pineal gland and its position relative to the midline, which may change in subsequent radiographs.

(v) The presence of intracranial air.

Therefore the radiograph on admission may provide an essential base line when later deterioration occurs, and in the early management it can be argued

that the radiographs are more important in patients who are relatively well than in those who are deeply unconscious. Unless radiographs are of reasonable quality they are valueless and may be dangerously misleading (figs. 416, 417), and in those circumstances it is wiser to defer radiography

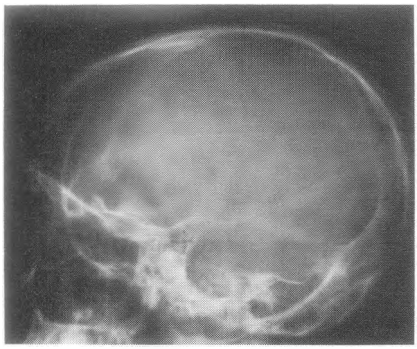


FIG. 416.—Skull radiograph of poor quality fails to show fracture.

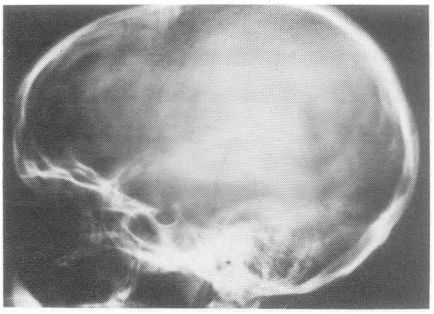


FIG. 417.—Good quality radiograph shows fracture.

until films of good quality can be obtained. The skull radiographs usually required are the right and/or left lateral, the half-axial (35°) and the antero-posterior (20°). However a *chest radiograph* is essential, as is a *lateral radiograph of the cervical spine* to demonstrate clinically undetectable fractures or fracture dislocation (p. 479). If patients are in urgent need of intensive care, time should not be wasted in trying to obtain skull radiographs.

4. Continuing Care.—The objects of the further care are: (*a*) by nursing and other intensive care measures to enable the patients to survive a period of 'unconsciousness'. (*b*) By repeated observation to detect at the earliest possible moment the development of complications which may need surgical or medical action.

Care of the 'unconscious' patient.—The quality of care given, which largely depends upon the availability of experienced nursing staff will determine the outcome in the majority of patients. Attention to airway, skin, eyes, mouth, bladder and bowels, limb joints, nasogastric tube feeding and intravenous infusions are very demanding and the surgeon must take a personal interest in these matters if morale is to be maintained. *Controlled ventilation* is used widely if spontaneous respiration is inadequate to maintain normal levels of pCO_2 and pO_2. *Tracheostomy* is very rarely necessary in the first few days after injury because an endotracheal tube may be used for up to seven days; thereafter if the airway or respiration is inadequate, tracheostomy may be required. As the level of consciousness improves there may be a stage of irritability which is difficult to control; drugs are best avoided because all *sedatives* tend to depress respiration to some extent, which may lead to a gradual rise in the pCO_2 and deterioration due to brain œdema even ten days after injury. Clearly if the patient is conscious enough to experience severe pain from other injuries (e.g. limbs) it is inhumane to withhold *analgesics* such as Codeine Phosphate (100 mg), but morphia is best avoided. Because

epilepsy is a major cause of deterioration (see below) all patients should be on prophylactic *anticonvulsants*; adult drugs and dosages are phenobarbitone 60 mg 8 hourly, or phenytoin 100 mg 8 hourly, and neither will depress the level of consciousness.

5. Deterioration.—The most important indication of deterioration following head injury is a decline in the level of consciousness. Its recognition requires accurate observation and accurate sequential recording (see Glasgow Coma Scale, fig. 415). The major causes of deterioration are:—

(i) Airway obstruction/hypoventilation *causing* brain œdema *causing* rising intracranial pressure.
(ii) Brain œdema *causing* rising intracranial pressure.
(iii) Intracranial hæmatoma.
(iv) Epilepsy.
(v) Fluids and dehydration.
(vi) Fever—infection—meningitis.
(vii) Blood loss from other injuries.
(viii) Aerocœle.

Although œdema is a more frequent cause of deterioration than compressing intracranial hæmatoma, it must be assumed that the deterioration is due to a hæmatoma until proved otherwise. The exceptions to this rule are: epilepsy, fluid imbalance, infection and fever, and blood loss. Unfortunately airway obstruction and inadequate ventilation may not only cause increased brain œdema, but also the addition of brain œdema in the presence of a hæmatoma and therefore it is not always safe to assume that œdema alone is the cause of the deterioration following airway obstruction.

Epilepsy may cause rapid deterioration in level of consciousness and if a convulsion is not witnessed it may be difficult to differentiate this from the occasionally rapid effects of cerebral compression. A convulsion does not indicate the presence of a compressing hæmatoma.

Fever from any cause will produce deterioration. *Meningitis* may follow injury especially with basal skull fractures and tends to present at about the third to fifth day after injury. At that time, in the presence of fever and neck stiffness diagnostic lumbar puncture is indicated.

Fluid imbalance and particularly dehydration may produce a gradual deterioration over the course of 48 hours; clinical examination, review of fluid charts and plasma electrolytes should detect the imbalance, and the level of consciousness improves with correction.

6. Indications for Surgery without Special Investigations (Decision Making).

The SIGNS OF CEREBRAL COMPRESSION are DETERIORATION IN LEVEL OF CONSCIOUSNESS, SLOWING PULSE, RISING BLOOD PRESSURE and SLOWING RESPIRATION.

In the presence of these signs, and having reasonably excluded other causes (see above), the conclusion must be that:

This patient has a compressing hæmatoma until proved otherwise.

In these circumstances neurosurgical advice should be sought because either (*a*) The patient should be transferred to a neurosurgical department or (*b*) Surgical action must be taken without delay and without special investigations. The decision taken will depend upon the rate of deterioration, dilatation of one or both pupils, the travelling time to (rather than the distance from) the neurosurgical department, the experience of the surgeon and the availability of special surgical instruments.

Surgery without special investigations.—The prime purpose of such surgery is to locate and evacuate, or exclude an *acute extradural hæmatoma* (fig. 418), an essentially remediable lesion, in a patient who is rapidly deteriorating. (The clinicopathological aspects of acute extradural hæmatoma are described on p. 421, and the reader may need to refer to this section before that which follows.) The major decisions before embarking upon urgent exploratory surgery concern:—

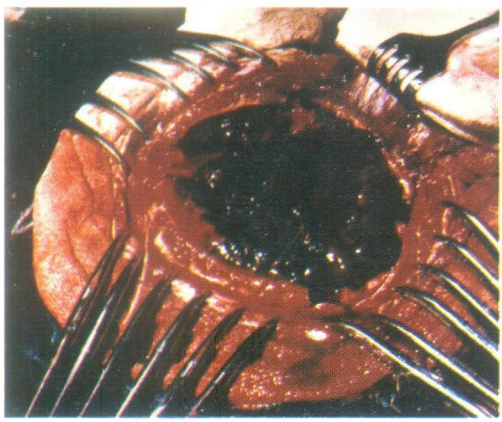


FIG. 418.—Acute extradural hæmatoma.
(*M. B. Yorston, F.F.A.R.C.S., Southampton.*)

(i) *The presence or not of a hæmatoma* (determined by the signs of rising pressure, see above).

(ii) *The side for exploration.*—For this the essential guides are in order of reliability (*a*), the shift of a calcified pineal on X-ray and (*b*), the side of the first pupil to dilate, provided it was not dilated immediately after injury. (See casualty reception.)

(iii) *The position of exploratory burr holes* on the appropriate side. In order of importance the guides are:—

(*a*), the position of a vault or squamous temporal fracture; (*b*), the site of external trauma; and (*c*), the standard temporal position (see below).

Pre-operative.—Ideally general anæsthesia with endotracheal intubation and controlled ventilation should be used. However, if that is not available, time should not be lost, an endotracheal tube should be inserted, and operation done under local anæsthetia. Having determined the likely side of the compressing lesion (see above) the patient is positioned supine with a sandbag under the appropriate shoulder and the face turned to the opposite side. It is wise to remove all the hair, or at least that of the appropriate half of the head, and that side is shaved. The sterile towels should be arranged in a way that allows access to frontal, temporal and parietal regions. Blood should be available, but surgery should not be delayed if it is not.

Operation

The exploratory burr hole (position see above).—A 3 cm vertical incision is made *down to bone*; a self-retaining mastoid-type retractor is inserted and opened forcefully thus controlling scalp and muscle bleeding. The incision for the *standard temporal burr hole* is *immediately* above the zygomatic arch mid-way between the posterior margin of the orbit and the external auditory meatus (fig. 419). At that level the squamous temporal bone lies relatively deeply, but no damage can be done in reaching it, and it is essential to use the self-retaining retractor to control the scalp

and muscle bleeding. Using a brace and bit with first the sharp point, and, when the inner table of the skull is pierced, the reamer which has a protective flange, a burr hole is made.

If a significant extradural clot is present the dura will not be seen because the dura will have been separated from the brain by the clot which has the appearance of black-currant jelly. For the same reason the responsible bleeding dural artery (e.g. middle meningeal) will not be seen.

Further action.—A. Extradural clot is not present.—Bearing in mind that the essential purpose of this exploratory surgery is the exclusion or evacuation of an *extra*dural clot it is unwise to open the dura at this stage. If the first burr hole has been placed over or next to the fracture, it is reasonable to make a second burr hole in the *standard temporal position.* If that also is negative it is reasonable to make a frontal or parietal burr hole if the original guides have not already included those

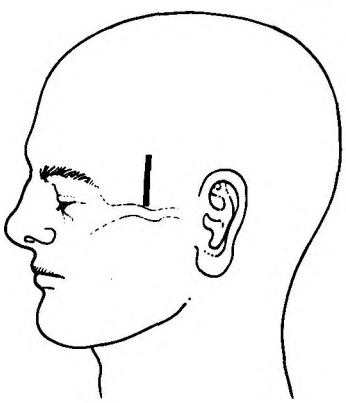


FIG. 419.—Incision for standard temporal burr hole.

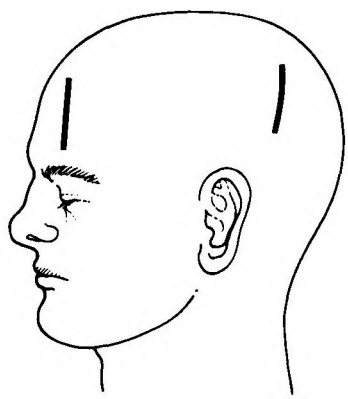


FIG. 420.—Incisions for frontal and parietal burr holes.

positions (fig. 420). If none of these burr holes on one side have revealed an extradural clot two further questions arise:—

1. *Should the dura be opened?* The assessment of the thickness and therefore significance of an acute *subdural* clot by inspection through a burr hole is extremely difficult. Only specialised radiology can assess this accurately and show the extent as well as the thickness of the lesion. Whenever there is primary brain damage (contusion, laceration) there is some clot in the subdural space but this is rarely a significant compressing lesion. Furthermore the satisfactory evacuation of a significant subdural clot requires a major and often larger craniotomy (osteoplastic flap) with a wide dural opening; these procedures require particular neurosurgical expertise and facilities, and the surgery itself may lead to further technical problems such as herniation of a swollen brain. Therefore if an extradural hæmatoma is not found, *the dura should not be opened,* ventilation should be continued, an osmotic diuretic given (Mannitol, p. 437) and the patient transferred for special radiology.

2. *Should a burr hole be made on the other side?* If the radiologically demonstrated shift of the pineal has been used to determine the side of the compressing lesion nothing will be gained by making a burr hole on the opposite side. If the side of the first dilating pupil and the side of the fracture and/or site of external trauma coincide, a contralateral burr hole need not be made. If however the fracture or evidence of external trauma (e.g. bruising and swelling in the temporal region) is on the side opposite to the first dilating pupil, the compressing lesion is likely to be *contre-coup* (p. 425). However, very rarely the pupil may mislead and therefore a final burr hole should be made over the fracture and/or site of external bruising or swelling.

Further action.—B. Extradural clot present.—Assessment of thickness of the clot is made by using a blunt-nosed brain cannula with centimetre marks and feeling the resistance of the dura; any clot greater than 0·5 cm thick is significant. Sometimes the burr hole will have been placed just at the edge of the hæmatoma which is

not, therefore, immediately visible; only by gently inserting a dissector and depressing the dura will the clot be detected (fig. 421).

Evacuation of clot.—(*a*) *By craniectomy:* The burr hole incision is extended in a straight line to a length of about 8 cm (fig. 422), a second self-retaining retractor is inserted and with bone nibblers the burr hole is enlarged to a craniectomy of at least 7 cm diameter (fig. 423). The clot is then gently lifted off, this and the bone removal

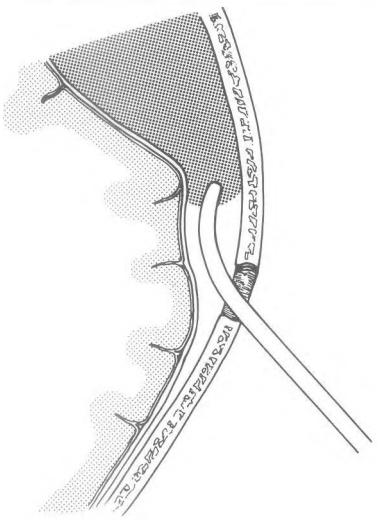


FIG. 421.—Burr hole near edge of extradural haematoma.

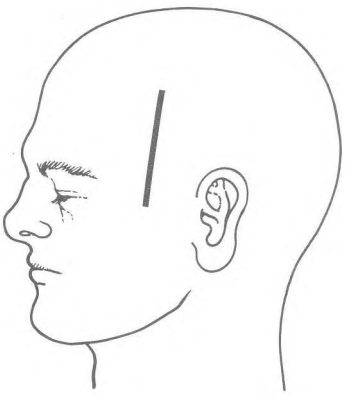


FIG. 422.—Extension of incision for craniectomy.

decompressing the brain. Small fragments of clot adherent to the dura are left because their removal only produces troublesome oozing from the dura to which ' Oxycel ' may be applied. If a major dural vessel is bleeding it is controlled either by diathermy or by a stitch passed under the vessel. The dura is then ' hitched ' to the surrounding bone by passing sutures (e.g. black silk) through the superficial layer of the dura and the surrounding muscle or pericranium; this step is essential to prevent the re-accumulation of a hæmatoma (fig. 424). The muscles, temporal fascia, galea and skin are then closed in layers without drainage and a full head dress using gauze and a crepe bandage is applied.

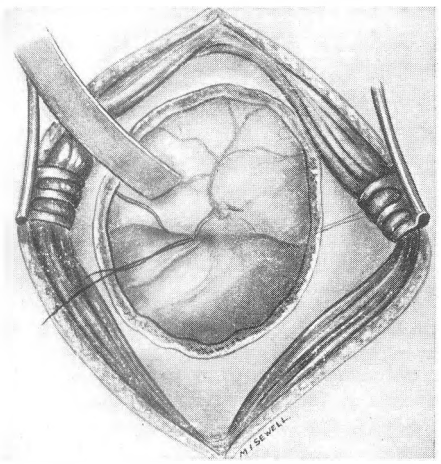


FIG. 423.—Craniectomy and control of meningeal artery by dural stitch.

(*b*) *By craniotomy and osteoplastic flap.*—In experienced hands this gives wider access without any appreciable loss of time in decompressing the brain and is generally used in neurosurgical departments, but does require familiarity and neurosurgical instruments. The stages are the turning of a scalp flap, the placing of multiple burr holes which are then joined by a Gigli (note p. 415) saw and the raising of a quadrilateral bone flap hinged at its base upon the temporal muscles (figs. 425 and 426).

Postoperative.—As soon as the conscious level permits the patient should be mobilised; skin sutures are removed on the second or third day. Antibiotics are not used routinely. Prophylactic anticonvulsant drugs (see Continuing Care) should be continued for about six weeks in the absence of any other indications for their longer term use.

7. Special Investigations.—Once the decision has been taken that there is time for the deteriorating patient to be transferred to a department with access to special neuroradiological facilities, it is reasonable to give osmotic diuretics (p. 437) to gain temporary improvement during transfer. However, even in neurosurgical departments, there are occasions when, with patients who are deteriorating very rapidly and whose second pupil is dilating, there can be no further delay for radiology and exploratory burr holes are made (see ' 6 ').

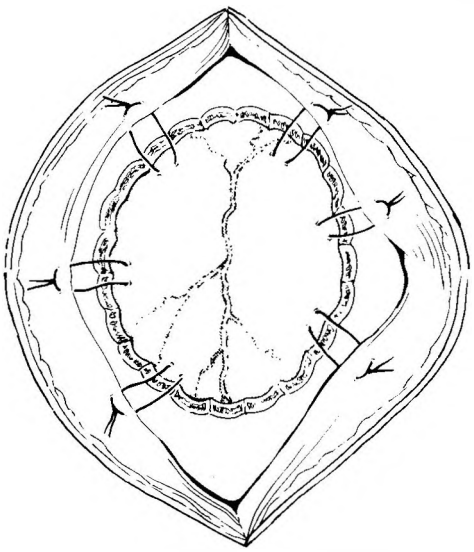


FIG. 424.—Dural ' hitching ' stitches.

Computerised Axial Tomography (CT scan).—This non-invasive type of investigation provides the most accurate guide to intracranial pathology following injury, and is available in all neurosurgical departments and to many general departments. It has revolutionised the investigation of head injury, intracranial hæmorrhage and infarction, and space occupying lesions. When available it displaces the use of arteriography and air study. The investigation causes no disturbance to the patient and eliminates exposure to conventional X-ray investigations.

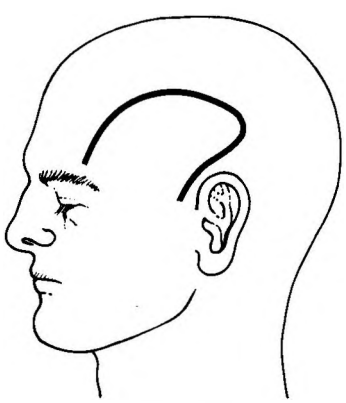


FIG. 425.—Incision for temporal craniotomy.

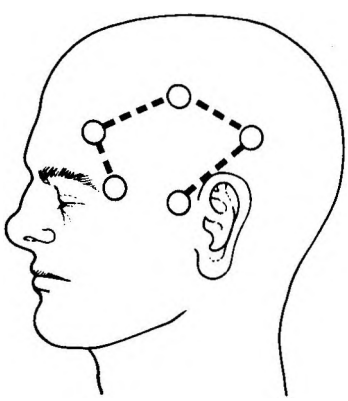
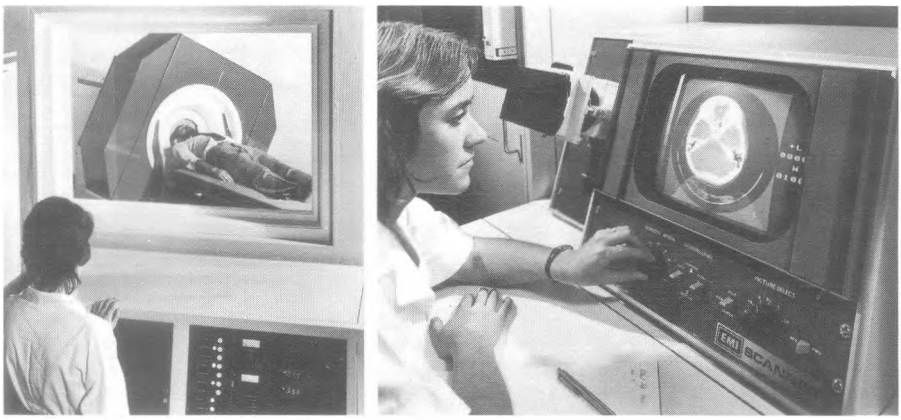


FIG. 426.—Burr holes and saw cuts for temporal craniotomy.

The patient lies on a motorised couch in order that the part to be examined can be positioned within the scanning gantry (fig. 427). The head is scanned from above downwards in a series of transversing planes like a tomograph from vertex to base of skull by tilting the X-ray tube and detector array within the gantry at each scan.



FIGS. 427 and 428.—The Brain Scanner. The technique of computerised tomography originated in 1968 in EMI's Central Research Laboratories.

The information is fed to a computer and produces a record in which high density objects such as bone, clot or tumour appear white, the brain substance appears grey, areas of œdema a dark mottled colour and the ventricular fluids black. Collectively the scanner produces a complete picture in which tumours, clots, infarction, ventricular displacement or hydrocephalus are readily distinguished (fig. 428).

Its major value is in the differentiation between brain swelling, contusion and compressing hæmatoma, and the accurate demonstration of the extent and thickness of a hæmatoma, and therefore the need for surgical evacuation. The demonstration of œdema provides a guide to the use of osmotic diuretics (p. 437) and of controlled respiration (figs. 429, 430). So valuable is this

FIG. 429.—CT Scan. Acute extradural hæmatoma.

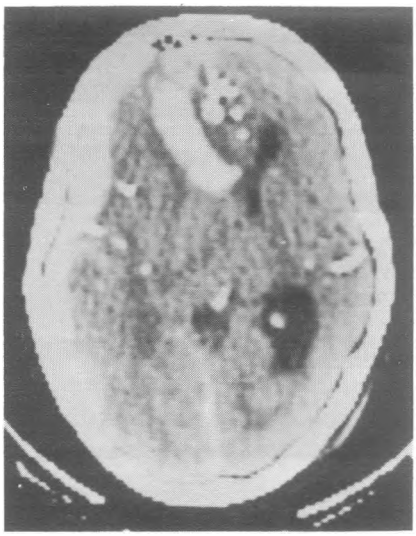


FIG. 430.—CT Scan shows left hemisphere swelling, ventrical displaced to right, contusion, intracerebral blood and subdural clot.

method of investigation that there are now strong grounds for transferring all patients with severe head injuries to hospitals where CT scan is readily available. It has largely replaced other methods of investigation.

Carotid arteriography, in the absence of CT scan, remains a valuable method of investigation particularly in the demonstration of extracerebral hæmatomas (extradural and subdural). In those conditions there may be displacement of the middle cerebral artery, the anterior cerebral artery may be displaced from its midline position away from the side of the hæmatoma and the cortical vessels do not reach the inner table of the skull (fig. 431).

Ultrasound has been of value in demonstrating the position of midline structures by their characteristic echo but it does not approach the accuracy of the calcified pineal visible on plain radiographs or the information gained from CT scan.

Intracranial pressure monitoring is a very specialised method of investigation which may be of value in continuing and postoperative care but is not relevant to the acute situation on admission to hospital.

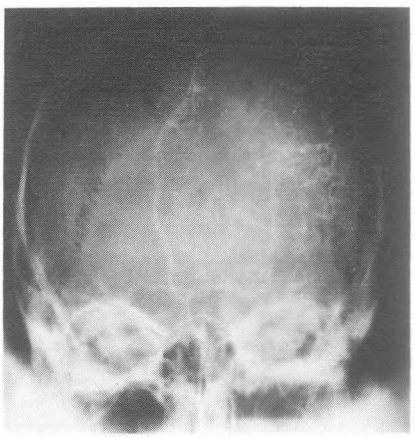


FIG. 431.—Carotid arteriogram showing mid-line shift and bare area over cortex due to clot.

8. Non-surgical Therapy.

Osmotic diuretics.—The basis for the use of these substances is that by raising the osmolarity of the plasma, fluid will be drawn from the brain extravascular compartment into the blood, thereby reducing brain œdema and swelling. Therefore solutions given intravenously for this purpose must be hyperosmolar, non-diffusible (i.e. they should not pass across the blood-brain barrier), non-irritant and be not excreted too rapidly by the kidney, or metabolised rapidly. In the past 50% sucrose was used but to little effect; lyophilised urea was more effective but because it was diffusible there was a considerable 'rebound' effect. Today *Mannitol* as a 20% solution given intravenously to an adult as a volume of 250 ml (or in dire circumstances 500 ml) over the course of 20 to 30 minutes is the agent in general use. It may be used repeatedly at 6 or 8 hour intervals but the following must be noted:—

(*a*), There should be satisfactory renal function; (b), if a diuresis has not occurred following Mannitol, further administration should be delayed; (*c*), fluid and electrolyte replacement should be meticulous, particularly with the loss of electrolytes with each diuresis; (*d*), if hyper-osmolarity of the blood is to be produced, the rate of administration is critical.

There are *potential dangers* in the use of Mannitol before either a diagnosis has been established or a course of action instituted. Thus although the cause of a patient's deterioration may well be brain œdema, giving Mannitol may produce temporary improvement in the presence of a large hæmatoma, soon to be followed by profound deterioration. Therefore Mannitol should not be used in the acute stage following injury unless the possibility of an

intracranial hæmatoma has been excluded in a patient who is deteriorating, except to gain time prior to surgery. It should never be used as a diagnostic test to differentiate between œdema and hæmatoma.

Frusemide (40–80 mg by intramuscular injection) is an alternative agent which produces a very rapid diuresis and thereby raises the osmolarity of the vascular compartment. It is non-irritant, but requires good renal function for its effect.

Cortico-steroids (Dexamethasone and Betamethasone).—In high doses (up to 24 mg a day) these drugs are effective symptomatically in malignant brain tumours, the main effect being upon surrounding brain œdema. In the management of severe head injuries they have been used widely for some years, often in doses of up to 60 mg a day. Unfortunately this practice is based more upon tradition than objective evidence, because at present there is no evidence that their use improves the outcome in severe head injury.

9. Miscellaneous Complications—Early

'Brain-stem injury.'—This term is used loosely to describe a clinical picture which is ascribed to primary damage in the brain-stem (medulla and pons). It should be stressed that the changes may follow unrelieved supratentorial compression. The patients are often children or young adults, the latter being motor cyclists who, wearing crash helmets, suffered an acute flexion/extension movement of the neck at the moment of impact on the vertex. Signs of external trauma may be absent, but the patient is 'unconscious', with spontaneous extensor spasms of all four limbs, arching of the trunk (opisthotonus), a rapid pulse, rapid and often shallow and therefore inadequate respiration, small pupils, pyrexia and sweating. Any stimuli will tend to increase the tendency to extension. If this state has been present from the time of injury the likelihood of a compressing hæmatoma is remote. Prognosis depends upon intensive nursing care, and control of respiration; the ultimate intellectual deficit may be considerably less severe than following major supratentorial brain damage, but there is often marked spasticity and inco-ordination.

Posterior Fossa Compression.—Compression of the cerebellum and medulla by posterior fossa hæmatoma is rare. However in the presence of a fracture which passes towards the foramen magnum and therefore crosses the lateral sinus an extradural hæmatoma may occur. These tend to present somewhat later than the supratentorial extradural hæmatoma, usually after 48 hours and the most important physical sign is *slowing and irregularity of respiration*, which may precede other evidence of deterioration. Special investigation by CT scan or ventriculogram is needed prior to a posterior fossa craniectomy.

CSF Rhinorrhœa.—For CSF rhinorrhœa to occur there must be a communication between the intracranial (intradural) cavity and the nose. Therefore CSF rhinorrhœa indicates a tear of the dura (usually basal) and a fracture involving the paranasal sinuses, frontal, ethmoid or sphenoid. At the moment of impact not only does the dural tear and fracture occur but also a

plug of brain may be forced into the dural tear; although this may temporarily seal the defect it also prevents dural healing and therefore the CSF rhinorrhœa may persist and the patient be at risk from meningitis which is usually pneumococcal. CSF rhinorrhœa may occur in association with displaced fractures of the middle third of the face (p. 534).

Although there are different views on the management of CSF rhinorrhœa a reasonable course is:—

1. Initially the patient is given prophylactic antibiotics (penicillin and sulphonamides).

2. Fractures of the middle third of the face are reduced; in many cases the rhinorrhœa may cease.

3. The indications for anterior fossa exploration are CSF rhinorrhœa persisting for more than ten days, the presence of a fracture involving the frontal or ethmoid sinus, an aerocœle (see below), and an attack of meningitis which has been treated. Factors such as age, neurological state, and degree of disruption of the anterior fossa floor are taken into consideration.

Aerocœle.—Entry of air into the cranial cavity usually occurs in association with CSF rhinorrhœa. Air may enter the subarachnoid space and ventricular system (a ' spontaneous ventriculogram ') or the substance of the frontal lobe because the brain is adherent to the margin of the dural defect. This occurs particularly if the patient blows his nose. Rarely a frontal aerocœle may cause compression and therefore deterioration about two or more weeks after injury (fig. 432). Surgery is similar to that for CSF rhinorrhœa.

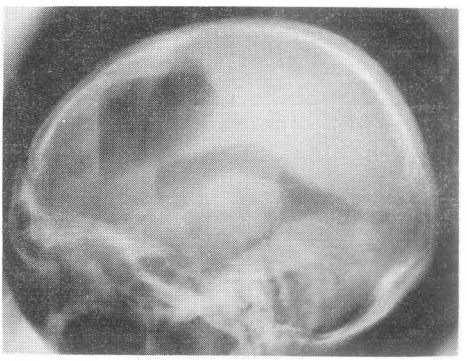


FIG. 432.—Frontal aerocœle and spontaneous ventriculogram fracture through frontal sinus.

Meningitis.—Apart from its association with CSF rhinorrhœa and otorrhœa, meningitis may occur after any major head injury particularly if a basal skull fracture is present but often undetected (see p. 444). Bleeding into the subarachnoid space is common with head injury, and neck stiffness immediately after injury is *not* an indication for lumbar puncture. Meningitis when it occurs does so after the first 48 or 72 hours and, therefore, provided there are no signs of cerebral compression, fever and neck stiffness are, at that stage, an indication for lumbar puncture. If there are also signs of cerebral compression the patient should be transferred for urgent intracranial investigation before lumbar puncture.

Pituitary Failure.—Occasionally basal fractures may pass across the pituitary fossa causing acute pituitary damage and endocrine failure. This may lead to a profound fall in blood pressure, tachycardia, pallor and hypothermia with deterioration in level of consciousness. Once the condition is

recognised high doses of steroids should be given (Hydrocortisone 200 mg 6 hourly for the first 24 hours, followed by reduced and then maintenance dosage).

Fat Embolism.—Systemic fat embolism may cause diagnostic difficulties when patients with multiple injuries which include a head injury show neurological deterioration at about 48 hours after injury or surgery for limb fractures (p. 156). Neurological features which suggest fat embolism rather than an intracranial hæmatoma are the time of deterioration, the delayed onset of features of a brain-stem injury (see above), pupils which vary in size from moment to moment but which remain equal, the presence of small retinal hæmorrhages, and the absence of any firm lateralising signs. The finding of fresh petechiæ over the upper part of the trunk and in the axillæ is of help. However in the absence of positive signs of fat embolism, it is usually wise to check the position of the pineal if it is visible, or to obtain a CT scan.

FRACTURES OF THE SKULL

Mechanism.—Fractures of the vault and base of the skull are produced: (1) by compression of the sphere; (2) by local indentation; (3) by tangential injury.

Fractures of the vault produced by compression of the sphere are the commonest skull fractures, and result from distortion of the skull when it comes into contact with a hard flat surface. Usually closed linear fissures are produced which, although not important in themselves, are often associated with severe diffuse cerebral injury resulting from brain displacement occurring at the moment of impact.

Fractures of the vault produced by local indentation give rise to closed or compound depressed fractures according to the surface area of the injuring agent.

Blows from large rounded objects produce *closed pond depressed fractures*; the scalp is bruised but remains intact. The brain surface is bruised under the intact dura, but not penetrated. This bruising may cause local signs, such as paralysis or aphasia. Healing occurs by absorption of the tips of damaged convolutions forming a shallow

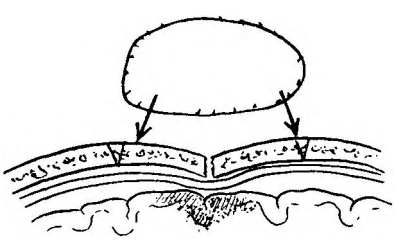


FIG. 433.—Pond fracture produced by large round object.

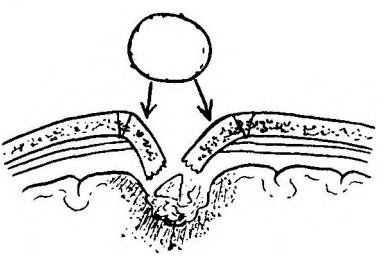


FIG. 434.—Compound fracture produced by small round object.

erosion on the brain surface, surrounded by a zone of reactive gliosis. The risk of epilepsy is about 4 per cent. (fig. 433).

Blows from small round objects produce *compound* depressed fractures. The scalp is irregularly torn by the indentation. The fractured bone is depressed, lacerating both the dura and subjacent brain. Fragments of bone may separate from the inner table and lodge in the brain. There is immediate risk of infection, and a later risk of epilepsy resulting from the contracting fibrous scar through the open dura, which increases the incidence of epilepsy to 25 per cent. in untreated cases (fig. 434).

Fractures of the Vault by Tangential Injury.—Tangentially directed violence may secure a grip on the skull and prise up large segments of bone, which are elevated leaving the dura intact.

When a window cleaner, falling to earth, catches his brow on a window ledge, the vault of the skull can lift like a lid of a coffee pot, a horse-shoe fracture surrounding the calvarium. Such injuries are compound and the skull X-ray is alarming, but there may be little or no associated cerebral injury, as the injuring force was not directed on to the brain.

Closed depressed fractures of the vault are rare. The scalp is relatively non-elastic and an injury sufficient to fracture and depress the bone is almost certain to lacerate the scalp. Hæmatomas in the scalp sometimes simulate depressed fractures. Clotted blood in the margin of a hæmatoma may, on palpation, resemble the edge of a fractured zone (fig. 435) but the edge of the clot can sometimes be indented, often X-ray is required to distinguish the two conditions.

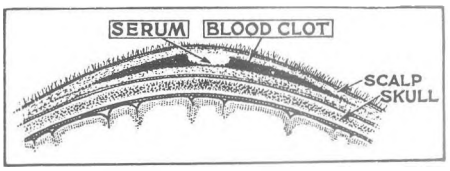


FIG. 435.—Hæmatoma simulating a depressed fracture.

Treatment.—Operation is not indicated if the fracture is small provided that there are no sharp spicules of bone which have penetrated the dura (fig. 436) and the fracture does not overlie an important cerebral area. Intervention is required if the fracture lies over the speech or motor areas, or if the X-ray suggests that spicules may have penetrated the dura mater, when scarring may cause adhesions between the brain and membranes, with risk of subsequent epilepsy (p. 446). Depressed fractures related to the sagittal sinus should be managed conservatively (fig. 437).

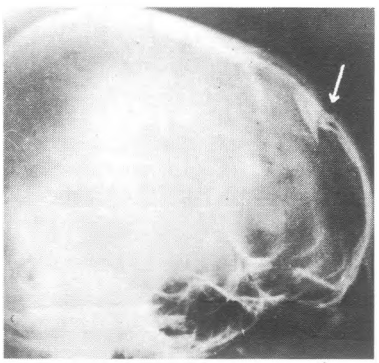


FIG. 436.—Sharp bone spicules penetrating the dura.

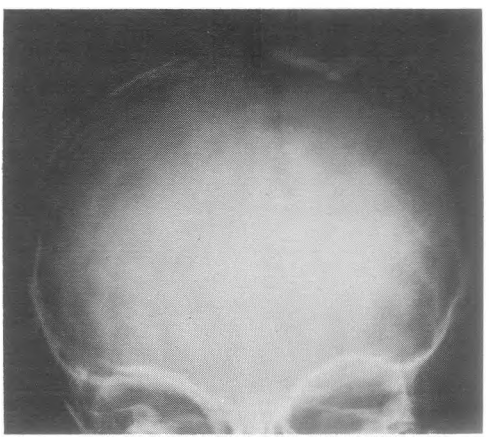


FIG. 437.—Closed depressed fracture involving saggital sinus. Management is conservative.

Operation.—Merely to trephine and elevate the depressed bone is quite inadequate in this case as a proper examination of the brain and membranes is not possible. An osteoplastic flap, which includes the fracture, is raised, and the depressed bone is moulded into position. The dura is examined, penetrating spicules are removed, and the membrane is then opened. Pulped brain is carefully removed. Adhesions are separated. Hæmostasis is secured and the dura is carefully sutured, if necessary a fascial graft being inserted to prevent the ingrowth of fibrous tissue. The flap is then replaced and the wound closed.

Pond-shaped depressions occur in infants, as a result of prolonged pressure of the head against the promontory during birth, or of direct injury during the first few months of life. Spontaneous elevation usually occurs within a few weeks. Early elevation is required if an extensive depression overlies the speech or motor areas or

if there are signs of compression. Operation is also indicated if a depression anterior to the hair line constitutes a cosmetic deformity which does not undergo a spontaneous elevation within six weeks. As the formation of an osteoplastic flap would lead to extensive damage to the extremely thin dura of an infant, a simple elevation is performed by means of a guarded gimlet, i.e. silk is wound around the gimlet to within 6 mm. of its point. A small incision is made over the centre of the depression, and the skull is perforated by the gimlet. An aneurysm needle is introduced through the aperture, and by this means traction is applied to the depressed bone. The elastic skull of the infant readily yields and springs back to its normal position.

An alternative is the application of the obstetric vacuum extractor which is frequently sufficient to elevate the fracture.

Compound fractures of the vault vary in severity, from severe crushes, with the escape of cerebral substance, to relatively insignificant compound fissured fractures. High-speed motor-cycle accidents produce the worst examples of severe frontal crushes, many are rapidly fatal. Severe cases which survive exhibit serious disability, such as hemiplegia or mental deterioration from loss of cerebral substance. The incidence of these injuries has been reduced by the wearing of crash helmets.

When the velocity of impact is low, large compound depressions can occur with less diffuse cerebral injury, particularly when produced by blows from the side—in which the midline septum of the falx will limit the associated brain displacement.

Clinical Features.—An irregular lacerated wound of the scalp is associated with a boggy subgaleal swelling produced by blood escaping from the fracture line, which is partly exposed in the depth of the wound. A normal suture line should never be mistaken for a fissured fracture, for a suture line is irregular and free from oozing blood. Detection of fractures by probing scalp wounds is potentially dangerous; only skull radiographs can provide accurate information safely. In compound depressed fractures the fracture lines extend far laterally beyond the limits of the surface wound (fig. 438), and this must be allowed for in planning the requisite exploration.

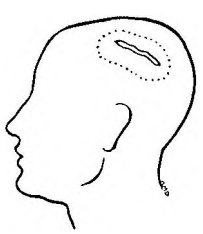


FIG. 438.—Relative extent of the fractured area (shown by dotted line) in comparison with the surface wound.

Treatment.—Operation should be undertaken within 48 hours unless the patient's condition is so precarious that delay is advisable. Antibiotics should be given in full doses.

Pre-operative X-ray is essential to determine the extent of the fracture.

The head is shaved well beyond the margins of the fracture.

Unconscious patients may be operated upon under local anæsthesia.

In frontal fractures severe bleeding from the nose must be cleared by suction.

Small compound fissure fractures may be exposed by extending the primary scalp wound after excision of its edges. Extensive depressions are exposed by forming a horseshoe scalp flap around the margins of the fracture site after previous excision and suture of the scalp wound (fig. 439). A burr hole is formed beside the fracture to permit unlocking of the fractured particles (fig. 440). Bone which has been contaminated by dirt is discarded. Clean bone is preserved. The dura is inspected. If intact, it is not opened. If penetrated, indriven material and damaged brain tissue are removed by suction and the dura is then sutured. Clean portions of bone may

now be moulded into place, the wound being then sutured and antibiotic treatment commenced. Careful judgement is required concerning the discarding of bone. If a tangential injury has lifted up large areas of the vault, the frontal plates, or roof of the orbit, far beyond any *exposed wound*, it is only necessary to remove contaminated areas in the wound. Removal of the entire detached area of bone would produce enormous unsightly deformities which could not be adequately repaired.

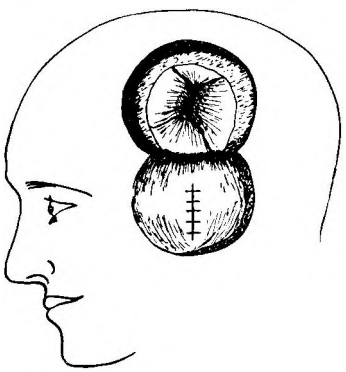


FIG. 439.—Exposure of depressed compound fracture. The original scalp wound has been sutured and a horse-shoe scalp flap reflected to expose the full extent of the fracture. (*G. B. Northcroft, F.R.C.S., London.*)

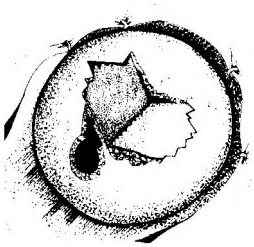


FIG. 440.—Formation of burr hole beside a fractured area to allow unlocking of the bone fragment.

Compound fractures of the frontal sinus become infected from the nose; hence the sinus must be obliterated. After excision of depressed bone and removal of the mucous lining a pressure pad is applied over the site to cause the scalp to sink into the depression resulting from excision of the sinus and to close the cavity by adherence to the dura. Three months later the sinus area is exposed by turning a scalp flap forward, the scalp is freed from the dura, and the deformity is restored by skull reconstruction.

Skull Reconstruction.—Areas of skull defect are restored at intervals of three to six months after injury, by the insertion of moulded tantalum plates or acrylic inlays. Concealed horseshoe or transverse incisions within the hair line are used for these purposes.

Head Wounds due to Missiles.—The outcome depends on the explosive impact of the missile in the cranium, which is the commonest cause of fatality, and the relation of the wound track to the great vessels and the ventricles. Through-and-through tracks, from side to side or front to back, may be survived. Survivable injuries can be transported to a suitable hospital. Projection of the swollen brain through the dura at the site of entry or exit forms a hernia cerebri, which seals off the subarachnoid space during transport (fig. 441). A pressure dressing is applied.

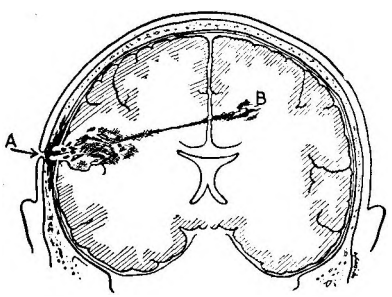


FIG. 441.—Transverse penetrating wound, entry at (A) showing: (1) Protrusion of brain through the dura at entry site forming hernia cerebri. (2) Pulped brain and bone close to entry. (3) A metal fragment at (B) has crossed the open superior fissure.

Operative treatment consists of the excision of the surface wound and suction, cleansing, and removal of foreign material from the track. The dura is closed by suture or grafting at entry and exit points. Intrathecal penicillin treatment will then prevent serious infection unless an infected track crosses the ventricles or median fissures (see also p. 448).

Penetrating wounds produced by sticks are always to be regarded very

seriously. Although scissors or pokers may enter the roof of the orbit when a child falls and thereafter be successfully withdrawn, a stick which goes in through the orbital roof, or backwards behind the orbit into the temporal lobe, cannot be withdrawn intact; portions which are left behind may, from previous contact with the ground, be infected with gas gangrene or tetanus, with probable fatal infection.

Fractures of the base of the skull are usually produced by compression of the sphere and extension of fissures radiating from the vault. Elsewhere the fracture lines tend to wander into the foramina for the exit of cranial nerves. The fractures become compound at the middle ear, accessory air sinuses, and the cribriform plates, but the evil reputation of fractures of the base is derived from the severe brain injury accompanying the fracture rather than from the risk of sepsis.

Clinical Features.—The essential features are:

(1) The escape of cranial contents, i.e. blood, or cerebrospinal fluid, or rarely, brain.

(2) Injury to cranial nerves. Laceration of nerves in fracture lines produces an immediate paralysis which will not recover. Compression of nerves by blood clot produces paralysis which, after a period of a few days, usually recovers. Paralysis occurring after a period of weeks is produced by contraction of scar tissue or callus and will not recover.

Anterior Fossa Fractures.—Epistaxis occurs if the cribriform plate is involved. Should the fracture extend into the orbit and subconjunctival hæmorrhage follow, the patch is wedge-shaped with the apex in front, and the posterior limit cannot be seen (the converse to what is observed with a 'black eye').

Cerebrospinal fluid trickles through the cribriform plate, often in surprising quantities, a condition referred to as 'cerebrospinal rhinorrhœa'.

The olfactory nerve is frequently torn, but unless its fellow is also damaged, the partial anosmia is likely to pass unrecognised. If the optic nerve is contused, any loss of vision will be permanent. Third nerve palsy produces a dilated pupil in a conscious patient.

Middle Fossa Fractures.—Epistaxis occurs if the fracture involves the nasal sinuses. Escape of cerebrospinal fluid and blood from the ear is common. Blood from torn tympanic membrane will clot, but when blood continues to drip for days, it is because it will not clot, owing to mixture with cerebrospinal fluid.

Serious arterial hæmorrhage from the ear may follow injury to the posterior branch of the middle meningeal artery (p. 422).

If the 7th nerve is torn, facial paralysis is permanent. Occasionally paralysis occurs at an interval of a few days after injury from compression by blood clot. Recovery is to be anticipated if the facial muscles are supported. If paralysis develops later, it is produced by pressure from fibrous tissue or callus, and the paralysis will be permanent. The 6th and 8th nerves may also be damaged.

Posterior Fossa Fractures.—Extravasation of blood in the suboccipital region produces a boggy swelling at the nape of the neck, or discoloration posterior to the mastoid process. The ninth, tenth, and eleventh nerves are occasionally damaged at the jugular foramen, but the twelfth nerve usually escapes, as it is protected by a bony buttress.

Management of a fractured base is that of the associated brain injury and CSF rhinorrhœa (see above).

LATE EFFECTS OF HEAD INJURY

Chronic Subdural Hæmatoma

This is produced by rupture of the veins passing from the cerebral hemispheres to the venous sinuses as the result of displacement of the brain inside

the skull. Usually the superior cerebral veins are ruptured, producing hæmorrhage over the convexity of the hemispheres; very rarely veins passing from the temporal lobe to the sphenoid or petrosal sinuses may produce clots which collect on the under-aspect of the brain. This complication, which is potentially fatal, is produced particularly by blows of small magnitude applied to the front or back of the head which may be insufficient to produce even transient concussion, but which are sufficient to move the brain suddenly. Cerebral atrophy renders this displacement easier, and hence the condition becomes commoner with advancing age. The superior cerebral veins pass from the convexity of the hemispheres and pierce the arachnoid membrane before crossing the potential subdural space to join the inner aspect of the dura 2·5 cm or more from the middle line; they then run inwards to drain into the lower compartment of the superior longitudinal sinus. Sudden displacement may snap the vein at the level of the arachnoid, allowing blood to pass downwards into the potential subdural space between the arachnoid and dura. Frequently, corresponding veins on both sides are affected and the condition is bilateral in 50 per cent of cases. The hæmatomas are often large, bilateral collections up to 60 ml a side or unilateral collections of 120 ml being quite usual. There is a progressive change in the nature of the subdural fluid which becomes thinner, lighter in colour and eventually is similar to CSF.

Clinical Features.—The symptoms may follow a preceding concussion, but owing to the slight nature of the force required to produce displacement, this complication may occur without preceding loss of consciousness and without the head being even struck. It can follow a sudden jolt, as when a driver is thrown against the steering-wheel of a car, or be produced by knocking the head against the lintel of a door, or landing heavily on the feet when jumping from a height; it has followed dental extraction and electroconvulsive therapy. The interval between ' trauma ' and onset of symptoms may be of weeks or months.

The symptoms are undramatic and consist of mental apathy, slowing of cerebration, slowness of response to questions merging into stupor. When the stupor develops, it comes and goes as the brain volume varies, the patient being inaccessible at times and then rousing sufficiently to answer questions accurately, but very slowly and after a considerable pause. When the level of consciousness deteriorates further the operative mortality rises to 30 per cent; hence the significance of the early symptoms.

Physical signs vary. In older patients, where more room is available and if the fluid collects slowly, there may be no signs, or at most a unilateral or bilateral extensor plantar response from pressure on the motor cortex or brain-stem displacement. Pupillary changes occur last when the brain stem is affected and pressure-cone formation is imminent. Lumbar puncture shows a fluid at low pressure with protein increased to 120 mg/100 ml (1·2 g/l), often stained yellow from the transudation of pigment, but no cells. Papillœdema is exceptional. Success in treatment comes from acting on suspicion and employing skull radiographs for the position of the pineal, CT

scan, or exploratory burr holes. Carotid arteriography should be avoided in the elderly.

Treatment.—Bilateral posterior parietal burr holes are made under local anæsthesia to expose the dura which often has a blue-green tinge. On incising the dura there is a gush of brown fluid. This should be allowed to flow out of the burr hole spontaneously, aided by lowering the head. The brain surface will frequently remain at a considerable distance from the dura. If the CT scan has shown a large collection of fluid but only a small amount has been obtained from the posterior parietal burr hole, another burr hole should be made further forward on that side (fig. 442A and B).

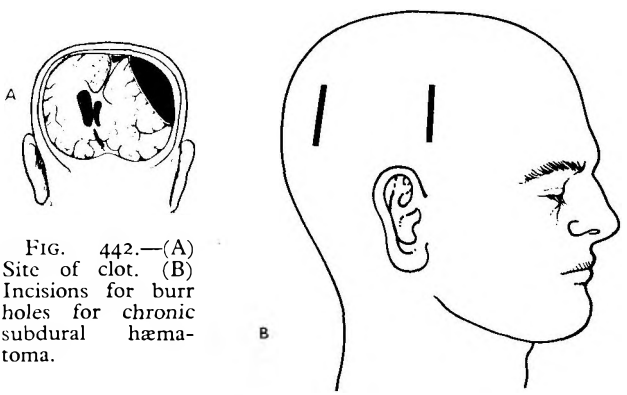


FIG. 442.—(A) Site of clot. (B) Incisions for burr holes for chronic subdural hæmatoma.

Postoperatively the patient is nursed flat initially and the subdural space can be re-tapped using a blunt-nosed brain cannula through the closed burr hole wounds.

Post-Traumatic Epilepsy.—Epilepsy occurring soon after injury does not indicate the presence of a compressing intracranial hæmatoma. It reflects degrees of primary brain damage which may be trivial and from which total recovery occurs. Children are more susceptible to early epilepsy than adults, and it is probably a reflection of an inherent epileptic tendency. *Epilepsy within one week* of injury occurs in about 10% of all cases admitted to hospital with a head injury; factors which tend to increase the incidence are prolonged post-traumatic amnesia, intracranial hæmatoma and depressed fracture. In these circumstances prophylactic anti-convulsants should be given for about six weeks, but for up to three years in those who have suffered from any post-traumatic epilepsy.

Late Post-Traumatic epilepsy has an overall incidence of about 5% but of up to 25% in those who have suffered from early epilepsy, and therefore similar factors are involved. It probably represents cortical scarring and reactive gliosis. The epilepsy may be focal, general or essentially temporal, and although in the majority of patients its onset is within the first year, in about 20% the onset is delayed for more than four years. When the onset is very late (e.g. 10 years) it may be wiser to investigate the patient as a case of 'late onset epilepsy' rather than assume that the epilepsy is post-traumatic. The use of long-term prophylactic anti-convulsants, e.g. for 3 years, is advisable in those who have suffered early epilepsy, major compound fractures with dural and cortical laceration, and intradural compressing hæmatomas.

Post-Traumatic Hydrocephalus.—Late deterioration with apathy and mental retardation may rarely be caused by post-traumatic hydrocephalus requiring the insertion of a ventricular shunt (see p. 470).

Post-Traumatic Headache is not always cerebral in origin. Referred pain of spinal origin resulting from associated strains in upper cervical joints may be referred

through the great occipital and posterior auricular nerves to the vertex, forehead, or temple. This pain is increased by neck flexion, and since the neck is flexed during reading or writing, the pain may appear to be a post-traumatic headache which is made worse by mental concentration. Examination, however, reveals tenderness over the upper interspinous ligaments and great occipital nerves. Many cases respond well to local treatment by injection of local anæsthesia or occipital neurectomy. The post-contusional state, with vertigo, defective memory and concentration, abnormal fatigue, irritability, and defective emotional control, may result from temporary or permanent damage to neurons.

Minor contusions recover speedily, but in serious cases prolonged convalescence is needed. Usually some symptoms of the post-contusional state consisting of headache, giddiness, defective memory, defective concentration, irritability, impaired emotional control, impaired sleep, or susceptibility to alcohol, persist for a period of eighteen months. Post-traumatic dementia constitutes a permanent handicap sometimes necessitating institutional care. Schizophrenia is a rare complication of even a minor injury.

INTRACRANIAL ABSCESS

Intracranial abscess is of three types. (1) Extradural. (2) Subdural. (3) Intracerebral.

Extradural abscess is produced by osteomyelitis of the skull (p. 416 for causes), and is usually secondary to spread from infection from the middle ear or frontal sinus. In the case of the middle ear, infection most commonly reaches the extradural space by extending through the tegmen tympani. Following frontal sinusitis, a large collection may form behind the frontal bone, infection having passed through the posterior wall of the sinus. Spread in the diploic veins may carry the infection to bone areas an inch or more above the ear or frontal sinus and cause local sequestration.

FIG. 443.—Pott's puffy tumour. (*The late Professor Lambert Rogers, F.R.C.S., Cardiff.*)

Acute localised headache, tenderness on local percussion of the skull, and localised pitting œdema of the scalp over the affected area, collectively form Pott's puffy tumour (fig. 443).

Subdural Abscess.—This condition, at one time invariably fatal, can now be treated with a 30 per cent. mortality. It is produced by septic thrombophlebitis of the superior longitudinal sinus, spreading usually from infections of the frontal sinus or accessory air cells. Infection extends from the superior sinus to the superior cerebral veins, and thus infects the subdural space. The abscess extends in this space over the cerebral hemispheres, often bilaterally, and must be treated before it spreads to the inner or under aspects of the hemispheres.

Intracerebral Abscess.—Success in treatment is determined far more by the pathological type of the abscess than by the method of treatment adopted. Some abscesses rapidly spread to infect the ventricle; others localise readily, becoming walled off, and pass into the favourable subacute or chronic stages.

Intracerebral abscesses are produced by (1) implantation of infection; (2) by blood metastasis; and (3) by local extension of adjacent infection.

Percivall Pott, 1714–1788. Surgeon, St. Bartholomew's Hospital London. Described this condition in 1760.

Implantation abscesses result from penetrating wounds and have, as a rule, a low mortality, except in the case of wounds from sticks (p. 443). The infecting organisms are normally derived from the skin surface and are of low virulence. The entry track is closed at the dural level by protrusion of a hernia cerebri, which seals the subarachnoid space and provides a pathway for the extension of the infection to the surface away from the ventricles (fig. 444). There may be progressive extrusion of a hernia cerebri during the acute stage following a penetrated wound, but many abscesses pass insidiously into a chronic stage and merely produce a discharging sinus, or evidence of an intracranial space-occupying lesion at the site of a wound years after injury.

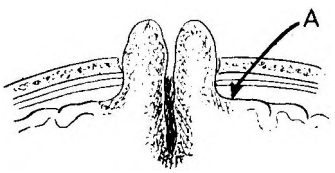


FIG. 444.—Protrusion of a hernia cerebri through a penetration of the dura, closing the subarachnoid space at A.

Metastatic abscess is a complication of intrathoracic sepsis, especially subacute bacterial endocarditis, lung abscess, bronchiectasis and empyema; it may occur in other infective states. Infected clot from the lung passes to the left heart, thence via the carotid, usually into the middle cerebral circulation, which forms a direct continuation of the carotid syphon; hence the infection is implanted deep in the white matter of the parietal or temporal lobe close to the ventricle. The blood supply of the brain is poor in the white matter, but rich in the cortex; tissue reaction is therefore feeble, and the septic encephalitis spreads rapidly and produces fatal septic ventriculitis within a matter of days.

Local extension abscesses are produced by septic thrombosis spreading from foci of infection in the ear or frontal sinuses. Usually three out of five abscesses arising from the ear are situated in the corresponding temporal lobe; the others are in the cerebellum. Frontal lobe abscesses are usually on the same side as the infected frontal sinus.

Very rarely, aberrant abscesses may occur, for example, contralateral frontal abscesses produced by infection crossing the superior sinus, or frontal or parietal abscesses from ear disease. The site of implantation may be (*a*) *subpial*, or (*b*) *subcortical*.

Subpial Abscess.—The infection is conveyed as far as the cortex only. A minute abscess forms, which is covered on its surface only by the pia mater. This invariably ruptures into the subarachnoid space, producing meningitis without focal signs. It is probable that most cases of meningitis complicating ear or sinus disease are produced in this way ; hence the need for prolonged antibiotic and intrathecal therapy to sterilise the subpial abscess and prevent relapse.

Subcortical Abscess.—The infection is conveyed across the unobliterated subarachnoid space to lodge in the white matter beneath the cortex. Three stages may be recognised—acute, subacute, and chronic.

In the acute stage there is septic encephalitis without pus formation. This may extend to produce ventriculitis or localise to form an abscess.

The subacute stage commences at three weeks by the formation of a glial wall, the thickness of which is determined by the local blood supply and is therefore thickest towards the cortex and thinnest towards the ventricle. A unilocular or multilocular cavity is produced, containing active organisms. The wall becomes thick within six weeks and infecting organisms die, staphylococci living the longest. The pus gradually becomes sterile and extremely thick, at which stage the abscess is inert.

Diagnosis and Management

The clinical diagnosis and management of intracranial abscess is a matter of urgency. The results of treatment are directly related to the neurological state and especially to the level of consciousness when neurological management starts. Cerebral abscess is often associated with surrounding brain œdema (fig. 445), which increases the mass effect of the abscess and causes a rapid rise in intracranial pressure. Rupture of an untreated cerebral abscess into the ventricular system may be rapidly fatal. Therefore once the sus-

picion of intracranial abscess arises the patient should be referred for urgent intracranial investigation.

Clinical diagnosis depends upon three aspects:—

1. Evidence of past or present infection, especially in the middle ear, paranasal sinuses or thorax.

2. Focal neurological symptoms and signs.

3. Symptoms and signs of raised intracranial pressure.

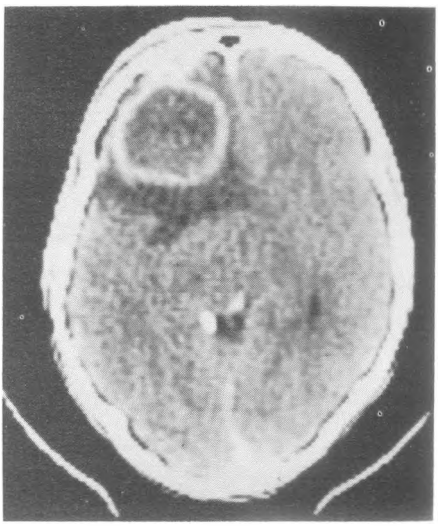


FIG. 445.—CT scan showing frontal abscess and œdema.

1. Evidence of infection.—Chronic suppurative otitis media, an attic perforation and recent mastoidectomy should immediately raise the possibility of intracranial abscess which may be temporal subdural, temporal intracerebral or cerebellar. Chronic frontal sinusitis raises the possibility of frontal subdural or cerebral abscess; even an acute history of upper respiratory tract infection such as a heavy cold without positive radiological evidence of sinusitis, may be relevant.

General features of infection may be few or absent. Thus fever is frequently absent as in tachycardia. The peripheral white blood count, especially with intracerebral abscess, is above 10,000 in only 50% of patients and above 20,000 in only 10%. The ESR (erythrocyte sedimentation rate) is raised (45–50 mm/hour) in the majority of patients.

2. Focal neurological symptoms and signs.—These will depend upon the site of the lesion. *Epilepsy* is a common early symptom in subdural abscess and frontal intracerebral abscess and may be a reflection of the early cortical thrombophlebitic stage, or of an area of focal infarction in which infection develops later. *Dominant temporal lobe abscess* produces dysphasia, and a contralateral hemiparesis, and *cerebellar abscess* nystagmus and incoordination on the side of the lesion.

3. Symptoms and signs of raised intracranial pressure.—As with other intracranial space occupying lesions these are:—

Symptoms	*Signs*
Headache	Depressed conscious level
Deterioration of level of consciousness	Papillœdema
Vomiting	Slowing pulse
Failing vision	Rising blood pressure

The particular features relevant to intracranial abscess are persisting headache, vomiting, deterioration in the level of consciousness and a slowing pulse.

Because the history is often relatively short, failing visual acuity does not occur and *papillœdema is frequently absent.*

Differential diagnosis between abscess and meningitis.—Chronic suppurative otitis media may cause meningitis, or a meningeal reaction leading to headache, drowsiness and neck stiffness. However these signs may also be present with intracranial abscess. *Lumbar puncture in the presence of an abscess is dangerous* because it may precipitate coning and rupture of the abscess. At the same time the early diagnosis of meningitis is essential for satisfactory treatment. Furthermore the CSF findings in cerebral abscess are often unhelpful. In view of the dangers and diagnostic difficulties a guide is:—

If meningitis is suspected *lumbar puncture should not be done* if (*a*), There is evidence of a focal neurological disturbance or (*b*), There are symptoms or signs of raised intracranial pressure.

The patient should be referred for urgent intracranial investigation to exclude an abscess.

Often the symptoms and physical signs of a chronic abscess develop immediately or within a day after operation has been performed on the ear or frontal sinus for the relief of a chronic headache—in these cases operation reveals an abscess with thick walls. Clearly a subacute or chronic abscess was present at the time that the operation was performed, and was itself the cause of the head pain for which the operation had been conducted. The local disturbance of the operation causes a reaction in the abscess cavity, and stimulates the early post-operative onset of physical signs. It is important to realise that the presence of well-marked localising signs developing within a period of days usually indicates re-activation of a chronic abscess requiring immediate treatment.

In every case of suspected cerebral abscess it is important to examine all possible sources of infection. In many cases the probable cause is evident, but discharge from an ear may cease when intracranial complications develop, for hindrance to discharge by inspissated pus predisposes to the extension of infection.

Further Management

Intracranial investigation and localisation.—In the past EEG, ventriculography and carotid arteriography have been very valuable and are still used. Isotope brain scanning provides a useful guide to the presence of supratentorial abscesses. However the investigation of choice today is the CT scan (fig. 445).

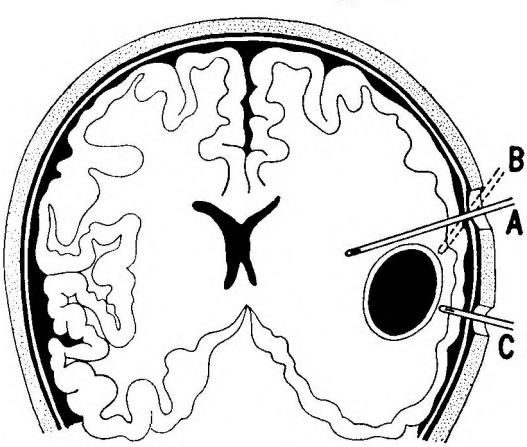


FIG. 446.—Method of localisation of abscess by burr-hole exploration. Needle at A encounters resistance at B. Puncture is performed by a second burr hole over the site of the abscess C.

Surgery.—Surgery is both diagnostic and therapeutic. Drainage, formerly the recognised method of treatment, has been replaced by repeated aspiration of the abscess cavity or by excision. As soon as an abscess has been demonstrated radiologically, a burr hole is placed in the appropriate position, the dura opened and a blunt brain cannula inserted (fig. 446); if an abscess is entered the pus is gently aspirated, an antibiotic inserted (e.g. Penicillin 20,000

units), and, if CT scan is not available, a radio-opaque contrast medium (e.g. Steripaque 1 ml) also inserted.

Further surgical management consists of repeated aspiration through the burr hole, the progress being assessed by the clinical state, and the shrinkage of the abscess as shown by CT scan or the injected contrast medium. Some surgeons favour early excision of the abscess; others only if the abscess fails to shrink.

There are occasions when time does not permit special investigations if the patient is deteriorating rapidly or special investigations are not available. The most critical of situations is that of a patient who has chronic suppurative otitis media, deterioration of level of consciousness and a dilated pupil. A burr hole must be made immediately above the down-turned ear and the temporal lobe needled (fig. 447).

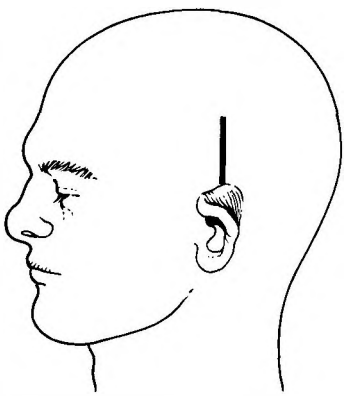


FIG. 447.—Incision for burr hole for temporal lobe abscess.

The surgery of cerebellar abscess is more difficult and although as an emergency measure exploratory needling through a posterior fossa burr hole may be done, it is desirable to excise the abscess at a formal posterior fossa craniectomy.

Antibiotics.—The pus aspirated must be examined immediately for organisms and culture set up. Since streptococci are still the most common organisms to be found, the patient should be started on high doses of penicillin intravenously (up to 24 mega units in 24 hours). Other antibiotics are added in the light of the bacteriological findings. Even when progress is rapid it is wise to combine appropriate antibiotics in smaller doses for six weeks. Prophylactic anti-convulsants should be given for six months.

Treatment of implantation abscess.[1]—In the acute stage the track is explored from the surface, bone and foreign material are removed, and pus is evacuated by repeated aspiration or tube drainage. Antibiotic treatment is given freely. Chronic abscesses may be excised intact.

INTRACRANIAL TUMOURS

Tumours arise in connection with the meninges, nerve sheath, or cerebral substance (gliomas). Tumours of the pituitary gland, vascular malformations, gummas, tuberculomas, blood-clots, and chronic abscess contribute to the total. Secondary carcinoma is far more common than primary intracranial tumour. Secondary deposits are most common from the lung, but may originate from any organ in the body and from the naso-pharynx. When these have been excluded, an average surgical series will be as follows:

Meningioma, 18 per cent.
Neurinoma, 8 per cent.
Glioma, 43 per cent.
Pituitary adenoma, 12 per cent.
Craniopharyngioma, 5 per cent.
Metastatic carcinoma, 6 per cent. (not previously excluded on clinical grounds).
Blood-vessel tumour, 2 per cent.
Granulomas and unclassified and rare tumours.

[1] Pietro de Marchettis, a monk in Padua, treated such cases successfully by the insertion of lampwick drains in 1665.

Meningiomas, 18 per cent., vary in structure and vascularity and include psammomas (calcified meningioma, usually spinal), fibroblastomas, endotheliomas, and angioblastic meningioma. They are usually globular (fig. 448), but occasionally form a carpet, meningioma *en plaque*, which spreads widely in the meninges. Arising from the arachnoid, the tumour gains secondary attachment to the dura, the arteries and veins of which enlarge to provide a tumour circulation (fig. 449). Dilated emissaries between the bone and dura carry the venous return to the veins of the diploe and scalp, and along these veins tumour cells invade the bone, causing bone destruction and reactive hyperostosis (fig. 450).

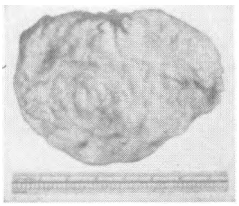


FIG. 448. — Characteristic globular meningioma. (⅓ scale.)

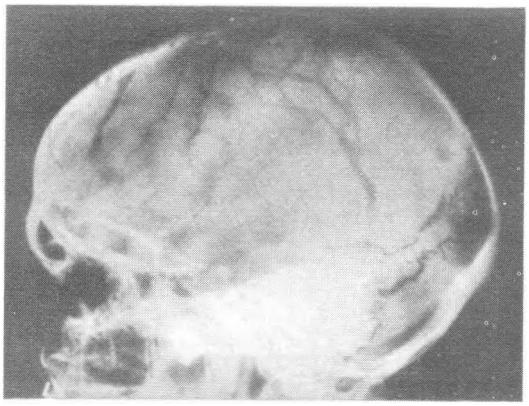


FIG. 449.—Converging vascular markings overlying a meningioma.

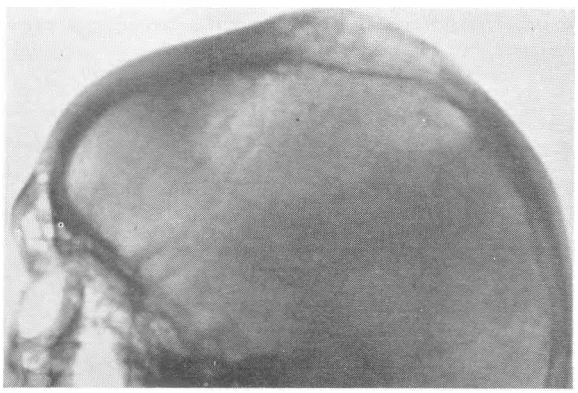


FIG. 450.—A large meningioma hyperostosis extending from the right to left parietal bones. Successfully removed together with underlying tumour. This necessitated a nine-hour operation.

Meningioma occurs in the following situations :

(1) **Parasagittal**—arising from the lateral lacunæ and pressing down on to the upper aspect of the frontal parietal and occipital lobes.

(2) **Fronto-basal**—occurring on the cribriform plate, outer, middle, and inner third of the sphenoid wing and tuberculum sellæ, pressing on the olfactory, oculomotor and optic nerves as well as the brain.

(3) **Posterior fossa**—in the region of the cerebellopontine angle and jugular foramen.

(4) They may also arise from the falx or from the choroid plexus within the ventricle, or may develop from the depth of a sulcus expanding subcortically as intracerebral ' hæmangioblastoma '.

Neurinoma, 8 per cent., is usually found on the sheath of the auditory nerve (eighth nerve tumour), and may be multiple in association with von

Recklinghausen's disease and sometimes occurs in association with multiple cerebral or spinal meningiomas.

Gliomas, 43 per cent.—The malignancy of the tumour varies in proportion to the degree of reversion to the primitive type of cell.

Astrocytoma in its most adult form is composed of star-shaped cells which resemble adult neuroglial tissue. It occurs in three forms: (1) The diffuse or infiltrating, which cannot be totally removed as its margins are unrecognisable and it often affects the brain stem. (2) The solid, relatively circumscribed. (3) The cystic, in which the nodule of tumour secretes fluid from its surface, forming a cystic cleft between itself and compressed normal brain. The tumour projects into the cyst cavity, and removal of this nodule may produce a complete cure. Astrocytoma occurs throughout the cerebral hemispheres, cerebellum and brain stem. However, most astrocytomas contain a variety of cells, the proportion of which varies in different parts of the same tumour. Biopsy from a single favourable point may thus give a totally erroneous impression of a tumour's malignancy. In the modern classification of Kernohan, astrocytic gliomas are classified Grades 1, 2, 3, 4, according to the proportion of adult and primitive cells which they contain. Grade 1 corresponds to the pure astrocytoma. Grade 4 to glioblastoma multiforme (*Bailey* and *Cushing's* Classification). Malignant gliomas affect particularly the middle central territory in male subjects and arise as a result of malignant degeneration in an astrocytoma nodule.

Oligodendro-glioma.—An adult cell tumour consisting of cells with short stunted processes affects the deep portions of the hemispheres in adults. It is prone to undergo central cystic degeneration, and forms a ragged cyst the walls of which consist of tumour tissue. They are often less malignant than other gliomas.

Spongioblastoma polare arises from primitive uni- or bi-polar spongioblasts and affects inaccessible regions, such as the optic chiasma, third ventricle, and hypothalamus in young subjects. It is irremovable, somewhat radio-sensitive, and rarely produces seedling metastases in the cerebrospinal fluid.

Medulloblastoma occurs usually in young children, affecting the vermis of the cerebellum. It grows rapidly and produces seedling metastases throughout the meninges. Microscopically, it resembles a small round-celled sarcoma, and it is highly radio-sensitive.

Ependymoma may occur throughout the cerebral hemispheres and in the fourth ventricle. The cells resemble the ependymal cells lining the ventricles. In the cerebral hemispheres they vary in malignancy and tend to behave like the malignant astrocytomas. In the fourth ventricle they are less malignant.

Clinical Features.—All tumours have an initial silent period which varies in length according to position and rate of growth. If the tumour is not near any area which will produce symptoms or signs, it will take up space provided in the subarachnoid cisterns; it will flatten and displace the

CLINICAL COURSE OF CEREBRAL TUMOUR

Stage 1	Stage 2	Stage 3	Stage 4
Initial period of silent growth.	Focal syndromes. Epilepsy.	Raised intracranial pressure.	Brain displacement. False localising signs. Cone formation.

ventricle and brain until it can gain no more room, and it will then produce symptoms of raised intracranial pressure, such as morning headache, effortless vomiting, and papilloedema. It may be several years or more before this occurs in the case of a meningioma. If the tumour is situated in an

Friedrich Daniel von Recklinghausen, 1833–1910. Professor of Pathology, Strasbourg.
Percival Bailey, 1892–1973. Professor of Neurology, Chicago, Ill., U.S.A.
Harvey Cushing, 1869–1939. Professor of Surgery at Johns Hopkins and Harvard University.

important area, it may, by its local effect, produce symptoms of epilepsy or progressive neurological syndromes before any evidence of intracranial pressure is produced. Hence the absence of headache, vomiting, and papillœdema does not exclude a tumour.

Epilepsy arising for the first time in adult life should always be suspected as being due to a tumour until this possibility has been disproved (fig. 451).

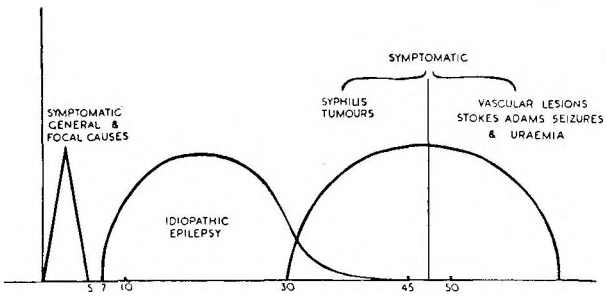


FIG. 451.—The principal causes of epilepsy at different ages.

Idiopathic epilepsy does not occur before the age of six. Ninety per cent. of cases of idiopathic epilepsy have their first seizure before the age of thirty. After the age of thirty, epilepsy is usually symptomatic, and in patients between the ages of thirty and fifty tumour is a common cause.

Progressive focal syndromes should likewise be regarded as indicating a tumour until this possibility is disproved. Degenerative conditions such as disseminated sclerosis are characterised by periods of remission and exacerbation. Vascular lesions occur instantaneously and are followed by some degree of improvement. If repeated on several occasions, as in multiple emboli, an appearance of steady progression is produced, but there is only one condition that will produce a steadily progressive syndrome, and that is a tumour.

The stage of raised intracranial pressure can develop in association with focal symptoms or be the sole evidence of tumour formation. Pressure symptoms occur late in the case of tumours in the frontal lobes which push the ventricles back. They occur earlier in tumours in the temporal and parietal lobes which partially obstruct the outflow of fluid from the adjacent ventricle. They occur earliest in midline and posterior fossa tumours which obstruct the flow of fluid from both ventricles, producing an internal hydrocephalus. Tumours of these types add to their own bulk the effect of the retained ventricular fluid. The effects of pressure include headache, vomiting and papillœdema, bradycardia, and retarded cerebration. The headache often wakes the patient in the early morning, and is aggravated by coughing and straining, which promote cerebral congestion. Vomiting occurs without warning, and is not preceded by nausea or related to food. Papillœdema may cause blindness.

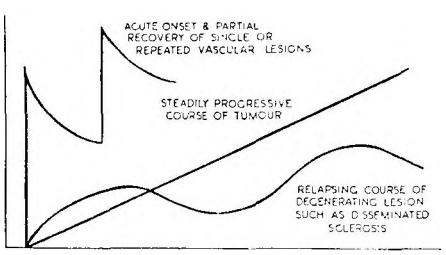

ACUTE ONSET & PARTIAL RECOVERY OF SINGLE OR REPEATED VASCULAR LESIONS
STEADILY PROGRESSIVE COURSE OF TUMOUR
RELAPSING COURSE OF DEGENERATING LESION SUCH AS DISSEMINATED SCLEROSIS

FIG. 452.—Clinical courses of vascular, neoplastic and degenerative lesions.

The Stage of Cone Formation.—Certain patients do not present until cone formation is imminent. When intracranial pressure becomes high, the inner border of one hemisphere may be forced under the falx plugging the superior longitudinal fissure and blocking the pathway for the cerebrospinal fluid absorption. The temporal lobe may be forced down from above into the tentorial opening. The cerebellar vermis may be pushed up into this opening or the cerebellar tonsils may be forced down into the foramen magnum. Ominous signs of a threatened cone include violent paroxysmal headache, drowsiness, slow cerebration, slow pulse, and neck stiffness. Unilateral pupillary dilatation is an urgent sign. *Lumbar puncture must be rigidly avoided at this stage.* (See fig. 413).

Frontal lobe tumours if deeply situated produce a progressive change, in personality, with lack of insight, neglect of normal pastimes, occupations, and duties, and an alteration in emotional reaction particularly noticeable to relatives, consisting usually of a simplicity and unwarrantable cheerfulness (euphoria) or irritability. Epilepsy is generalised in type, and localising signs are limited to contralateral facial weakness.

Parietal lobe tumours produce Jacksonian epilepsy and progressive hemiparesis. Examination reveals loss of touch analysis resulting in astereognosis. Deeply situated left-sided tumours may exhibit defects of spatial relationship and loss of power of calculation (acalculia).

Occipital lobe tumours.—Epilepsy is generalised in type, preceded by an aura of flashing lights in the contralateral visual field. Signs, other than homonymous hemianopia, are few.

Temporal lobe tumours on the left side may produce progressive aphasia or visual or auditory hallucination sometimes with generalised convulsions. Lesions of the uncinate region produce uncinate hallucination of smell and taste and dreamy states of unreality. Right-sided lesions may cause similar uncinate attacks of generalised epilepsy without aura. Localising signs include hemiparesis and superior quadrantic hemianopia.

Parasagittal meningioma overlying the various lobes produces similar syndromes but with characteristic skull X-ray changes (figs. 449 and 450).

Basal meningioma produces involvement of the olfactory and optic, oculomotor, and trigeminal nerves, in addition to pressure on the brain. Bone changes in the region of the orbit may cause proptosis or fullness in the temporal fossa.

Midline tumours always produce bilateral internal hydrocephalus with pressure symptoms.

Tumours of the third ventricle.—Colloid cysts at first cause a ball-valve obstruction producing intermittent blockage of the foramen of Monro. Severe unilateral headache is produced by lying on one side of the head and relieved by turning to the other side. Later, persistent bilateral hydrocephalus is accompanied by severe pressure symptoms or progressive cerebral atrophy and dementia. Glioma of the floor of the ventricle causes hydrocephalus associated with endocrine disturbances, disturbances of sleep rhythm, and sexual precocity.

Pineal tumours by pressure on the quadrigeminal plate produce hydrocephalus with paralysis of the oculomotor nuclei with loss of upward, lateral, and downward movement of the eye.

Subtentorial Tumours.—Tumours of the posterior fossa distort the aqueduct and fourth ventricle, and by obstructing the escape of CSF, produce raised intracranial pressure. The effect of the hydrocephalus varies with the age of the patient.

Cerebellar vermis tumours are usually medulloblastomas and occur in young children before the sutures have united. The hydrocephalus causes progressive enlargement of the head. At a later age, when the sutures have united, there are serious pressure symptoms and stiffness of the neck from herniation of both cerebellar

Alexander Monro, 1733–1817. Professor of Anatomy, Edinburgh.

tonsils into the foramen magnum. Since the vermis controls the co-ordination of trunk and legs, localising signs are only seen if the child is taken out of bed; it will then be seen to walk with the feet wide apart and have a tendency to fall over forwards when standing with the feet together.

Cerebellar hemisphere tumours are often astrocytomas. They produce hydrocephalus and pressure symptoms in older subjects. Since the hemisphere controls the co-ordination of the corresponding side of the body, there is deviation to the affected side on walking and unilateral inco-ordination of the arm. Nystagmus may or may not be present.

Acoustic neuroma grows from the auditory nerve at the internal auditory meatus and produces enlargement of this structure, visible on X-ray. Arising from the eighth nerve the first symptom is unilateral deafness, which is often first detected by the patient on the telephone. The tumour projects into the cerebello-pontine angle and presses upon the adjacent seventh, sixth, and fifth nerves, causing a syndrome of unilateral deafness, facial weakness, and sometimes squint. The corneal reflex is reduced from pressure on the fifth nerve. There may be trigeminal neuralgia from this cause or trigeminal anæsthesia. Later the tumour presses upon the cerebellum and brain stem, producing cerebellar signs and raised pressure. Finally it grows up into the tentorial opening from below, and here it not only blocks the up-flow of cerebrospinal fluid but assumes a position anterior to the brain stem coming into close relationship with the basilar artery and twisting the pons. Cerebrospinal fluid protein is always high, often over 200 mg/100 ml (2·0 g/l).

The investigation of cerebral tumour must define the site and nature of the tumour.

History taking.—The history alone will sometimes indicate the site of the tumour and also give a hint as to its pathological type. If symptoms have been long-standing, they suggest a slowly growing and favourable tumour. A short history may, however, be due to the final breakdown of adaptation in a slowly growing tumour or indicate a rapid malignant growth.

The history may suggest a primary disease to which the cerebral condition is secondary. Since metastatic carcinoma, especially bronchial, is far more common than primary cerebral tumour, particular attention must be paid to history of loss of weight, recent cough, or hæmoptysis. Secondary brain abscess is suggested by a history of lung abscess or bronchiectasis; chronic otitic abscess by symptoms of cachexia and a discharging ear. Weight loss is always suspicious, as there is no wasting with primary cerebral tumour.

FIG. 453.—Separation of sutures, beaten silver appearance, and erosion of posterior clinoids.

Clinical examination must include the general examination in search of primary disease.

Neurological examination of cranial nerves and nerve tracts may localise *where* a tumour is, but never indicates what it is.

Accessory investigations are essential. X-ray of the skull, X-ray of the chest, and the erythrocyte sedimentation rate must be taken in every case. A high E.S.R. is strongly suggestive of secondary tumour or abscess.

An X-ray of the chest may reveal an unsuspected bronchial carcinoma. Thirty per cent. of bronchial carcinomas present with cerebral symptoms before any chest symptoms have occurred.

An X-ray of the skull may show that a tumour is present :

(1) By pressure changes including : (*a*) A beaten silver appearance of the vault from the pressure of tight convolutions (sometimes normal in thin skulls). (*b*) Separation of sutures in young subjects. (*c*) *Most important*—erosion of the posterior clinoid processes, a very valuable sign which indicates long-standing pressure and therefore a possibly removable tumour (fig. 453).

(2) By lateral displacement of a calcified pineal shadow indicating the side of the tumour.

(3) By characteristic intracranial calcification produced by astrocytomas, angiomas, 50 per cent. of craniopharyngiomas, and some meningiomas and tuberculomas (fig. 454).

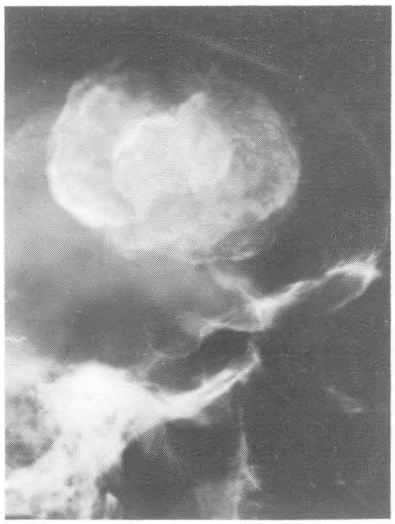


Fig. 454.—Calcified meningioma

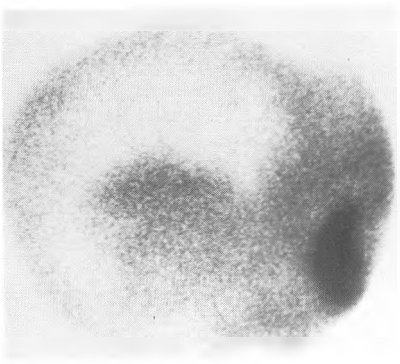


FIG. 455.—Isotope scan showing large sphenoidal meningioma.

(4) By alteration in skull vascular markings in meningioma, including converging diploic channels, and an increase in the size of the meningeal groove (fig. 449).

(5) By changes in the skull bones, including meningioma hyperostosis, local expansion at the site of a cyst, and evidence of bone destruction in secondary tumours.

Electroencephalogram.—Characteristic wave-forms indicate the site or presence of focal or deep-seated tumours, and distinguish between epileptic seizures produced by focal lesions and idiopathic epilepsy.

Isotope scan is a valuable method of investigation for supratentorial tumours (fig. 455).

CT scanning (see p. 435), when available, provides reliable information concerning the presence of a tumour, and ventricular displacement or distension. It is possible to distinguish the type of tumour. Meningiomas appear densely white. Gliomas are less dense, any central necrosis shows as a dark area within the glioma shadow. Astrocytoma secretion cysts show a dark shadow with the lateral mural nodule indenting. It is possible to distinguish the vascular edge of malignant gliomas. Secondary tumours appear as multiple shadows or cysts. Intravenous contrast infusion enhances the reliability of scan interpretation (figs. 456, 457, 458).

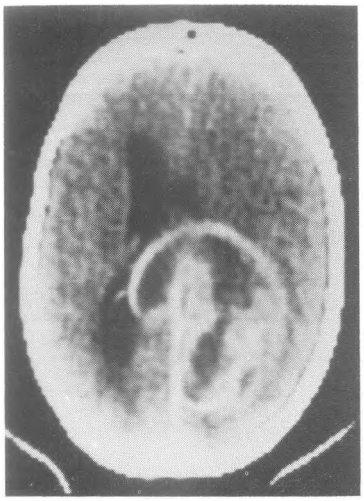


FIG. 456.—CT scan showing necrotic malignant glioma.

Surgical investigation by arteriography or air study is employed to localise a silent tumour producing pressure but no physical signs, or indicate the extent of a known tumour, and to provide evidence of its type.

Arteriography was originated by Egas Moniz. In the absence of CT and Isotope scan it remains a valuable method of investigation of supratentorial tumours. The site of the tumour is shown by the displacement of cerebral vessels, e.g. upward or downward deviation of the middle cerebrals or displacement of the anterior and middle cerebrals in the antero-

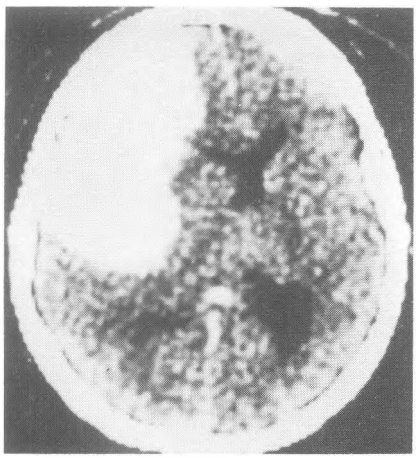


FIG. 457.—CT scan showing sphenoidal meningioma.

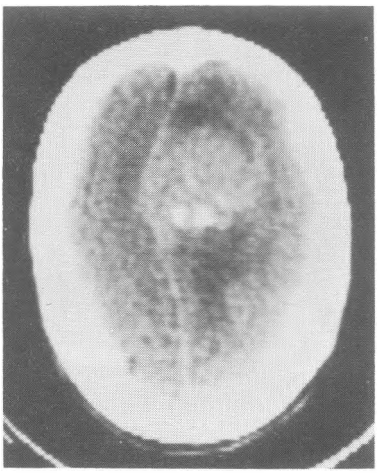
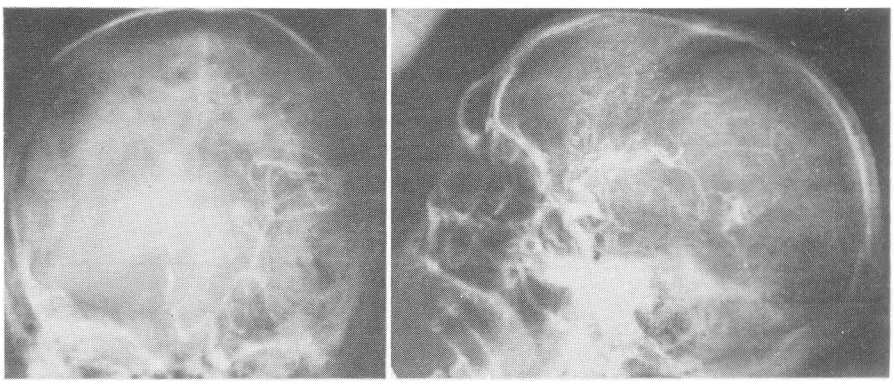


FIG. 458.—CT scan showing parasaggital meningioma with surrounding brain œdema.

posterior view. Pathological tumour circulation is seen in meningioma and glioblastoma multiforme. Meningiomas produce a diffuse blush; glioblastomas produce a group of very primitive and imperfect vessels (fig. 459).

Investigation by Air Study.—Ventriculography was originated in 1918. Bilateral parietal burr holes are formed under local anæsthesia. A hollow ventricular cannula is inserted to a depth of up to 5 cm to puncture the vestibule of the ventricle (fig. 460). Available cerebrospinal fluid is removed and replaced with 10 per cent less volume of air. Since air rises and fluid falls the patient is positioned in such a

Antonio Cætano de Abreu Freire Egas Moniz, 1874–1955. Professor of Neurology, Lisbon. Introduced cerebral arteriography in 1927 and leucotomy in 1936.



A *B*

FIG. 459.

(*A*) A.P. arteriogram showing inward displacement of the anterior and middle cerebral arteries and tumour circulation causing a blush in a meningioma at the outer third of the sphenoid ridge. (*B*) Lateral arteriogram showing primitive glioblastoma vessels in posterior temporal region.

way that the portion of the ventricular system which it is wished to fill with air is brought to the highest level during arteriography, films being taken in various planes to show displacement and distortion of the ventricle. Investigation is dangerous as tapping of a displaced and expanded ventricle on the side opposite to the tumour may allow the brain to be pushed over causing cone formation as the temporal lobe sinks into the tentorial opening. Arteriography is therefore preferable. However *ventriculography* remains a valuable method of investigation for posterior fossa and third ventricle tumours when the lateral ventricles are dilated and there is no lateral displacement. In an emergency situation it can be performed with simple radiographic equipment.

Myodil ventriculogram in hydrocephalus will demonstrate the third and fourth ventricles and aqueduct after the injection of 3 ml. of Myodil.

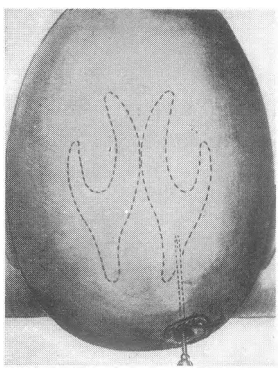


FIG. 460.—Diagram of ventriculography.

Further Management and Surgery

Having established on clinical grounds that there is evidence of raised intracranial pressure (see Abscess, p. 448), and having demonstrated radiologically the presence of a space occupying lesion the objects of further management are:—

1. The relief of raised intracranial pressure.
2. Establishment of a pathological diagnosis.
3. Removal of benign tumours.
4. Operative and non-operative treatment of malignant tumours.

1. Relief of raised intracranial pressure.—Raised intracranial pressure may be responsible for distressing symptoms (e.g. severe headache, vomiting, failing visual acuity) and if pressure is not relieved the outcome is soon fatal. The degree of urgency of action depends upon the severity of the symptoms and signs of pressure, the most important being severe headache and deterioration in level of consciousness. Papillœdema is present in only 30%

of patients with intracranial space occupying lesions and therefore absence of papillœdema never excludes such lesions. Although CT scanning has improved pre-operative pathological diagnosis, in most patients this cannot be established without some form of surgery and therefore relief of pressure is essential unless a malignant (and untreatable) pathology has been proven.

Pressure may be relieved by:—

(*a*) Ventricular tap and drainage through a posterior-parietal burr hole when the lateral ventricles are symmetrically dilated as in posterior fossa and third ventricle tumours.

(*b*) Tapping of supratentorial cystic tumours and abscesses. If the lesion is solid needling will not lower pressure and may raise it further by provoking hæmorrhage and œdema.

(*c*) Administration of Mannitol (p. 437) which may produce *temporary* improvement.

(*d*) Urgent removal or partial removal of supratentorial tumours, so creating an ' internal decompression '.

(*e*) Dexamethasone in doses initially of up to 24 mg a day may bring relief of raised pressure and even of focal neurological deficit in patients with malignant tumours.

It is of little value in meningiomas but is most valuable in necrotic malignant gliomas and necrotic secondary tumours. In these the reduction of œdema may restore a comatose patient to consciousness and remove signs of paralysis and other localising phenomena. Repeated administration may prolong life. In frontal lobe tumours the relief of paralysis by dexamethazone will indicate that the paralysis is merely due to œdema and not to direct invasion of the motor areas by tumour tissue, providing an indication for removal of the tumour by frontal lobectomy. Regular administration of dexamethazone is also useful during X-ray therapy by countering the increased œdema occurring during treatment. If dexamethazone is given to reduce œdema around a tumour which is still present and if the treatment is stopped, sudden development of œdema may cause death from pressure cone in 24 hours.

2. Establishment of a pathological diagnosis.—This is essential if further decisions about tumour removal are to be taken. Tissue for histology is obtained either by burr hole biopsy or by craniotomy and tumour removal, the latter achieving relief of intracranial pressure at the same time. Craniotomy is therefore safer. The choice of method depends upon many factors which include accessibility of the tumour, the likelihood of abscess, the anticipated quality of survival and the age of the patient. Whatever method is used it is essential to have facilities for immediate histological examination by frozen section.

3. Removal of benign tumours (see meningioma, p. 452).

4. Treatment of malignant tumours.—Much depends upon the histology and degree of malignancy of the tumour. For astrocytoma of grade 3 and 4 malignancy the outlook remains poor however extensive and ' complete ' the removal may be, survival being between 3 and 18 months; an average radiotherapy improves survival by about 6 months. The results of chemotherapy are as yet inconclusive. Medulloblastoma responds more favourably to radiotherapy and chemotherapy.

Burr Hole Biopsy.—If investigation suggests the presence of an unfavourable lesion in which craniotomy is contraindicated, such as secondary carcinoma or malignant glioma, or if a cyst is suspected, a burr hole is fashioned over the tumour site and the tumour is aspirated by ventricular cannula. If a cyst is defined, air is injected into the cavity in order to produce a picture of the cyst.

A favourable cyst produced by astrocytoma or hæmangioblastoma contains golden-yellow clotting fluid.

A malignant cyst contains muddy non-clotting fluid like stale beer.

Astrocytic glioma Grade 3 and 4 renders the brain soft, necrotic, and highly vascular whereas meningiomas are resistant.

If stained smears show the presence of astrocytic glioma Grade 3, lobectomy can provide a useful decompression prior to X-ray therapy in favourable situations. Open decompression of the dura however is contraindicated as the tumour contains

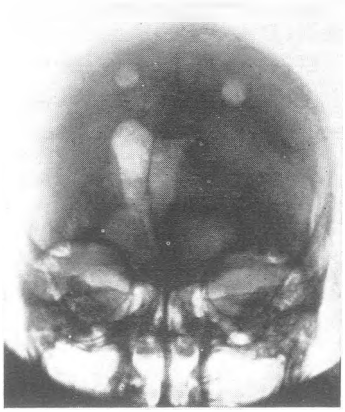


FIG. 461.—X-ray indicating dangers of ventriculography. Both ventricles are already on one side of the head. This is not unusual.

FIG. 462. — Antero-posterior ventriculogram showing flattening and downward displacement of left lateral ventricle by large parasaggital meningioma. The middle meningeal groove is enlarged on the left side.

primitive vessels and will enlarge owing to acute œdema if the pressure of the dura is relaxed. This 'acute brain swelling' will cause additional symptoms of aphasia and hemiplegia. It may be possible to secure internal decompression by sucking out the necrotic centre of a tumour through a small dural opening prior to deep X-rays. Astrocytoma Grade 3/4 can be usefully palliated by internal decompression or lobectomy followed by radiotherapy.

Operation must not be delayed if pressure is high as sudden and unexpected collapse may occur. Improvement in neurosurgical technique permits a radical excision in many cases.

Craniotomy.—An osteoplastic flap is fashioned over the site of the tumour and must be sufficiently large to expose either the lobe to be excised or an adequate margin of normal brain around the tumour which is to be enucleated. In the case of meningiomas a length of the superior longitudinal sinus greater than the known length of the tumour must be displayed. Various scalp incisions are employed, transverse or horseshoe, all of which are hidden as far as possible within the hair line.

The field of operation is infiltrated with saline. The skin incision is outlined, the area of operation is sealed off by lint trephine cloth, which is gummed to the scalp with

gum mastiche and cut through in the line of the proposed incision. The scalp is incised. The assistant and surgeon exert digital pressure with the fingers on either side of the cut. Before pressure is released, artery forceps are applied to the epicranial aponeurosis on either side; these are folded back so that the vessels in the scalp are bent on themselves, and each bunch of four or five forceps is secured with a rubber ring. By degrees the incision is enlarged and an extensive exposure can be obtained, almost bloodlessly, using a sufficient number of forceps. The scalp flap is now reflected and wrapped in a warm swab. Skin towels are applied to the wound edge. Burr-hole openings are then fashioned in a suitable pattern, being placed at the summit of each convexity in order to reduce pressure on the dura when the saw guides are passed from hole to hole. Gigli guides and saws are now passed between the burr holes, and the intervening areas of bone are sawn through, the cut being fashioned obliquely, so as to form a mortise edge in the bone in order to prevent the bone flap sinking in when it is replaced. The base of the flap is designed to hinge on the temporal muscle. The bone flap is now elevated and the base fractured, the osteoplastic flap then being turned down on the hinge of temporal muscle and wrapped in a warm-cloth. If a meningioma is being dealt with, the bone flap is extremely vascular, and on lifting the flap, hæmorrhage occurs from the torn emissary veins crossing between the bone and dura. The exposed dura matter is now inspected for an excess of vessels and torn emissary veins, indicating an underlying meningioma, or for surface evidence of underlying glioma, such as discoloration of the brain or widening of convolutions visible through the dura.

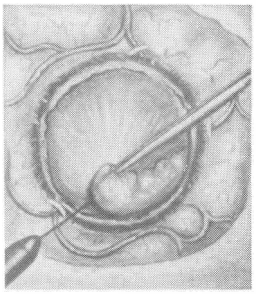


FIG. 463. — Operative sketch of removal of tumour.

To delineate the extent of a subcortical tumour, ventricular needles are inserted at three or four points in order to detect the resistance of a tumour margin. When the position and nature of the growth have been decided, the dura is opened by a horseshoe flap, either with guarded scissors or a knife cutting on to a dural guide. Meningeal vessels of large size are secured with Cushing's silver clips.

Excision of a Meningioma.—Parasagittal.—The dural incision is completed all round the base of the tumour unless this invades the superior longitudinal sinus. The margin between the brain and tumour is defined, and vessels crossing this are secured with diathermy or silver clips; the tumour is then rotated upwards from its bed and dissected from the sinus.

Basal Meningioma. — Occasionally it is necessary to amputate the frontal lobe of the brain to expose tumours on the middle or inner third of the sphenoid ridge, otherwise the lobe of the brain is retracted and the bulk of the tumour is reduced by cutting out its centre with punch forceps and diathermy, following which the capsule is gently drawn away from important branches of the circle of Willis which surround it. Finally, the collapsed capsule is excised intact, together with the adjacent dura (fig. 464).

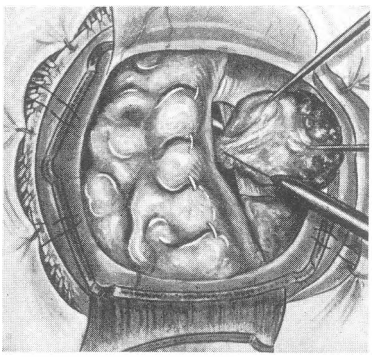


FIG. 464. — Excision of collapsed capsule of a basal meningioma exposed by frontal lobectomy.

Excision of a Glioma.—(*a*) *By Enucleation.*—The tumour nodule is exposed either by opening a cyst or by 'uncapping' the area of overlying cortex. A medallion of cortex of adequate area is defined. Vessels entering this zone are secured with diathermy or silver clips, the defined area being then removed down to the level of the tumour margin. Vessels crossing from brain to tumour are gradually secured and the nodule is rotated from its bed.

(*b*) *By Lobectomy.*—Veins passing from the frontal or occipital lobe to the superior sinus are coagulated on a blunt hook, or secured with silver clips, and divided. Veins passing from the temporal lobe to the sphenoidal or petrosal sinuses are likewise

Thomas Willis, 1621–1675. Professor of Natural Philosophy, Oxford. Afterwards practised as physician in St. Martin's Lane, London. The drawings of the circle of Willis were made by Sir Christopher Wren.

secured. The plane of section is now defined, and vessels crossing this are secured with silver clips or diathermy. The incision is now deepened across the white matter to the opposite aspects of the lobe, where vessels are similarly dealt with and the lobe is excised.

If a tumour is irremovable, central necrotic areas are removed by suction in order to reduce pressure. In all cases hæmostasis must be complete before the wound is closed. The dura is sutured completely, and if necessary with the introduction of a fascial graft. If bone has been invaded by meningioma hyperostosis, it is excised. Finally, the scalp is sutured in two layers. Blood transfusion is required during operation in vascular cases.

POSTERIOR FOSSA CRANIECTOMY

Intratracheal anæsthesia is essential. The head is fully flexed and supported with the patient in the sitting-up position, which reduces venous congestion. A fine catheter is inserted into the lateral ventricle through a posterior parietal burr hole to provide ventricular drainage and reduce intracranial pressure. A midline incision is formed from above the external occipital protuberances to the level of the third cervical spinous process. The lateral skin flaps are raised from the occipital muscles, retractors are inserted and the ligamentum nuchæ is divided with diathermy to expose the arch of the first cervical vertebra. The occipital muscles are cut through 6 mm below their attachment and dissected with diathermy from both occipital plates. The arch of the atlas is removed. Both occipital plates are drilled through and then removed with biting forceps including the rim of the foramen magnum. This exposes the posterior fossa dura as high as the transverse sinus, the foramen magnum, and upper cervical dura. The dura is palpated, and if the pressure is high, it is reduced by releasing fluid from the ventricles or tapping the cisterna magna. The dura is incised transversely, the occipital sinus being secured with Cushing's silver clips, and a vertical incision is now made downwards in the midline to the upper cervical region to release the pressure cone at the foramen magnum. If both tonsils are forced down, the tumour will be found in the midline. If one tonsil is down, the tumour is in the corresponding hemisphere. Astrocytoma nodules are excised, but medulloblastomas are best left intact with an open decompression for deep X-ray treatment.

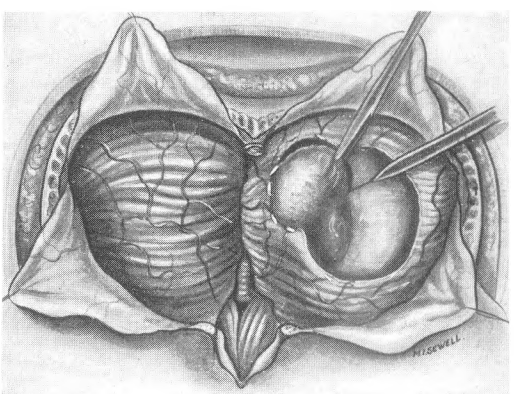


FIG. 465.—Removal of astrocytoma nodule of cerebellum.

Unilateral exposure may be performed through a hook incision extending up from the third cervical spine to the external protuberance and then laterally and finally downwards to the mastoid tip. The ligamentum nuchæ is split to expose the arch of the first cervical vertebra, and the occipital muscle is reflected downwards from the occipital plate, which is then removed. The cisterna magna is tapped to reduce pressure and the dura is opened.

Eighth nerve tumours are exposed by this route. The lower third of the cerebellar hemisphere is cut away and the tumour exposed anterior to the cerebellum. The capsule is incised, its contents are evacuated, and the capsule is gently drawn away from the side of the brain stem and downwards through the tentorial opening before being excised. A small portion is left over the facial nerve in order to prevent facial paralysis.

Large tumours are dangerous as they extend into the tentorial opening anterior to the brain stem, distorting and compressing the basilar branches. A fatal brain stem thrombosis may occur as a postoperative complication. Good results and

smooth postoperative recovery are claimed for the method of lateral extradural approach since retraction of the cerebellum is avoided.

TUMOURS OF THE PITUITARY BODY

Comprise three varieties of adenoma; chromophobe, acidophil, and basophil, and congenital tumours derived from Rathke's pouch (craniopharyngiomas).

Chromophobe adenomas occur most commonly in female patients between the ages of twenty and fifty. The tumour is solid and slow-growing and expands steadily to form a mass the size of a walnut in a period of several years. Spontaneous involution may occur with cessation of growth and cystic change (cf. adenoma of thyroid). Occasionally highly cellular rapidly growing tumours assume the characteristics of local malignancy invading laterally into the cavernous sinus or extending into the lobes of the brain. Three stages of development may be recognised:

(*a*) The stage of intrasellar development.

(*b*) The stage of extrasellar extension.

(*c*) The stage of massive intracranial extension.

(*a*) *Intrasellar Development.*—In the sella turcica, the expanding chromophobe cells, which possess no active secretion of their own, compress the acidophil and basophil cells of the pars anterior and inhibit their secretions which are concerned with growth and sex functions. Since the tumour arises at an age when growth is complete, the effect is felt by the thyrotropic and gonadotropic hormones. The patient becomes fat and sluggish with a lowered metabolic rate, as in myxœdema, and amenorrhœa is invariable. Pressure on the diaphragma sellæ causes severe headache. X-ray of the skull reveals pressure changes of the sella (fig. 466). The floor is depressed towards the sphenoidal air sinus, lateral pressure on the petroclinoid ligaments leads to separation of the anterior clinoid processes, and the posterior clinoids become thin.

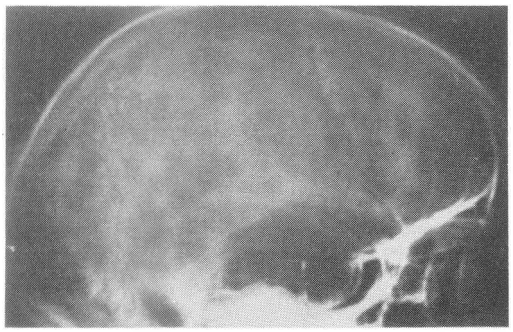


FIG. 466.—Typical X-ray appearances of enlarged sella in chromophobe adenoma.

(*b*) *The Stage of Extrasellar Extension.*—The tumour breaks through the diaphragm at a weak point between the anterior clinoid processes and extends up in front of the pituitary stalk to press on the under aspect of the optic chiasma. Headache becomes less severe from relief of intrasellar pressure, amenorrhœa persists, and gradually bitemporal hemianopia is produced by stretching of the decussating fibres in the optic chiasma over the posterior border of the tumour. Involvement of these fibres which come from the inner half of each retina, leads to loss of vision towards each temporal side. Stretching of the optic nerves anterior to the chiasma produces primary optic atrophy

Martin Heinrich Rathke, 1793–1860. Professor of Zoology and Anatomy, Königsberg, which was in Germany, but since 1945 is Kaliningrad, U.S.S.R.

with pale white optic discs. The diagnosis is usually made at this stage and confirmed by C.T. scan or lumbar air encephalogram.

Acute infarction may rarely produce a sudden swelling of the tumour with rapidly increasing visual loss. The risk of infarction occurring after deep X-ray therapy or the implantation of radioactive Yttrium contraindicates the use of either method when the chiasm is compressed. Infarction may cause complete blindness and then be followed by atrophy and disappearance of the tumour.

(*c*) *The stage of massive intracranial extension* is rare. If the optic chiasma is in an abnormal position, either pre- or post-fixed, the tumour may slip by it without causing visual disturbance and enter the frontal lobe, or highly cellular rapidly growing tumours may spread laterally into the temporal lobe or upwards and backwards behind the chiasma into the third ventricle. These extensions will act as a space-occupying tumour and produce symptoms of raised pressure or epilepsy. Extension into the temporal lobe produces an additional homonymous hemianopia from involvement of the optic radiation.

Differential Diagnosis.—The syndrome may be simulated by a meningioma of the dorsum sellæ or an aneurysm of the anterior communicating or internal carotid artery. Arteriography is required in doubtful cases.

Treatment.—Few chromophobe tumours are radio-sensitive. Many alleged successes attributed to deep X-ray have been due to spontaneous cystic involution or an abnormality of the chiasma which allows the tumour to go on growing without serious visual loss developing. Years later the growth is found to have formed a massive intracranial extension.

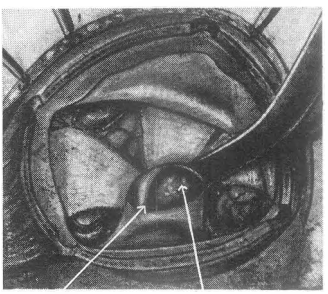


FIG. 467.—Intradural exposure of pituitary tumour, showing (*a*) optic nerve and (*b*) pituitary tumour.

Operation performed before the stage of massive extension has a mortality of 2 to 4 per cent.; after extension 30 per cent., hence early operation is advisable. Pre- and post-operative treatment by steroids is advisable in the presence of hypopituitrism in order to prevent post-operative adrenal deficiency.

The tumour is exposed by frontal osteoplastic craniotomy, after opening the dura and elevating the frontal lobe. The extradural approach dividing the dura at the sphenoid wing gives less adequate exposure. The tumour looks like a dark plum stretching the optic nerve like a white ribbon around its margin. Cysts are opened or solid tumour is removed by punch forceps. The capsule of the tumour is then drawn down from behind the chiasma and excised. Post-operative deep X-ray is given, and recurrence of these tumours is very rare.

When the extrasellar extension of the tumour is small and strictly in the midline the trans-sphenoidal route is now favoured by many surgeons. With severe chiasmal compression, when a solid lesion has been shown by CT scan or the diagnosis is in doubt the transfrontal approach is advisable.

Acidophil adenomas are small in size and rarely cause pressure on the optic chiasma except in cases of fugitive acromegaly, in which the tumour reverts almost to a chromophobe type. Normally the symptoms are due to

excessive production of growth hormone by the acidophil cells and inhibition of the basophile sex secretion.

In children the tumour causes gigantism, while patients whose epiphyses have united develop acromegaly (a term which implies enlargement of the extremities).

Acromegaly is characterised by thickening of the subcutaneous tissues of the scalp, lips, and tongue, the face, hands and feet, and overgrowth of the frontal sinuses, jaw, and distal phalanges. There is also overgrowth of hair and sebaceous glands. Asthenia causes slackening of ligaments with kyphosis, so that the enlarged hands hang below the knees. This combined with the atavistic appearance produced by the beetling brow, prognathous jaw, and overgrowth of hair on the chest, produces the 'ape man' of the circus. Fatty degeneration of the heart and herniæ are associated conditions.

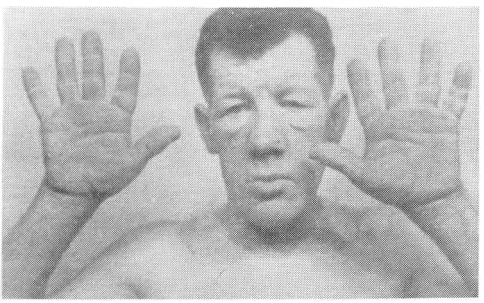


FIG. 468.—Acromegaly.

Treatment.—There is still uncertainty about the ideal treatment of acromegaly. Methods used have included bromocriptine, external radiotherapy, implantation of radio-active Yttrium, transfrontal surgery and trans-spenoidal surgery. Currently the trans-sphenoidal route with micro-surgical removal of the adenoma is the method of choice.

BASOPHIL ADENOMAS AND CUSHING'S SYNDROME

Basophil adenomas are small, usually only a few millimetres across; their effects are produced by secretion of adreno-corticotrophic hormones leading to excessive production of hydrocortisone.

Cushing described a syndrome associated with basophil adenomas, although a similar condition may be due to adrenal dysfunction (adreno-genital syndrome, p. 652). Females are more commonly affected. Fat accumulates on the trunk, neck, and face which becomes red and moonshaped but the limbs remain normal. Purple striæ appear in considerable numbers on the skin, which is thin and atrophic. Hirsutism, arterial hypertension and glycosuria develop early. There is moderate polycythæmia and atrophic changes affect the bones of the os calcis and pubis. A psychotic state of schizophrenia sometimes develops.

Treatment is directed to reduction of the secretion of hydrocortisone either by adrenalectomy or by operation on the pituitary. Russell Fraser has shown that these tumours may be effectively treated by the intrasellar implantation of radio-active Yttrium 90.

Craniopharyngioma.—Remnants of Rathke's pouch may persist at four sites: (1) In the sphenoid bone; (2) in the sella turcica; (3) in the pars nervosa; (4) in the floor of the third ventricle, from all of which tumours may develop. Although congenital in origin, a proportion of cases do not produce symptoms until late in adult life.

The term craniopharyngioma describes these tumours best. In structure, the growths form large masses in which cystic cavities lined with ciliated epithelium and containing cholesterol crystals are separated by areas of connective tissue. More than 50 per cent. of these growths are calcified and may form coral-like masses filling the inter peduncular space and extending backwards to the pons. Since they are

Thomas Russell Cumming Fraser, Contemporary. Professor of Clinical Endocrinology, Royal Postgraduate Medical School, London.
Harvey Cushing, 1869–1939. See footnote, p. 453. Described this disease in 1932.

adherent to the basal arteries and adjacent nerves, they are irremovable. Occasionally single cysts are found in or above the sella, but the term suprasellar cyst describes only this one variety and is deceptively encouraging, as it suggests an invariably removable lesion. In fact, these tumours are by no means always suprasellar, nor are they necessarily cystic.

Clinical Features.—The symptoms depend upon the site of the tumour and the age of the patient.

(1) Intrasellar craniopharyngioma cysts produce symptoms like the inert chromophobe tumour, but in young subjects they inhibit growth by compression of the acidophil cells as well as inhibiting sexual maturation. The result is a fat, impotent dwarf,

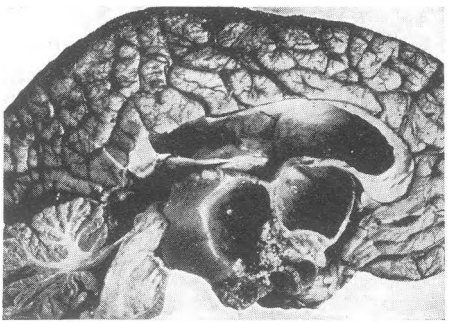


FIG. 469.—A large craniopharyngioma.

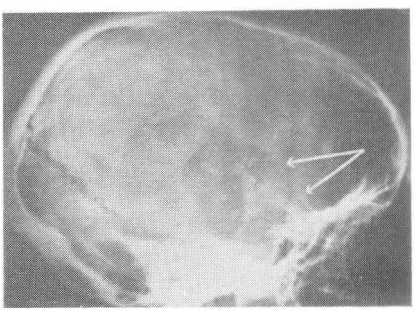


FIG. 470.—Arrows indicate suprasellar calcification in craniopharyngioma.

who may subsequently develop bitemporal hemianopia from upward pressure on the chiasma (Fröhlich's syndrome). Intrasellar calcification is often visible in X-ray.

(2) Suprasellar craniopharyngiomata in young subjects, by downward pressure on the sella, also produce a Fröhlich syndrome, but in this case involvement of the optic tract behind the chiasma causes homonymous hemianopia (fig. 470).

Pressure on adjacent centres of the hypothalamus, which control sleep and water metabolism, produce pathological somnolence and diabetes insipidus, so that the subject is liable to fall asleep during the day, and to drink and pass abnormal quantities of fluid. Similar symptoms may occur at a later age in adults.

Intraventricular tumours may produce blockage of the third ventricle with symptoms of acute intracranial pressure, headache, vomiting, and papillœdema, without localising signs; or an organic confusional state and dementia from cerebral atrophy following long-standing pressure.

Treatment.—Cystic tumours in and above the sella may be evacuated by frontal craniotomy. When large masses block the third ventricle, the cerebrospinal fluid may be short-circulated to the cisterna magna by leading a catheter from the ventricle through a burr opening in the skull, thence under the skin and occipital muscles to drain into the cisterna magna, which has been exposed above the foramen magnum (Torkildsen's operation)—ventriculo-cisternostomy.

Simmonds' disease, 'pituitary cachexia', results from depression of pituitary secretions affecting the gonads, adrenals, and thyroid gland following destruction of the anterior lobe of the pituitary by local thrombosis, hæmorrhage, cyst distension, post-partum necrosis, head injury, abscess, or tumour. The condition has also been caused by swabbing out the pituitary fossa with sclerosing fluid following local operation. Amenorrhœa, impotence, or persistent infantilism occur in a patient whose skin is typically yellow, smooth, dry, and atrophic with loss of hair. Unlike Fröhlich's syndrome, the patient is thin and cachetic from anorexia, suffering from weakness, lack of energy, and apathy, with low blood-pressure causing syncopal attacks and hypoglycæmia which occasionally gives rise to coma.

HYDROCEPHALUS

Hydrocephalus is usually spoken of as being congenital or acquired. Subdivision of these two groups is useful from a clinical point of view. When the

Alfred Fröhlich, 1871–1953. Formerly Professor of Pharmacology, University of Vienna, described his syndrome in 1901.
Arne Torkildsen, Contemporary. Neurosurgeon, Rikshospital, Oslo, Norway.
Morris Simmonds, 1855–1925. Practised medicine in Hamburg.

ventricular system is sealed off at some point, which may be as caudal as the exit foramina of the fourth ventricle, then the hydrocephalus is described as non-communicating. If the distended ventricles communicate freely with the subarachnoid space then the hydrocephalus is communicating.

FIG. 471.—Ventriculogram showing enormous size of ventricles in congenital hydrocephalus.

Congenital hydrocephalus is often associated with other abnormalities of the neuraxis, such as spina bifida or myelomeningocœle. It is produced by defective absorption of, or obstruction to, the cerebrospinal fluid. The commonest cause is failure of development of the arachnoid villi through which absorption takes place and a communicating hydrocephalus is thereby produced. Obstruction of the cerebrospinal fluid pathways most often occurs at points of normal anatomical narrowing, i.e. the various foramina and the aqueduct of Sylvius. The commonest causes are the Arnold-Chiari malformation, in which the roof of the fourth ventricle lies below the level of the foramen magnum and there is obstruction of the exit foramina and, at a higher level, congenital stenosis of the aqueduct of Sylvius (fig. 471).

Clinical Features.—The enlargement of the head may be pre-natal and constitute a rare cause of obstructed labour. After birth, rapid enlargement usually occurs. The scalp veins become distended, the fontanelles widen and are abnormally tense and the brow overhangs the roof of the orbits giving the eyes a 'sunset' appearance. Although the ventricles become enormously dilated and the cortex reduced to a mere shell, there are no motor symptoms. A certain minority of cases stabilise spontaneously, and intelligence is then preserved despite the enormous size of the head. Secondary endocrine effects result from pressure of the distended third ventricle in the pituitary fossa. Congenital stenosis of the aqueduct may not produce serious symptoms until the age of six or much later. It then causes raised intracranial pressure which may be associated with secondary endocrine disturbances.

Acquired Hydrocephalus

Non-communicating hydrocephalus is produced by obstruction within the cerebrospinal fluid pathways either by a tumour or as the result of a previous inflammatory process. Obstruction most commonly occurs at the points of anatomical narrowing (*vide supra*) and that part of the ventricular system cranial to the block dilates. This dilation may be unilateral in the case of obstruction of one foramen of Monro, or bilateral and symmetrical in the

François de le Boe (or Franciscus Sylvius, according to the Latinised form of his name), 1614–1672. Professor of Medicine, Leyden.
Julius Arnold, 1835–1915. Professor of Pathological Anatomy, Heidelberg University.
Hans Chiari, 1851–1916. Professor of Pathological Anatomy, Strasbourg University.

case of midline tumours, e.g. colloid cysts of the third ventricle, craniopharyngiomas, pineal and cerebellar tumours, and arachnoid cysts over the roof of the fourth ventricle. Communicating hydrocephalus is usually post-inflammatory in origin and complicates acute or chronic meningitis and sarcoidosis

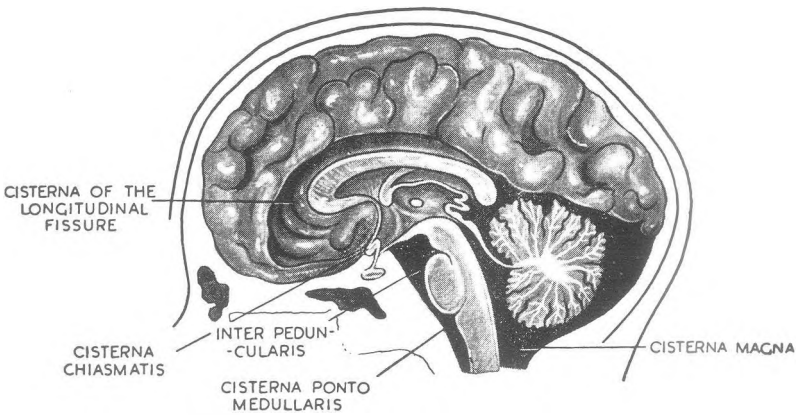


FIG. 472.—The principal arachnoid cisterns.

which has failed to respond to treatment. In acute meningitis surgical treatment consists of the formation of bi-frontal burr holes through which the ventricle can be tapped to relieve acute pressure symptoms or induce antibiotics. Where prolonged treatment is required a rubber reservoir with a tube leading through the burr hole to the ventricle (Ommaya) may be inserted under the skin, injection or aspiration being performed through this reservoir. In chronic cases blockage of the basal cisterns, cisterna interpeduncularis and cisterna chiasmatis, by inflammatory exudate or arachnoiditis secondary to the inflammatory process or subarachnoid hæmorrhage, throws a dam across the main channel for the ascent of cerebral spinal fluid to the absorption areas, and hence leads to stasis in, and distension of, the lateral ventricles, which nevertheless remain in communication with the subarachnoid spaces.

In the above types of hydrocephalus the intra-ventricular pressure is usually very high. An interesting type of acquired communicating hydrocephalus is becoming increasingly recognised in which the ventricular pressure is either normal or low. This type of hydrocephalus, usually discovered during the course of investigations for dementia, appears to be a latent effect of either trauma or some degenerative process.

Treatment.—Hydrocephalus is investigated by CT scan, ventriculography and air encephalography; isotope cisternography has been used. In cases of obstructive hydrocephalus the cause of the obstruction is removed if possible, e.g. tumour. The treatment of all forms of hydrocephalus however has advanced considerably in the last few years. During this time various shunting procedures have been developed and refined and have now largely replaced the older operations of third ventriculostomy.

Ayub Khan Ommaya, Contemporary. Professor of Neurology, Georgetown University School of Medicine, Washington, U.S.A.

The shunting procedures are of two general varieties:—

Intracranial Shunts.—These connect the ventricles to other intracranial areas such as the basal cisterns. Ventriculocisternostomy, in which one or both lateral ventricles are connected to the cisterna magna by polythene catheters, was devised by Torkildsen for treating obstructive lesions in the region of the aqueduct of Sylvius (p. 468).

Ventricular or Lumbar Shunts.—The basic principle involved with this type of shunt is the diversion of the cerebrospinal fluid from the lateral ventricle into a body cavity or into the right side of the heart.

Shunts without valves into the pleural and peritoneal cavities still have a limited part to play in the treatment of hydrocephalus but most surgeons now use the ventriculo-atrial shunt utilising one of the several valves now available for this purpose. All these valves have a similar function of promoting unidirectional flow of cerebrospinal fluid from the ventricles to the heart. The most popular of these are the Pudenz-Heyer and Holter valves.

The operation is simple and carries a low mortality. A tube passes from the ventricle of the brain to beneath the scalp where it connects with the valve (Holter) or a pump (Pudenz-Heyer). A further tube then passes down beneath the scalp and into the neck where it enters the common facial vein and thence into the internal jugular vein and right atrium. In the Pudenz-Heyer valve the valve is situated at the lower end of the tube in the heart.

The great disadvantage of these shunting procedures is that they necessitate the implantation of foreign material into the body and are liable to become blocked and infected. Despite these drawbacks the ventriculo-atrial shunt is a satisfactory treatment for hydrocephalus irrespective of its cause.

INTRACRANIAL ANEURYSM

Intracranial aneurysms may be considered in two main groups:

(1) *Subclinoid.*—On the carotid syphon of the internal carotid artery within the cavernous sinus.

(2) *Supraclinoid.*—On the main branch of the carotid above the cavernous sinus and on the circle of Willis.

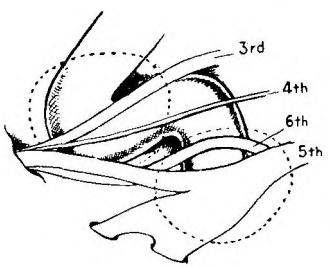


FIG. 473.—The relationship of aneurysmal sacs to structures in wall of the cavernous sinus.

Subclinoid Aneurysms

These are produced by weakening of the muscle coat of the internal carotid by uneven distension of its walls at the anterior and posterior curves of the carotid syphon leading to the protrusion of a sacular berry aneurysm. The condition is most common in women owing to the finer structure of their arteries.

These aneurysms are produced on the anterior and posterior bends, and hence anterior and posterior syndromes may be recognised which vary only in regard to the physical signs produced by pressure of the aneurysm sac on adjacent structures. Because they are below the level of the dura they do not cause subarachnoid hæmorrhage.

Anterior Aneurysms.—The commonest type press on the third and fourth nerves and first division of the fifth, causing supraorbital pain associated with ptosis, a dilated pupil, and divergent squint, owing to paralysis of the third nerve and unopposed action of the sixth.

Posterior aneurysms press on the sixth nerve and the whole of the trigeminal,

Robert Harry Pudenz, Contemporary. Neurosurgeon, Huntington Memorial Hospital, Pasadena, California, U.S.A. Heyer, Schulte Corporation. Santa Barbara, California, U.S.A.

John W. Holter, Contemporary. Engineer, Holter Instrument Company, Bridgeport, Pennsylvania. The valve was first used by Eugene B. Spitz, Neurosurgeon, Children's Hospital, Philadelphia, Pennsylvania, and is also known as a Spitz-Holter valve.

causing pain in the entire face with a convergent squint from sixth-nerve paralysis and unopposed action of the third, the eye remaining open.

Treatment.—Ligation of the internal carotid might cause hemiplegia if the circle of Willis were sclerotic or if the anterior communicating artery were absent. Ligation of the common carotid, however, is safer. This reduces pressure in the sac and relieves the nerve symptoms, since there is a second-cross cirulation above the level of the ligature. Blood passes across through the branches of the external carotid to reach the site of the ligature, then downwards to the carotid bifurcation above the ligature and upwards with the internal carotid with reduced force but in sufficient volume to prevent cerebral ischæmia. However, in the elderly, common carotid artery ligation carries a significant risk of hemiplegia, and therefore it may be wiser to treat the patient conservatively unless severe pain continues.

Arterio-venous fistula in the cavernous sinus may occur *spontaneously*, probably from rupture of an atheromatous plaque in the wall of the carotid artery in the elderly, or from the effects of *trauma* on the arterial walls in association with head injury. Acute back pressure in the veins draining into the cavernous sinus produces pulsating exophthalmos, distension of the orbital and supraorbital veins, and bone erosion and enlargement of the orbit. A few cases heal spontaneously. Usually, however, the condition will progress if untreated, and produce severe proptosis, chemosis and loss of vision.

Treatment.—The surgery of carotido-cavernous fistula is difficult and controversial. Common carotid artery ligation is usually ineffective and methods used include embolism of the fistula by a plug of muscle, closure of the fistula by a carotid balloon catheter and intracranial trapping procedures.

Supraclinoid Intracranial Aneurysms

Management of these intracranial aneurysms forms a large part of major neurosurgical practice. Aneurysms may cause:—

1. Spontaneous intracranial hæmorrhage (subarachnoid hæmorrhage).
2. Pressure upon local structures.
3. Epilepsy—very rarely.

Pathology.—*Congenital 'berry' aneurysms* are caused by a weakness in the media of the major arteries at the base of the brain. They occur particularly at points of junction or division of the arteries and assume different forms which may be saccular, fusiform, multilocular, unilocular, the base of the aneurysm may be narrow or broad. About 20% of patients who present with subarachnoid hæmorrhage have more than one aneurysm. The main sites of congenital aneurysm and their relative incidence are: Internal carotid artery (40%), Anterior communicating artery (28%), Middle cerebral artery (20%), Vertebro-basilar system (6%) (fig. 474).

Internal carotid artery aneurysms occur most commonly at the origin of the posterior communicating artery, and middle cerebral artery aneurysms at the trifurcation of that artery. Aneurysms on the vertebro-basilar system most commonly arise at the termination of the basilar artery.

Why aneurysms should rupture at a particular time is still uncertain but relevant factors are hypertension, the development of atheroma in the wall of the aneurysm, and

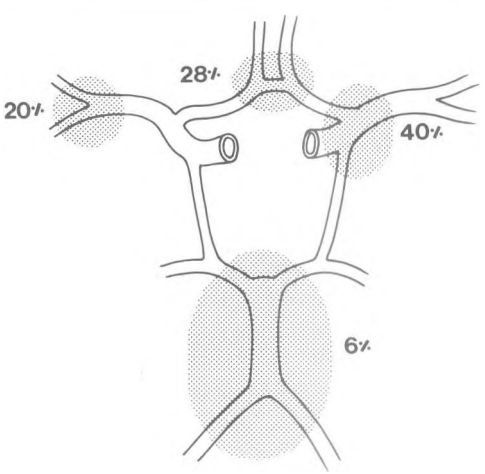


FIG. 474.—Main sites of intracranial supraclinoid aneurysm.

ischæmia of the wall of the aneurysm which weakens it further. When rupture occurs there is usually a rapid discharge of blood into the basal cisterns, an acute rise in intracranial pressure which may be fatal, disturbance of blood flow in critical vessels (e.g. perforating arteries) and ischæmic changes which may be fatal. Disruption of nerve cells and fibres, and hæmatoma, may produce persisting focal neurological deficits. Hæmorrhage is usually brief and is stemmed by the natural formation and organisation of clot over the tear in the wall of the aneurysm. However this clot is unstable and if lysis occurs, as is common, a second hæmorrhage may prove fatal.

Mycotic aneurysms occur in association with bacteræmia, as in bacterial endocarditis, and are produced by infection within the wall of the cerebral vessels, most commonly peripheral branches of the middle cerebral artery.

Subarachnoid hæmorrhage.—The term ' subarachnoid hæmorrhage ' is used loosely to indicate spontaneous intracranial hæmorrhage which has occurred primarily into the subarachnoid space of the basal cisterns, the most likely cause being intracranial aneurysm. However, aneurysm may rupture and bleed initially into the ventricles (e.g. anterior communicating artery aneurysm) or into the brain substance producing an intracranial hæmatoma (e.g. middle cerebral artery aneurysm). The peak frequency for aneurysmal hæmorrhage is between 50 and 54; in approximately 50% of patients who suffer a subarachnoid hæmorrhage an aneurysm is the cause.

Clinical Presentation.—The characteristic features are:

1. Sudden onset of severe headache.

2. Sudden loss of consciousness from which the patient may not recover (approximately 25%).

3. Onset of focal neurological deficit due either to the acute disturbance of an eloquent part of the brain (e.g. hemiplegia, dysphasia) or to direct pressure of the aneurysm upon an adjacent structure (e.g. ptosis, dilated pupil and defect of elevation and adduction of one eye from compression of the oculo-motor nerve by a ' posterior communicating artery aneurysm '). The onset of such defects is not usually as abrupt as in an ischæmic ' stroke '. Sometimes the development of an oculo-motor nerve palsy may precede the hæmorrhage, and is probably due to enlargement of the aneurysm prior to rupture.

4. Neck stiffness, photophobia and vomiting due to irritation of the meninges by blood. The blood pressure may rise and the ECG show changes similar to ischæmia.

5. In the majority of patients who survive the initial hæmorrhage, the conscious level improves after some hours. However a secondary gradual deterioration over the ensuing days is most commonly due to *spasm* of the cerebral arteries as a reaction to the discharge of blood into the basal cisterns. This spasm may lead to irreversible and often fatal ischæmic damage. Less commonly the subsequent progressive deterioration is due to an intracranial hæmatoma and brain œdema.

6. A second fatal hæmorrhage occurs in approximately 40% of patients within the six weeks following the first hæmorrhage.

Other causes of subarachnoid hæmorrhage include: hypertension, arterio-venous malformation (angioma) (see below), blood dyscrasias,

especially leukæmia, anticoagulant drugs, malignant brain tumours, especially metastases.

Management.—The objects of management are first to provide care to improve the chances of survival from the initial hæmorrhage, and second to prevent by surgical treatment a second fatal hæmorrhage from an aneurysm if it is shown to be the cause of the hæmorrhage. The risks of dying from a second hæmorrhage from an aneurysm rise to about 40% during the second to fifth weeks after hæmorrhage and from the end of the sixth week remain between 10 and 20% over the ensuing years. The factors which determine the favourability of patients for aneurysm surgery are complex and include younger age, good neurological state and especially level of consciousness, interval since hæmorrhage, absence of hypertension, absence of arterial spasm shown by arteriography, absence of significant cardiac and pulmonary disease, and the site, configuration and relationships of the responsible aneurysm. It is often a matter of weighing these factors against the natural history.

The usual steps in management after clinical assessment are:

1. **Lumbar puncture.**—It is important to take at least two consecutive specimens into separate containers for comparison. In subarachnoid hæmorrhage from any cause the fluid is evenly blood stained, and the finding of xanthochromia in the supernatant fluid confirms the diagnosis.

2. **Bilateral carotid and vertebral arteriography.**—The timing and indications for arteriography depend upon the exclusion of ' medical ' causes of hæmorrhage (see above), and the factors determining suitability for surgery. Therefore arteriography is usually deferred in patients who remain ' unconscious '.

3. **Surgery of Intracranial Aneurysm.**—With improvements in the methods of selection of patients suitable for surgery, and in operative conditions, and above all with the use of the *operating miscroscope* the surgical treatment of intracranial aneurysm has become increasingly exposure of the neck of the aneurysm, careful dissection and preservation of adjacent and parent arteries, and then *obliteration of the aneurysm by the application of a metal clip*. Despite the technical problems this method is favoured for most aneurysms. If the configuration and arterial relationships of the aneurysm prevent clipping, the risks of hæmorrhage may be reduced, but less effectively, by *wrapping* the aneurysm with gauze, muscle or plastic.

Internal carotid artery aneurysms have been treated by common carotid artery ligation, thereby reducing the overall mortality from further hæmorrhage from about 30% to 8%. However there are significant risks of hemiplegia, and therefore the tendency now is for direct attack, the approach being through a fronto-temporal craniotomy.

Anterior communicating artery aneurysm (fig. 475) may cause severe intellectual and emotional disturbances. Operation approach is by fronto-temporal craniotomy.

Middle cerebral artery aneurysms are approached through a fronto-temporal craniotomy, and entering the Sylvian fissure. These aneurysms may be fusiform dilatations of the trifurcation of the artery and in these circumstances are wrapped rather than clipped.

FIG. 475.—Carotid arteriogram showing anterior communicating aneurysm.

Vertebro-basilar aneurysms require either a fronto-temporal approach (terminal basil artery aneurysm) (fig. 476) or a posterior fossa craniectomy (vertebral artery aneurysm).

Local pressure effects of aneurysm.—These may produce symptoms and signs which occasionally precede rupture of the aneurysm, or more commonly persist with-

out rupture. Aneurysms on the posterior branches of the circle, posterior cerebral and posterior communicating, produce symptoms of third nerve palsy. Posterior cerebral aneurysms also cause hemianopia or pressure on the crus. Aneurysms of the anterior branches, such as the internal carotid bifurcation, anterior cerebral, and

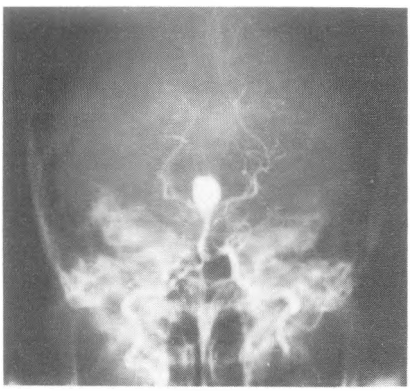


FIG. 476.— Vertebral arteriogram showing basilar artery aneurysm.

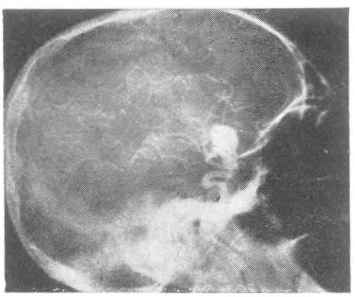


FIG. 477.—Large aneurysm of anterior cerebral artery, producing monocular blindness.

anterior communicating, produce pressure on the optic tracts, optic nerves, and chiasma respectively, producing hemianopia, monocular visual loss, or bi-temporal hemianopia and optic atrophy.

Prodromal symptoms are uncommon. There may be comparatively mild headache due to distension of the aneurysmal sac, lasting from a few hours to a few days before rupture. Unilateral supra-orbital pain, without tenderness or other signs of frontal sinusitis is suspicious of an enlarging posterior communicating aneurysm. Paræsthesia in a limb sometimes immediately precedes hæmorrhage from a vascular malformation.

Prodromal Signs.—The most important of these is unilateral 3rd nerve palsy, presenting as ptosis. When accompanied by the supra-orbital pain mentioned above it is an urgent warning of enlargement of a posterior communicating aneurysm demanding confirmative angiography and treatment before it ruptures. Rapidly advancing hemianopia or unilateral blindness may indicate enlargement of an internal carotid aneurysm adjacent to the chiasm. Subclinoid aneurysms may cause cranial nerve palsies (p. 472).

Vascular malformations involving arterio-venous shunts are similar to cirsoid aneurysms elsewhere. Arterialised varices burst into the substance of the brain and only secondarily into the subarachnoid pathways. An intracranial bruit may be audible by the patient or examiner, and occasionally, apart from rupture, they may be responsible for epilepsy or unilateral migraine (*vide infra*).

Intracranial angiomas are congenital malformations and not true tumours. Although an excess of blood passes through the large arterio-venous malformations, absence of a true capillary system causes defective nutrition of the affected area, with death of tissue, gliosis, and calcification. This may produce epilepsy and paralysis, often in adult life. Alternatively, the angioma may rupture spontaneously or following a minor trauma, producing apoplexy in young subjects.

Vascular malformations are treated by excision of that part of the lesion containing the actual arterio-venous shunt, including brain tissue immediately surrounding it. The risk of fatal recurrence from these lesions is much less than that from berry aneurysms, and treatment is neither so urgent nor so imperative. No attempts at removal should be made therefore, when the shunt occupies an essential area of the brain.

Intracerebral Hæmorrhage

The classical apoplexy, occurring typically in the ageing plethoric man with known hypertension and arteriosclerosis, is usually diagnosed correctly by the family doctor.

Fatal cases present with sudden stupor, stertorous respirations and hemiplegia. In younger patients with no known arterial disease, rupture of a berry aneurysm is the commonest cause of such a picture, and the early hemiplegia is due to encroachment of profuse hæmorrhage from the subarachnoid space into brain tissue. In both these cases only limited success may be hoped for from the removal of the blood clot from the brain, for much damage to neurones has already been done at the time of the hæmorrhage, and removal of the mass, though it may save the patient from death due to raised intracranial pressure, is often disappointing as far as restoration of cerebral function is concerned.

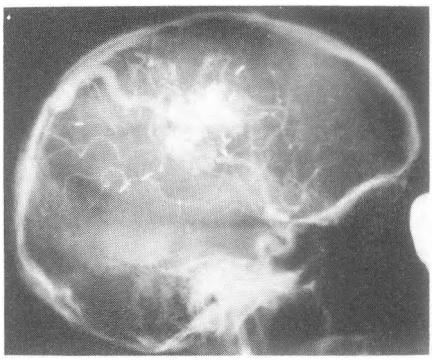


FIG. 478.—Carotid arteriogram showing parietal angioma and metal clips from surgery many years previously.

The diagnosis of intracerebral hæmorrhage is confirmed angiographically or by CT scan. Removal of the clot is done most effectively by craniotomy.

SURGICAL TREATMENT OF MENTAL DISORDERS

The surgical treatment of mental illness has undergone refinement. The early crude beginning followed the observations by Egas Moniz in 1936, that the injection of alcohol into both frontal lobes would produce a diminution in the intensity of emotional distraction in severely disturbed psychotic patients.

The operation of pre-frontal leucotomy sought to secure a similar effect by subcortical division of the entire central cone of white matter in both frontal lobes by a vertical incision in front of the ventricles. Although this operation relieved symptoms of agitation and tension it caused extensive brain damage with serious blunting of intelligence, flattening of emotion, and a high risk of epilepsy and could therefore only be considered in seriously disturbed schizophrenic patients who seldom made a satisfactory response—fewer than one fifth being discharged from hospital, the remainder confirmed in mental hospitals to form a permanent source of discouragement to psychiatrists. However, it could be seen that if a similar reduction in the intensity of emotional reaction could be secured without damaging effect, surgical treatment might be extremely valuable in cases of incurable psychoneurosis such as suicidal depression, melancholia, anxiety states and obsessional syndromes.

Ablation experiments in monkeys showed that lesions in the region of the primitive agranular cortex on the mesial and orbital aspects of the lobe produced an alteration in emotional tone without intellectual defect, indicating that the future of surgical treatment would lie in operations that interrupted the connections of the agranular cortex in the limbic system.

Le Gros Clark's observations on the distribution of the thalamo-frontal and fronto-hypothalamic pathways concerned in emotional feeling and reaction, provided a target for selective operations on limited cortical areas of the cingulate, convexity and orbital cortex which were excised by lobectomy or isolated by undercutting whilst leaving the majority of the frontal lobe undisturbed in attempts to produce a reduction in emotional tension in psychoneurotic subjects. Certain of these operations, notably those that affect regions of primitive agranular cortex such as cingulectomy and restricted orbital undercutting, were highly successful in the treatment of obsessional illness and depression.

In recent years the trend has been towards making small lesions by stereotactic methods, thus avoiding the more destructive aspects of open operations and the consequent scarring. Attention continues to be focussed upon the limbic system and its connections with the frontal lobe especially on the medial and orbital aspects of the hemisphere. Thus lesions of the medial frontal area involving the cingulate cortex and white matter may relieve, or at

Egas Moniz, 1874–1955. *See footnote, p. 458.*
Sir Wilfred Edward Le Gros Clark, 1895–1971. Professor of Anatomy, Oxford, England.

least improve, obsessional, tension and anxiety states; orbital cortex and white matter lesions may lessen the severe depressive illnesses. In the basolateral parts of the limbic system, stereotactically placed lesions of the amygdala may reduce aggressive behaviour. However it should be stressed that there is still debate over the indications for such surgery, much lay comment being ill-informed.

PARKINSONISM

Parkinson's disease (*syn.* paralysis agitans) is a condition characterised by tremor, rigidity, and akinesia. Unilateral or bilateral rhythmic tremor, producing the typical pill-rolling movement of the fingers and thumbs, may also affect other sites, e.g. the face, jaw, palate, tongue, and lower limbs. Excessive muscular tonus causes a rigid, expressionless, mask-like facies, an unblinking stare, and limb rigidity. The shuffling Parkinsonian gait with short steps results from rigidity and akinesia. A variable degree of dementia is also present in some cases. It is necessary to estimate the part played by each symptom in causing disability.

The syndrome is produced by degenerative changes in the extrapyramidal pathways—particularly at the substantia nigra in the brain stem. Degeneration of the large pallidal cells of the corpus striatum may be responsible for excessive rigidity.

The indications for surgical treatment of Parkinsonism have become much reduced in recent years due to the introduction of drugs such as læva-dopa. However stereotactically placed thermo-coagulating lesions in the ventro-lateral nucleus of the thalamus may still be necessary for the tremor of Parkinsonism and other basal ganglia disorders which fail to respond to medication, or are aggravated by medication.

James Parkinson, 1755–1824. General Practitioner, Shoreditch, London. Published 'An Essay on the Shaking Palsy' in 1817.

CHAPTER 27

THE SPINE

THE VERTEBRAL COLUMN AND SPINAL CORD

DISLOCATION

True dislocations are commonest in the cervical region, the oblique and vertical directions of the articular processes tends to result in their being fractured in the dorsal and lumbar regions.

Dislocation following hanging occurs between the atlas and the axis.

Forward displacement of the atlas follows rupture of the transverse ligament or fracture of the odontoid process. Death may occur immediately from injury to the brain stem and paralysis of the muscles of respiration. Dislocation at this level has also occurred from lifting a child by means of the hands encircling the neck from behind.

The base of the odontoid process may be cracked following head injuries and delayed displacement may occur after an interval of days. X-rays of the odontoid process, taken through the mouth, are essential where persistent occipital pain complicates head injuries.

Dislocations usually occur at the sites of maximum mobility, between the fourth and fifth, and fifth and sixth cervical vertebrae resulting from falls on the head when riding, or diving into shallow water, which causes excessive flexion of the upper part of the spine together with pressure on the vertex of the skull. The inferior articular process of the fifth vertebra is forced over the front margin of the superior articular process of the sixth. One or both of the articular facets may be involved, the situation being revealed by oblique radiographs. If both facets are involved the forward displacement is usually more than half the diameter of the vertebral body and vice versa. Serious neurologic involvement may accompany either situation. Pain is felt in the neck and may be referred along a local nerve root due to entrapment. Neck movements are limited and posterior tenderness is invariably present due to the rupture of the interspinous ligament which accompanies all dislocations.

Treatment.—Reduction of the dislocation should be carried out as soon as possible in all cases but where there is evidence of cord involvement the situation constitutes an emergency.

Skull traction should be instituted and under systemic analgesia the traction should be increased under radiological control with the neck in slight flexion. Weights of up to 18 kg (40 lbs) may have to be used. Once the facets have been seen to disengage the neck is slightly extended and the weights reduced to 3·5–4·5 kg (8–10 lbs). The facets should then settle back into their correct position. If this fails manipulation under anæsthesia may be attempted by a surgeon experienced in the technique. Otherwise the patient should be carefully turned into the prone position and open

reduction carried out under direct vision. In a fresh case this is fairly easily accomplished by levering the facets carefully over one another. Extensive removal of facet bone should be avoided because subsequent stabilisation of the neck will be rendered more difficult. At the end of the operation it is usual to wire the relevant spinous processes together over a bone graft. Some form of firm supporting collar is then used until consolidation has occurred.

In cases where closed reduction has been successful light skull traction should be retained for three weeks at the end of which a lateral radiograph should be taken. If this shows early evidence of interbody fusion then immobilisation should be continued until this is consolidated. Otherwise posterior fusion should be carried out as above.

Pathological dislocation of the atlanto-axial joint occasionally complicates a retropharyngeal abscess, or even tonsillitis. Flexion of the head and torticollis are obvious signs. The condition is confirmed by X-rays. Traction is required for a few weeks, followed by plaster fixation, and the pharyngeal abscess receives appropriate treatment (p. 691).

FRACTURES

Incomplete fractures do not interfere with the continuity of the spinal column. They include fractures of the spinous and transverse processes, laminæ, and fissured or compression fractures of a vertebral body. Fractures of the processes or laminæ are usually due to direct violence. Fractures of the spinous processes are common in the dorsal region for the processes are long and exposed to injury in falls on the back. Shovellers' fracture is a 'stress' fracture of spinous processes which occurs in men who use a shovel excessively, especially if they are undernourished. Localised pain and perhaps crepitus suggest the nature of the injury which is seldom shown by radiography. Fracture of the transverse processes occurs most usually in the lumbar region, where these processes are long and comparatively unprotected; a corresponding kidney may be injured. Fracture of a lamina may be associated with depression of bone and injury to the underlying cord.

Traumatic intraspinal hæmorrhage may occur in association with fractured laminæ or pedicles, or certain fractures of the vertebral bodies.

Hæmorrhage occurs either in the cord itself (hæmatomyelia) or as an extramedullary condition (hæmatorrachis); in the latter case the blood escapes either into the cerebrospinal fluid or the extradural space.

Intradural hæmorrhage produces symptoms of root irritation and is detected by finding old blood and an excess of lymphocytes in the cerebrospinal fluid at lumbar puncture.

Extradural hæmorrhage may fill the spinal canal gradually from below upwards causing progressive compression of the cord, followed by paraplegia (Thorburn's gravitation paraplegia).

Intramedullary Hæmorrhage.—Hæmatomyelia causes symptoms which follow the injury immediately. Destruction of the anterior horn cells causes a flaccid paralysis of the muscles concerned, while injury of the pyramidal tracts results in some spasticity of the legs. Pain and irritation are much less than in cases of extramedullary hæmorrhage. Bruising of the cord against the bony bars produced by cervical spondylosis sometimes results from minor falls or whiplash injuries. In the early stages symptoms and signs are often vague. Subsequently, obvious neurological signs, e.g. muscular wasting, present themselves, and are usually permanent.

Fracture Dislocations.—Fracture dislocations of the spine involve complete disruption of the column with rupture of the interspinous ligament, fracture or dislocation of the facet joints and a fracture through the vertebral body. These are commonest at the dorsi-lumbar junction and result from a

Sir William Thorburn, 1861–1923. Professor of Clinical Surgery, University of Manchester.

combination of flexion and rotation violence. They may also occur in the cervical spine although greatly outnumbered here by pure dislocations. The majority of these injuries are accompanied by damage to the spinal cord and the cauda equina. The lesion can usually be confidently diagnosed at the dorsi-lumbar junction by a combination of a typical history of flexion rotation injury, the presence of palpable displacement, and most important, the presence of a palpable gap between the relevant spinous processes as a result of the tearing of the interspinous ligament.

Burst Fractures.—Burst fractures are commonest in the mid-cervical and mid-lumbar regions. These are the result of axial violence such as a fall on the vertex or on to the heels and buttocks. The vertebral body disintegrates as the name suggests and posterior protrusion of portions of it frequently cause damage to the cord especially in the narrow cervical region.

SPINAL CORD INJURY

is produced by three factors:

(1) By long axis stretch occurring at the moment of acute flexion, causing concussion or rupture of nerve fibres and vessels within the cord.

(2) By nipping of the cord between the lamina of the vertebra above and the edge of the fractured body below, producing a local crush of the cord.

(3) By the protrusion of a disc at the moment of flexion leading to compression of the anterior aspect of the cord. This latter mechanism is however surprisingly rare.

In the adult the spinal cord ends at the level of the lower border of the body of the first lumbar vertebra; hence fracture-dislocations below this level can only be associated with injury to the cauda equina. Anatomical factors govern the extent of the cord and nerve lesion at various levels.

In the cervical spine the horizontal line of the articular processes and the large intervertebral discs produce a high degree of mobility with little strength, hence dislocation or fracture-dislocation occurs readily as a result of relatively minor forces sustained in injuries at sport, such as falls on the head in riding, diving, cycling, or football. Since there is relatively little ' follow-through ' produced by such forces and since the size of the neural canal in the cervical region is large in relation to the cord, cord injury may be partial or absent. When greater forces are in action, as when the neck is struck by a falling bough, considerable displacement occurs. Cord injury is then complete and often fatal.

In the dorsal spine the vertical articular processes and low mobility, combined with great strength, account for the fact that the spine can only be broken by major forces, such as the collapse of a roof in a mine-working; displacement is therefore considerable, little space is available around the cord, and cord injury is invariably present and usually severe.

In the lumbar spine vertical articular processes and great strength are combined with mobility. Fracture-dislocations are produced by major forces, often combined with some degree of rotation which causes fracture and displacement of the articular facets. But since the roots of the cauda

equina enjoy ample space in the lumbar theca, nerve injury may be absent or limited to a few roots of the cauda.

Injuries of the spinal cord occurring in association with fracture-dislocation and dislocation consist of (1) spinal concussion or (2) partial or complete spinal contusion.

Spinal concussion (**spinal shock**) is produced by long axis stretch on the spinal cord accompanying flexion. The grey matter of the cord consists of numerous nerve cells and synapses supported by a delicate tissue framework. A long axis stretch causes displacement of these structures with resulting disturbance of synaptic conduction. There is therefore an immediate loss of all functions which depend upon synaptic activity below the level of the lesion. Each anterior horn cell receives some hundreds of synapses, the stimuli passing through which maintain the central excitatory state in the nerve cell, by which the cell is maintained in a condition of readiness to respond and act as the final common path for voluntary movement, muscle tone, and reflex activity.

Cessation of the central excitatory state following synaptic disturbance therefore results in complete loss of voluntary movement, abolition of tone and reflex activity below the level of the lesion, producing a flaccid paralysis and retention of urine.

Pain and temperature sensation which cross a synapse in the inferior sensory decussation are also lost, but joint position sense which ascends the posterior columns is preserved. If the injury is merely spinal concussion, voluntary power and sensation will return within twenty-four to forty-eight hours. If concussion is superimposed upon an underlying partial or complete cord injury, the stage of spinal shock is prolonged. As time passes without improvement, recovery becomes increasingly improbable, and the appearance of a mass reflex at three to six weeks renders the outlook gloomy.

Partial or complete contusion of the cord is produced by nipping of the cord between the lamina of the displaced vertebra above and the edge of the fractured vertebra below; this causes a transverse zone of contusion and bruising. Above and below this level minute petechial hæmorrhages may be seen, resulting from the rupture of cord vessels by the long-axis stretching force. Œdema developing around these hæmorrhages may lead to a rise in the level of paralysis and sensory loss in the early days after the injury. Massive hæmorrhage ' hæmatomyelia ' is occasionally seen in the cord substance. Any damage inflicted on the cord is permanent and irreparable (p. 479).

Clinical Features.—The immediate effect of injury produces an initial picture of spinal shock resulting from the long axis stretch which masks the effects of the underlying organic rupture. There is, therefore, complete flaccid paralysis below the level of the lesion with retention of urine. The diagnosis of cord contusion can only be made with certainty in the early stage:

(1) If there is complete loss of all forms of sensation below the level of the

lesion. This indicates interruption of the posterior columns (which escape the synaptic disturbance of concussion) by bruising on the back of the cord.

(2) If the level of sensory loss and paralysis rises after injury, indicating ascending œdema.

(3) If the stage of spinal shock persists for more than forty-eight hours. In general terms, the duration of the stage of flaccid paralysis is proportional to the severity of the contusion. This stage of spinal shock may last for weeks in severe cases.

The clinical course of cord contusion falls into three stages:

(1) The stage of spinal shock.

(2) The return of reflex activity.

(3) The stage of septic complications.

In the stage of spinal shock, suitable measures are taken to protect the cord from further damage, and await the return of reflex activity in order to judge from the character of the reflex changes observed the nature and extent of the cord lesion. During this period precautions are taken to prevent or combat the onset of septic complications, such as urinary infection, bedsores, and pneumonia, in order to bring the patient to the final phase of active rehabilitation.

The Management of Cord Injuries.—In the stage of spinal shock every care must be taken to avoid unnecessary movement or examination which might increase the cord damage.

First Aid Treatment.—Cervical injuries may be transported on the back with the head supported between sandbags *without* flexion. Dorsal and lumbar fractures should be transported with the patient face downward with the pelvis and shoulders raised on pillows to hyperextend the spine. The patient must be lifted and turned on to a blanket, placed on a stretcher, by three people (or more, if the patient is heavy), controlling the head, trunk, and pelvis, without bending or torsion of the unstable spine. Morphia or other available sedation should be given to relieve pain and anxiety.

Clinical Examination.—On arrival at hospital the patient should be left lying on his stretcher until examination is completed and immobilisation is carried out. He should not be moved either for X-ray or clinical examination. Partial cord lesions may be rendered complete by excessive movement during clinical examination or over-zealous attempts to obtain a ' good ' X-ray picture.

Evidence or history of an accident is usually obtainable. Neurogenic shock frequently accompanies the injury, but the patient may be able to state that he has severe pain in the back, girdle pains encircling the body, or that he ' feels dead ' below a certain level.

The utmost gentleness must be exercised in order to prevent further injury to a cord which is only partially damaged or to avoid injury to an undamaged cord. Deformity of the vertebral column may be noticed. Paraplegia and anæsthesia extending from the level of the lesion are usually present, and are due either to spinal concussion, or crushing of the cord. The only examination required is to test the sensory level to pin-prick on the trunk,

which will indicate the segmental level of the cord lesion, and to test whether the patient can recognise movement of the foot or knee. If he can appreciate these movements, the case may be one of concussion. If there is complete sensory loss the outlook is grave. Having determined the segmental level of the lesion, as good an X-ray as possible should be taken by the lateral view at the corresponding vertebral level without disturbing the patient. Fracture of the manubrium sterni may be associated with fractures in the dorsal region. If a fracture-dislocation is present the fracture is immobilised as soon as the patient's general condition permits, but it may be necessary before this to apply restorative measures to counteract shock.

Reduction and Immobilisation

Cervical Injuries.—Dislocations have already been dealt with (p. 477) and crush fractures are relatively rare in this region. The more common ' burst ' injury which usually results from axial compression forces is usually accompanied by serious cord damage. If this is complete the prognosis is virtually hopeless and skull traction should be continued until the fracture consolidates in order to avoid the development of unacceptable deformity. In incomplete lesions the cord may be decompressed by removal of the damaged vertebral body from in front and replacing it with a massive bone graft. However the neurological benefits from this procedure have not been unequivocally demonstrated and some recovery frequently follows conservative management. Laminectomy is contraindicated since it has not been shown to improve the neurological prognosis and it greatly increases the instability of the spine.

Dorsal Spine.—These are often simple wedge compression fractures which are perfectly stable and pose no threat to the spinal cord. Symptomatic treatment and early mobilisation is indicated. More rarely fracture dislocation may occur, usually after extreme violence. The neural canal is narrow at this point and the cord invariably transected. The fracture is technically stable due to the support of the rib cage so that surgical intervention is almost never indicated, the treatment being that of the paraplegia itself.

Lumbar Spine.—Fracture dislocations are usually, although not invariably, associated with damage to the spinal cord and cauda equina, and are commonest at the dorsilumbar junction. Severe displacement requires open reduction to eliminate the deformity and to prevent late mechanical problems and also in the hope of relieving some pressure on the cord. The spine is then stabilised by plates bolted to the spinous processes or if these latter are fractured by the use of Harrington rods (p. 288). Spinal fusion is unnecessary except in the rare pure dislocations since the healing fractures will eventually stabilise the spine. Cases with minimal displacement may be treated without operation by postural nursing but this requires a degree of expertise usually found only in specialist Spinal Injury Units. Cases without neurological involvement may be mobilised post-operatively in a plaster jacket, but the use of plaster in the presence of a paraplegia is strongly contraindicated because of the inevitable risk of developing pressure sores.

Nursing.—Symptomatic treatment is now instituted. To prevent bedsore formation scrupulous drying and powdering of the skin is combined with regular turning of the patient every two hours to prevent local pressure, the patient being turned day and night in a Hey Groves bed or on banks of pillows. Attention to the bowels is necessary, and aperients or enemas are administered as required. If meteorism is allowed to occur upward displacement of the diaphragm causes pulmonary embarrassment. Hypostatic pneumonia is a grave danger, particularly if important accessory muscles of respiration, e.g., abdominal muscles, are paralysed, when coughing and expectoration are hampered. Contracture of muscles is prevented by frequent passive movements of the limbs.

Ernest William Hey Groves, 1872–1944. Professor of Surgery, Bristol, England. Co-founder (with Moynihan) and first Editor, British Journal of Surgery.

Diet.—The patient should be given a high-protein diet to compensate for loss of serum from bedsores.

Care of the Bladder in Cord Injuries.—All cord injuries suffer from retention of urine in the initial stages. This may last for a few days to a year in recoverable lesions, or be permanent in complete lesions. Sixty per cent. of the mortality of spinal injury is attributable to ascending urinary infection producing pyelonephritis, and this infection results from instrumentation.

The Care of the Bladder is described in Chapter 57.

The Return of Reflex Activity.—Spinal shock should show some evidence of abatement at the end of 48 hours. If no sign of recovery is evident then the prognosis is gloomy for there is bound to be partial or complete contusion of the cord in such cases. The phase of spinal shock and flaccid paralysis persists for days or weeks according to the severity of the cord lesion. When it passes off it will be found that lower motor neurone paralysis persists in a few muscle groups corresponding to the level of the lesion, owing to the crushing of the anterior horns at this level.

Partial cord lesions show spastic paralysis in extension with exaggerated reflexes and extensor plantar responses, and sometimes with uninhibited flexor spasms. There may, however, be some return of sensation. Retention of urine may persist for up to one year.

Complete cord lesions are followed by spastic paralysis in flexion with gross flexor spasms and the appearance of mass flexor reflexes of spinal automatism, which indicates complete lack of inhibition of the spinal flexor reflex arcs. In this reflex the slightest touch on the foot will produce flexion of the ankle, knee, and hip on the side of stimulation followed by a similar contraction of the opposite leg, sometimes associated with evacuation of the bladder and rectum. The result of this reflex may be distressingly encouraging to the patient as it may be misinterpreted as evidence of recovering function, whereas it indicates that function can never recover.

Cervical.—In high cervical lesions all the respiratory muscles are paralysed, including the diaphragm, which receives its motor supply from the phrenic nerves, mainly the C.4 segment. If the fifth segment is injured, the arms, trunk, and legs are all paralysed, and the patient only breathes with his diaphragm.

In lesions of the sixth cervical segment the only movement remaining is abduction of the shoulder by the deltoid muscles, the biceps and triceps and small muscles of the hand being totally paralysed. At the seventh segment flexion of the elbow is retained by the action of biceps and brachioradialis but the triceps is paralysed. Lesions at the first thoracic level produce paralysis of the small muscles of the hand with a Horner's syndrome from paralysis of the cervical sympathetic fibres which leave the cord at the first thoracic root (fig. 146). At lower levels a band of hyperæsthesia is usually detected encircling the trunk, one segment above the site of injury.

Lumbar.—The lumbar enlargement corresponds to the twelfth dorsal and first lumbar vertebra. It contains the centre for the nervous control of the bladder. In complete cord lesions above the tenth dorsal segment, after an initial phase of retention due to spinal shock, since the bladder centres are intact and uninhibited, an automatic bladder eventually develops, evacuation occurring when the intravesical pressure rises, the patient having to wear a receptacle.

Complete lesions involving the bladder centre at the lumbar enlargement produces permanent retention with overflow incontinence from paralysis of the detrusor

Johann Friedrich Horner, 1831–1886. Professor of Ophthalmology, Zurich, Switzerland.

muscle innervated by the third and fourth sacral nerves. But since muscles of the upper abdominal wall can be contracted voluntarily by the seventh to tenth dorsal segments, patients can be taught to evacuate the bladder by straining and by pressing their hands supra-pubically during the day. This is made easier if the resistance of the bladder neck is diminished by a transuretheral ' V ' resection of the internal sphincter (Chapter 58), but he must wear a receptacle at night.

The centre for defæcation is also situated in the lumbar enlargement and damage to this is followed by a patulous anus and incontinence.

Cauda Equina.—Injuries to the cauda equina give rise to disabilities which correspond to the actual nerves involved. Usually the injuries are in the lower lumbar region and spare the lumbar roots so that contraction of the muscles on the front of thighs and legs are preserved. Involvement of the sacral roots, however, produces anæsthesia in the back of the legs and saddle area of the perineum with urinary retention.

Rehabilitation.—Normally the patient is confined to bed for the first 2–3 months until the spine has become stabilised by bony healing. The start of rehabilitation need not be delayed on this account however. The work of Guttmann has revolutionised the prospects of the paraplegic patient, who can now be taught to work and earn a living, even with a complete lesion of the cord. The principles underlying Guttmann's methods are:

(1) To bring the patient into the best possible physical condition by the elimination of sepsis from bedsores and urinary tract.

(2) To over-develop the muscles above the site of the injury so that these may move the pelvis.

(3) To move the splinted legs by movement of the pelvis.

Pressure zones are normally preventable by good nursing and regular 2 hourly turning and re-establisment of this regime may heal existing lesions. In some resistant cases however recourse has to be made to plastic surgery (p. 166).

Bladder training and the treatment of refractory cases or urinary infection is described in Chapter 57.

Rehabilitation of Muscular Activity and Locomotion.—Contractures are first relieved by physiotherapy, tenotomy, or partial neurectomy. If necessary, the lower portion of the completely divided cord is *destroyed* by intrathecal alcohol injection to abolish mass reflexes and flexor spasms which would prevent walking and which may be extremely painful if any sensory fibres persist. When the legs are straight, rope climbing and trapeze work is started to encourage enormous development of the muscles of the shoulder girdle. Lying in a sling, the patient then learns to swing the pelvis from the shoulder girdle using the long muscles, latissimus dorsæ, erector spinæ, and abdominal recti and obliques. Then, with his arms supported on parallel bars, he learns to move the pelvis like a hula-hula dancer, and when proficient in this art light aluminium splints with controllable knee-locks are fitted. Using sticks, the patient moves the pelvis from his shoulder and with the pelvis go the splinted legs, thus permitting the patient to walk slowly for short distances to a wheeled chair in which he may move about and engage in sport and training in specialised occupations, such as watch-making, lens-making, diamond-cutting, etc.

Complications.—Death may result from the following causes :

(1) Shock, which may be immediately fatal.

(2) Cessation of respiration from ascending cord œdema within hours or days, if the lesion is near the fifth cervical segment.

(3) Hypostatic pneumonia within days or weeks, especially if the lesion is sufficiently high to cause paralysis of abdominal and intercostal muscles.

(4) Infection is prone to spread to the kidneys, causing death from pyelonephritis

Sir Ludwig Guttman, 1899–1980. Director of the National Spinal Injuries Centre, Stoke Mandeville Hospital, Bucks, England.

(*syn.* surgical kidneys) within months or years. Necropsy reveals the renal cortex and pelvis studded with abscesses and areas of acute inflammation.

(5) Urinary calculi may occur as immobilisation leads to decalcification, with increased renal calcium excretion, also recumbency encourages stagnation of urine in the kidneys. Infection further increases the risk. Such calculi are usually painless, even though the kidney is both obstructed and infected, since the anæsthesia of spinal injury affects visceral as well as somatic structures.

(6) Bedsores, which hasten death owing to toxic absorption.

Indications for Laminectomy

In the Acute Stage.—For the removal of a traumatic cervical disc protrusion causing cord compression without a fracture (p. 495) or for hæmatorrachis (p. 478). Apart from these rare conditions the indications for laminectomy are nil. The cord injury is irreversible and is made worse by meddling with indriven bone.

In the Late Stages.—Laminectomy is rarely required for the relief of cord compression produced by an arachnoid cyst which is impairing recovery of a partial lesion. Recovery occurs up to a point then deteriorates and lumbar puncture shows a block on Queckenstedt's test (p. 490).

Gunshot Wounds of Spine.—Portions of metal driven against the lamina or vertebral body and bouncing off away from the cord may nevertheless inflict an irreversible injury by exciting massive hæmorrhage in the cord substance. Direct penetration reduces the cord to a custard-like mass of diffluent cord and bone chips

INFECTIVE CONDITIONS

Meningitis.—Acute infective meningitis is likely to follow a penetrating wound, or may be a complication of septicæmia. Severe constitutional disturbances follow with local and referred pain, hyperæsthesia, muscular spasms, and increased reflexes. Extension to the basal meninges is usual, and the only hope lies in an adequate course of penicillin and chemotherapy.

Spinal Epidural Abscess.—Pus may collect in the extradural space of the cervical mid-dorsal or lumbar regions, to which infection has extended through the intervertebral foramina from adjacent sepsis, particularly from a perinephric abscess in the lumbar region. This produces a syndrome of progressive cord compression and meningeal irritation. The pus is evacuated by laminectomy.

SPINA BIFIDA

Embryology.—During the second week of intrauterine life a longitudinal furrow appears on the dorsum of the embryo, this groove being formed by infolding of the epiblast. The margins of the neural groove unite, so that it becomes converted into a tube from which the nervous system is developed. This epiblastic tube becomes separated from the surface by mesoblast, which grows over it from either side, and from which are developed the vertebræ, spinal muscles, membranes, etc. (fig. 479). In each segment bars of cartilage appear on either side of the neural tube, and during the fourth month they fuse with each other to form the vertebral arches. Failure of fusion of these arches gives rise to spina bifida, with which is frequently associated mal-development of the spinal cord and membranes. The incidence of spina bifida, excluding spina bifida occulta, is 0·1 per cent.

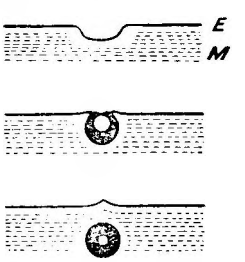


FIG. 479.—Development of the spinal cord. *E* epiblast, *M* mesoblast.

The types of spina bifida are as follows:

(1) *Spina bifida occulta* is due to failure of the neural arches to unite, but there is no protrusion of cord or membranes. Frequently only one vertebra is affected, most commonly in the lumbo-sacral region. A local patch of hair, a nævo-lipoma, or a depression in the skin are suggestive of underlying bony deficiency. A fibrous band, the *membrana reuniens*, connects the skin to the spina theca. Growth of the body causes the membrana to pull on the theca and nerve roots. Foot-drop, nocturnal enuresis, or backache occur when the child is older or in adult life. Many cases are symptomless and are undiagnosed unless an X-ray is taken for some other reason.

Hans Queckenstedt, 1876–1918. Chief of Medical Services, Hamburg, Germany, described the test in 1916. Killed by an army waggon on last day of 1914–18 War.

(2) *Meningocele.*—This is a protrusion of meninges through a defect in the spino-laminar segment (fig. 480). It contains only cerebrospinal fluid.

(3) *Meningo-myelocele.*—The normally developed spinal cord or cauda

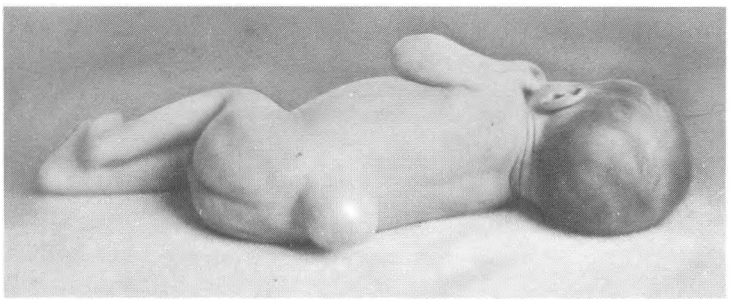


FIG. 480.—A typical meningocele which was excised successfully.

equina lies in the sac, and may be adherent to the posterior aspect. The cord or nerves can be seen as dark shadows on transillumination.

(4) *Syringo-myelocele.*—The rarest type of spina bifida, in which the central canal of the cord is dilated, and the cord lies within the sac together with the nerves arising from it.

(5) *Myelocele* results from arrest of development at the time of closure of the neural furrow. An elliptical raw surface is seen, which represents the ununited groove. At the upper end the central canal opens on the surface and discharges cerebrospinal fluid.

With the exception of spina bifida occulta, myelocele is the most common type of spina bifida, many cases are stillborn. If the child is born alive, death ensues within a few days from infection of the cord and meninges. Gross talipes is obvious.

Meningocele and meningo-myelocele are distinguishable on transillumination, a depression in the skin is sometimes produced by adherent cord or nerves in a meningo-myelocele. Interference with the spinal cord or nerves may occur in either condition but is more severe in meningo-myelocele, in which condition bilateral talipes with trophic changes are common, and in advanced cases extensive paralysis of the legs and incontinence occur. In serious cases no surgical intervention is indicated (see also p. 318).

Treatment.—Operation for meningocele or meningo-myelocele is advisable as soon as the surgeon is of the opinion that the child's strength and condition warrant the procedure (often within a few days of birth), otherwise the sac is liable to grow out of proportion to the growth of the child, and the overlying skin will become atrophic and ulcerate.

Operation is performed under local anæsthesia with the child tied face downwards on a splint. The child is kept with the head low to minimise escape of fluid, and given a feeding-bottle during operation. The sac is opened and redundant membrane excised. If the cord or nerves are adherent, they are either freed by dissection, or separated with a strip of attached membrane and replaced in the vertebral canal. Membranes are sutured over the cord, spinal muscles are approximated and the wound is reinforced with flaps of sheath from the erector spinæ muscles. Some cases subsequently develop hydrocephalus from the effect of associated congenital abnormalities at a higher level (p. 467). Those cases which survive with gross

paralysis of one or both lower limbs either after operation or without operation when the sac fails to rupture and develops some form of permanent coverage, may achieve some useful activity. Rehabilitation follows the lines of Guttman's rehabilitation of traumatic paraplegia (p. 484) the patient being provided with a suitable urinary receptacle and being taught to walk with the paralysed limbs supported with splints.

Operation for spina bifida occulta is required when neurological symptoms develop later in life. These symptoms are only rarely produced by traction from the membrana reuniens alone but are more likely to be due to compression of the cord by intra- or extradural lipomas or the effect of diastemato-myelia, a condition in which the cord is split in the mid-line by a bony spur which devides the neural canal beneath one laminæ into two lateral compartments. These abnormalities are almost always associated with the presence of a hairy tuft on the overlying skin, which is an important warning sign (fig. 481).

Myelography is required in all cases prior to operation.

At operation a tough fibrous band is encountered which leads down from the skin or deep fascia through a bony aperture to the dura mata. The dura is opened and the band excised together with any fibro-lipomatous tissue.

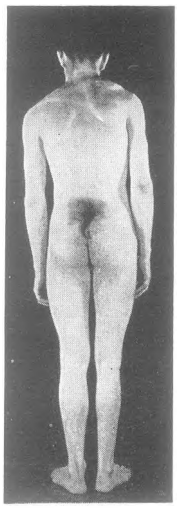


FIG. 481.—Spina bifida occulta with a tuft of hair, and wasting of the right leg due to traction on nerves in the spinal canal. Also right-sided Sprendel's shoulder. *(R. L. Benison, F.R.C.S., Wolverhampton.)*

INFLAMMATORY DISEASES

Acute osteomyelitis occurs in the epiphysis of the body of a vertebra or, more rarely, one epiphysis of the neural arch. Severe constitutional disturbance is associated with local pain and tenderness. Pain may also be referred along an adjacent spinal nerve. If the disease commences in a neural arch, some local evidence of inflammation is often detected. Acute osteomyelitis of a vertebral body has been mistaken for spinal meningitis, acute appendicitis, acute pancreatitis, and other abdominal conditions.

As soon as the disease is suspected, penicillin is administered, supplemented by chemotherapy. X-rays assist in determining the progress of the disease, and evacuation of pus or sequestrectomy is performed if necessary.

Tuberculous Disease—p. 225.

Spondylitis—p. 239.

TUMOURS OF THE VERTEBRAL COLUMN

Innocent tumours are extremely rare and include chondromas, osteomas, and fibromas. Many cases previously described as chondromas were in reality protrusions of an intervertebral disc. These tumours produce a syndrome of spinal compression affecting the anterior surface of the cord (p. 493) associated with characteristic X-ray appearances.

Malignant tumours of the spine are primary or secondary. Primary sarcoma is most frequent in children and young adults. The neural arches are usually affected, a palpable swelling may be detected in the paravertebral muscles, or invading tumour is discovered at this site during laminectomy performed for spinal-cord compression. Operative removal is always incomplete. Deep X-ray therapy is seldom effective.

Secondary deposits in the spine are common and far outnumber primary spinal or spinal-cord tumours. The majority are produced by bone metastasis chiefly derived from the breast or prostate. Deposits (from broncho-carcinoma) may rarely occur in epidural fat or in the cord itself. Malignant ganglion neuroblastomas of the sympathetic chain may invade through the intervertebral foramen.

Clinical Features.—Bone deposits produce severe local pain which may be the only symptom for a variable period. Later, as nerves become in-

volved, the pain becomes girdle in type and increases in intensity. It is aggravated by movement, so that the patient may remain crouched in a chair or huddled up in bed for hours at a time. If diagnosis is established at this stage, deep X-ray therapy may prevent vertebral collapse and cord damage.

The sudden collapse of a vertebra in which a deposit has developed painlessly may lead to instantaneous paralysis and severe back pain as the first symptom. A similar disaster may occur if manipulation of the spine has been unwisely performed in an attempt to relieve the local pain produced by a vertebral deposit which has not been diagnosed. The diagnosis of secondary tumour should always be suspected when severe local pain is associated with a high erythrocyte sedimentation rate. Most commonly, a period of local pain is gradually followed by the onset of paraplegia and spasticity with increased reflexes, but once paraplegia is established it may become complete in a few weeks.

Treatment.—Secondary deposits from the prostate respond remarkably to treatment with stilbœstrol, and those from the breast may regress with hormone therapy, adrenalectomy or hypophysectomy. In other cases, treatment is limited to a support to the spine, analgesics, and deep X-ray therapy. Intrathecal phenol injections are a most satisfactory method of relieving agonising pain. Laminectomy is not advisable in the presence of vertebral collapse.

TUMOURS OF THE SPINAL CANAL

Tumours of the spinal cord and membranes are only one-tenth as common as cerebral tumours. On the other hand, at least 70 per cent. can be cured by operation.

Tumours of the spinal canal are extradural or intradural, the latter being either extramedullary or intramedullary.

Extradural tumours are relatively uncommon. Lipomas are described but are very rare. The majority consist of meningiomas or neurofibromas. Meningiomas form a plaque overlying several segments of the cord and compressing multiple nerve roots and the cord in the cervical and dorsal region.

Dumb-bell tumours are neurofibromas. Part of the tumour is extradural, and is connected with a larger paravertebral portion by a narrow isthmus which passes through an intervertebral foramen. Spinal cord compression eventually develops, necessitating laminectomy and excision. Erosion of an intervertebral foramen is a useful radiological aid to diagnosis.

Extramedullary tumours (**75 per cent.**) are neurofibromas and meningiomas.

Neurofibromas are commoner in males and usually arise from a posterior nerve root forming a fusiform tumour about 2·5 to 3·75 cm. long present on the postero-lateral aspect of the cord. Occasionally there is an extradural dumb-bell extension through an enlarged intervertebral foramen.

Meningiomas occur almost exclusively in women and usually form a small globular tumour the size of a grape attached to the dura and indenting the cord.

Carpet-like tumours corresponding to the meningioma *en plaque* spread widely over the inner aspect of the dura in the cervical and lumbar regions, where there is considerable available space, and engulf the cervical cord or lumbar nerve roots before producing any symptoms of compression. In the author's experience, one tumour extended from the tenth dorsal vertebra to the bottom of the sacrum and measured 27·5 cm. in length.

Of all extramedullary tumours 12 per cent. occur in the cervical region,
60 per cent. in the dorsal region, and
28 per cent. appear in the lumbar region.

In relation to the cord, three-quarters are on the postero-lateral aspect and only a quarter on the anterior aspect. They are very easily accessible at operation and all except a few diffuse meningiomas, or meningiomas invading bone, can be removed completely.

Intramedullary Tumours (25 per cent.).—Approximately half these growths are diffuse gliomas which cannot be removed. Others consist of ependymomas, a growth derived from the ependyma lining the central canal, forming either solid tumours like pencils which can be excised, or cysts which can be evacuated. Vascular malformations comprise 4 per cent., and for these little can be done. More than half the intramedullary tumours lie in the cervical cord.

Symptomatology

Spinal cord tumours produce symptoms:

1. By involvement of posterior nerve roots causing root pain and local sensory loss.
2. By involvement of anterior nerve roots and anterior horns causing lower motor neurone paralysis with wasting and loss of reflexes.
3. By involvement of long sensory tracts causing distant sensory loss below the level of the tumour.
4. By involvement of pyramidal tracts, causing upper motor neurone paralysis.
5. By involvement of both pyramidal tracts, causing disturbance of bladder function.

Extramedullary tumours are lesions of small size usually situated on the postero-lateral aspect of the cord in relation to posterior nerve roots, hence root pain is the earliest symptom which may precede any evidence of cord compression by a period of years. In the early stage it is often mis-diagnosed; when referred to the shoulders and arms, it may be regarded as arthritis or neuritis. In the thoracic region it is regarded as angina pectoris or pleurodynia. In the abdomen it may be thought to originate from the gall-bladder, appendix, or pelvic organs.

The spinal origin should be suspected because the pain is *increased by coughing*, which raises the intraspinal pressure and forces the nerve root against the obstruction. Secondly, because pain follows the distribution of a spinal root and is often bilateral. Owing to the overlap of adjacent sensory areas, local sensory loss is absent or slight. Since most muscles receive a double nerve supply, the lower motor neurone paralysis is not recognisable unless extremely important roots are involved, such as the first thoracic.

The distant sensory loss is produced by pressure on the cord, which affects the longest sensory fibres first, hence whatever the level of the tumour the sensory loss starts in the legs and gradually spreads up the body as shorter and shorter fibres become affected. It seldom corresponds to the level of the tumour and is usually below the tumour level except in the latest stages. Pyramidal involvement occurs later, and the bladder symptoms last of all.

Examination shows bilateral spastic paralysis with a transverse sensory level on the trunk below the level of the tumour. The site of the tumour is reliably indicated by the level of the root pain.

	Extramedullary	*Intramedullary*
Root Pain . . .	Early	Late
Local Sensory Loss .	Nil or slight	Wide
L.M.N. Paralysis .	Absent except in C.8, T.1	Wide
Distant Sensory Loss	Ascending	Dissociated
U.M.N. Paralysis .	Late	Early
Bladder . . .	Late	Early

Intramedullary Tumours.—Fifty per cent. of intramedullary tumours occur in the cervical region of the cord. These tumours occupy a length of the centre of the cord. Root pain occurs last when the tumour presses against the walls of the spinal canal. Involvement of many posterior and anterior horns causes a wide belt

of local sensory loss and lower motor neurone paralysis, e.g. a dorsal tumour may cause extensive paralysis of the abdominal muscles. Distant sensory loss may be disassociated in type, as in syringomyelia, from involvement of spinothalamic tracts. Spastic paralysis and bladder symptoms from pyramidal involvement occur first of all. Examination reveals a wide belt of local sensory loss and lower motor neurone paralysis associated with spastic paralysis in a patient who has only complained of increasing paralysis and bladder disturbance of a few weeks' duration.

Extramedullary Tumours of the Conus Medullaris and Cauda Equina.—All lumbar and sacral nerve roots leave the cord between the tenth thoracic and first lumbar vertebra. Tumours of the conus medullaris lie at the eleventh and twelfth dorsal and the first lumbar vertebræ, and press upon multiple lumbar nerve roots and the tip of the cord. The symptoms include root pain, sensory loss, and lower motor neurone paralysis in the distribution of the lumbar roots which supply the front of the thigh and legs. There is pain in the thighs, wasting of the quadriceps, foot-drop and reduction of the knee-jerks. Pressure on the cord produces spastic paralysis in the legs with increased ankle-jerk and extensor plantar response.

Reflexes :	K.-J.	—	—
	A.-J.	+++	+++
	P.-R.	↑	↑

Tumours of the cauda equina occupy a central position among the sacral nerve roots below the conus, producing root pain, sensory loss, and lower motor neurone paralysis in the distribution of the sacral roots which supply the back of the legs. There is sciatic pain and wasting of the hamstrings and calves, and sensory loss in the saddle area. The knee-jerks are increased owing to weakness of the opposing hamstrings; the ankle-jerks and plantar responses are absent owing to sensory disturbance:

K.-J.	+++	+++
A.-J.	—	—
P.-R.	—	—

Spinal arachnoiditis may produce simple cysts of the arachnoid, form cystic collections which compress the cord, or multiple diffuse adhesions which produce their effect by traction on blood-vessels. The cysts should be localised and removed in the same manner as spinal tumours.

Accessory Methods of Investigation

1. *X-ray of the spine* may indicate the level of the tumour by showing absorption of the pedicles of the vertebra, enlargement of an intervertebral foramen on the lateral view, or erosion of the posterior aspect of a vertebral body.

2. *Lumbar puncture* may be required in early cases to differentiate such conditions as disseminated sclerosis. The presence of a tumour is indicated by an obstruction in the cerebrospinal-fluid circulation demonstrated by Queckenstedt's test. This is performed by charting the rate of rise and fall of the lumbar cerebrospinal-fluid pressure occurring during compression and release of the internal jugular veins.

In normal circumstances, such compression causes a rapid rise in cerebrospinal-fluid pressure and a somewhat slower passive fall. The first effect of obstruction is to prolong the passive fall, later there is a slight rise and fall, and finally no response at all.

3. *Examination of the cerebrospinal fluid*, including protein content. In neurofibromas the cerebrospinal fluid protein is raised from the normal 20 to 40 mg/100 ml (0·2–0·4 g/l) to figures of 200 to 400 mg/100 ml (2·0–4·0 g/l) without any evidence of block. When block becomes complete, the cerebrospinal fluid in the lower portion of the theca is encysted and transudation

occurs between the blood vessels and the cerebrospinal fluid with diffusion of protein and pigments. The fluid becomes yellow and may clot spontaneously (Froin's syndrome) and may contain as much as 50 g/l.

Cisternal Myelography.—Cisternal myelography is essential in order to demonstrate the level of the upper border of the tumour before operation. The shape of the shadow produced at X-ray indicates the type of tumour. The upper border of a meningioma which is irregular produces a wavy outline in contrast to the smooth crescentic margin produced by the curved upper surface of a neurofibroma. An intramedullary tumour produces separation of columns of contrast medium passing on either side of the expanding cord with resulting candle grease or trouser leg appearances (figs. 482, 483).

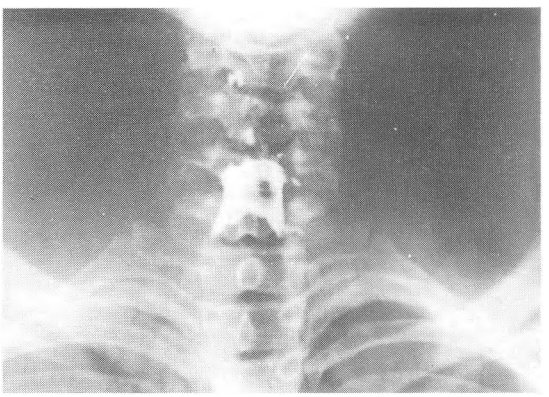


FIG. 482.—Descending cisternal myelogram showing complete obstruction of the down fall of myodil and the wavy outline produced by the upper border of a meningioma.

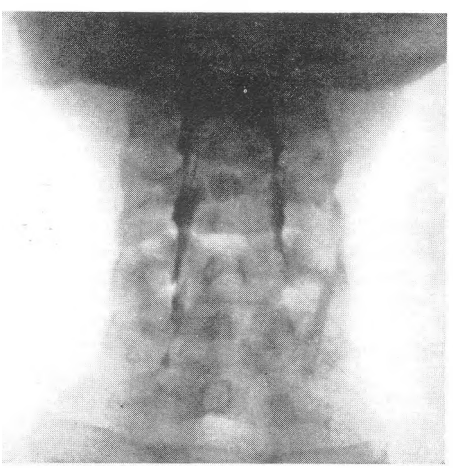


FIG. 483.—Ascending myelogram showing trouser leg appearance produced by columns of fluid ascending on each side of an expanded cord containing an intramedullary tumour.

Cisternal puncture is performed by inserting a lumbar-puncture needle in the mid-line at the level of the second cervical spine, the patient's head being flexed. The needle is advanced on a plane passing through the external auditory meatus to the outer margin of the eye. At a depth of 3 cm the stylet is removed. The needle is further advanced to a depth of 5 cm, when it pierces the occipito-atlantoid ligament, which feels like a sheet of brown paper; the moment this is pierced the operator should wait for fluid to flow out. It may not do so although the needle is in the cisterna magna, therefore the needle should be aspirated before it is advanced farther. If no fluid is obtained, it is put in a millimetre or two at a time until fluid escapes. Three ml. of Myodil is now injected and gravitates downwards until it is arrested by the tumour, the level of which is localised by examination on a tilting X-ray screen (fig. 484).

Treatment is by laminectomy when the upper level of the tumour has been accurately localised by cisternal myelography. A meningioma is excised together with a portion of the dura from which it arises.

A neurofibroma is removed with a portion of the nerve sheath to which it is attached.

Georges Froin, 1874–1932. French Neurologist. Described this syndrome in 1903.

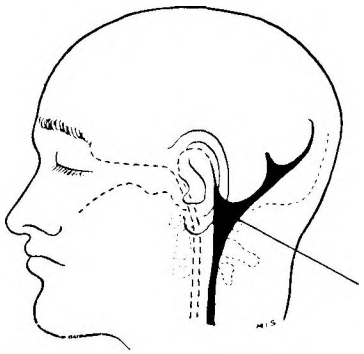


FIG. 484.—The technique of cisternal puncture.

Intramedullary solid ependymomas are excised after incision of the cord strictly in the mid-line between the posterior columns.

Intramedullary cysts are emptied.

LAMINECTOMY

In estimating the level of operation, the discrepancy between the level of the affected segment of the cord and that of the corresponding vertebra must be considered, owing to the disparity in the length of the spinal cord and vertebral column, a segment in the cervical region lies one vertebra above its corresponding vertebral body. In the upper dorsal region the tumour will be two vertebræ, and in the lower dorsal three vertebræ, above the corresponding vertebral body.

In estimating these levels by palpation of the spinous processes, in the dorsal region the processes overlap the vertebra below. Thus, presuming a tumour is localised to the ninth dorsal segment, it should be opposite the tip of the fifth spinous process, i.e. three vertebræ higher on account of the shortness of the cord as compared with the spinal column, and one vertebra higher because of the obliquity of the spinous process.

The patient is usually placed in the prone position with sandbags under the sternum and pelvis to leave the respiratory excursion of the diaphragm unimpaired in order to minimise back pressure on the epidural veins. The paravertebral muscles are injected with 1 : 100,000 of adrenalin in saline. A mid-line incision is made, the muscles are separated from the spinous processes by cutting diathermy, and raised from the neural arches with a raspatory after tendinous attachments have been cut through with curved scissors. Pressure from hot saline packs produces a dry field after any major vessels have been secured with coagulation. Self-retaining retractors are inserted and spines and interspinous ligaments are excised with bone-cutting forceps. The exposed raw edges of bone at the bases of the spines are now drilled through with a Hudson's burr to the level of the dura on each lamina, and finally the laminæ are removed with bone-biting forceps, thus exposing the dura and the ligamenta subflava beneath the site of each lamina. The ligamenta subflava and epidural fat are now incised in the mid-line and the ligaments removed. The dura is then inspected for evidence of the tumour, usually shown by a slight bulge at the tumour site or a zone in which pulsation is absent. If any doubt exists as to the correct level, the dura is picked up on a fine hook and a small mid-line incision is made, and stay sutures of fine silk are inserted into the dural edge. A No. 3 fine rubber catheter is passed up or down inside the theca to estimate the site of obstruction. If the exposure is correct and no further bone removal is required, the dura is opened fully in the mid-line by extending the original small incision by slitting the dura with guarded scissors.

Several fine silk stay sutures are inserted into the dural edge to hold the dura back against the bone and prevent bleeding from the epidural veins. Strips of fine lintine cotton tissue are inserted against the muscle to absorb any minor oozing in order to keep the field dry and free from cerebrospinal fluid. The tumour is now removed (fig. 485), following which the dura is sutured completely with fine silk, the muscles approximated with strong silk in layers, and the wound closed.

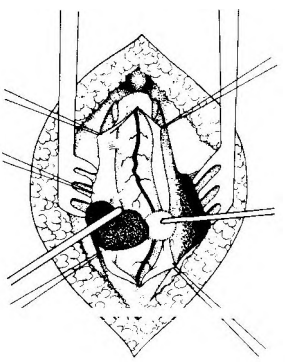


FIG. 485.—Removal of a meningioma indenting the dorsal cord.

Additional Indications for Laminectomy include:

1. Traumatic. These are considered on p. 485.
2. Inflammatory paraplegia complicating Pott's disease (p. 226).

William Henry Hudson, 1862–1917. Surgeon, Atlanta, Georgia, U.S.A.

3. Division of posterior roots to relieve chronic pain resulting from (*a*) malignant metastasis in lymph nodes or post-irradiation fibrosis in the high cervical region following malignant disease of the tongue and jaw; (*b*) from post-herpetic neuralgia following herpes zoster in the thorax and abdomen. Owing to the sensory overlap of adjacent spinal segments, more than one nerve root must always be divided. To relieve pain in three thoracic root areas, five must be cut to include those above and below the affected zone. This renders the operation of posterior rhizotomy inapplicable to the relief of pain in the arms or legs, as extensive denervation will abolish position sense and thus result in a severe ataxia.

4. Cordotomy. Division of the spinothalamic tracts for the relief of pain due to such conditions as an irremovable new-growth.

PROLAPSE OF INTERVERTEBRAL DISC

Intervertebral discs are interposed between the vertebral bodies, and serve not only as shock absorbers for the column but also provide the normal mobility between the adjacent vertebræ. Each disc consists of a soft central portion of spongy material, the nucleus pulposus, containing a remnant of the notochord, which is surrounded by a tough fibrous ring, the annulus fibrosus, which is attached to the adjacent vertebral bodies, the whole being enclosed between fibrocartilaginous plates above and below.

During normal flexion of the spine the disc is deformed and the annulus fibrosus and nucleus bulges backwards slightly into the neural canal.

Intervertebral disc protrusion is produced by the effect of flexion-forces acting upon the most mobile portions of the spine. A sudden strain with the spine in an unguarded position will rupture the tough annulus, allowing portions of the torn annulus and soft nucleus to escape into the spinal canal and form either a central protrusion in the midline under the posterior common ligament of the vertebræ, or a lateral protrusion at the side of the posterior common ligament adjacent to the intervertebral foramen (fig. 486).

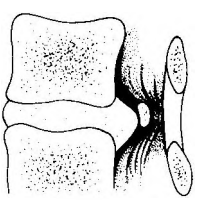


FIG. 486.—Intervertebral disc protrusion passing towards the intervertebral foramen, the posterior margin of which is constituted by the anterior edge of the subflavian ligament.

In 80 per cent. of cases the protrusion is traumatic in origin and there is either a history of sudden severe strain, or the patient's occupation is one in which flexion strain must be resisted, such as a packer, fireman, porter, etc. The condition is therefore more common in males.

In 20 per cent. of cases the condition is degenerative in origin. There is no history of injury. A small portion of the nucleus pulposus herniates through a weak area in the annulus without tearing that structure.

Since the mechanism demands the combination of stress and mobility, protrusions are most common in the most mobile portions of the spine which are subject to the greatest stress, hence approximately :

19 per cent. occur in the cervical region at the mobile C.5/6 and C.6/7 levels.

1 to 2 per cent. occur in the immobile dorsal spine.

80 per cent. occur in the lumbar regions, particularly at the mobile L.4/5 and L.5/S1. levels.

Escape of disc material leads to:

1. The narrowing of the invertebral joint space visible in X-ray in 50 per cent. of cases.

2. Slackening of the anterior common ligament of the vertebræ producing abnormal mobility between the vertebræ with local joint pain, and ultimately the development of intervertebral arthritis. Traction osteophytes form on the anterior aspect of the vertebræ and are visible in X-ray. Compensatory thickening of the ligamenta flava occurs in an attempt to check the abnormal mobility.

Massive protrusions may occur as a result of major cervical injuries (p. 495). Alternatively when the spinal ligaments are softened at the end of pregnancy the strain of labour may force out a massive protrusion in the lumbar region, giving rise to one form of obstetric paralysis.

A Schmorl's Node (fig. 487) is a radiological manifestation of an extrusion of the

Christian G. Schmorl, 1861–1932. Professor of Pathology, Dresden, Germany.

nucleus pulposus into the body of a vertebra. In itself, it has no clinical significance. As time goes by, the new bone activity which surrounds it condenses into a shell.

Lumbar Disc Protrusion.—In the lumbar region the roots of the cauda equina run obliquely over a number of intervertebral joint spaces, hence a lateral protrusion may press on two roots. The nerve root that issues from the corresponding intervertebral foramen may be compressed against the lateral margin of the disc, whilst the nerve root going to the intervertebral foramen next below is caught against its inner margin.

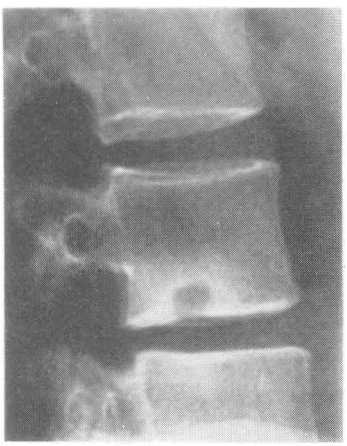


FIG. 487.—Schmorl's node in a lumbar vertebra (see text). Note also the narrowed disc space.

Clinical Features.—In all cases there is an initial period of low back pain resulting from injury to the disc. Later the pain will radiate to the leg when nerve roots become compressed. There is then root pain, accentuated by coughing, in the distribution of the affected nerve, paræsthesiæ and pins and needles in the peripheral portion of that area, cramps, and tenderness in muscles supplied by the nerve, and a variable degree of sensory loss and motor weakness. Since the commonest roots to be involved are the first sacral and fifth lumbar, pain is usually in the back and side of the leg radiating to the sole of the foot and the big toe, and is called sciatica. Sensory loss is found on the sole and side of the foot and the ankle-jerk is lost. But in higher disc lesions the pain may be referred to the front of the thigh or leg.

In an endeavour to reduce pressure on the nerve root, the patient adopts a position of scoliosis and walks with a limping gait. Attempts to touch the toes or at straight-leg raising, or neck flexion with the legs raised, increases pain by pulling the nerve root against the protrusion.

Physical Signs.—Pain is produced by friction of the nerve against the protrusion. Small protrusions only compress the nerve root slightly and the nerve is free to rub against the protrusion. *Small protrusions* thus cause very severe pain because there is maximum friction without much loss of conduction in the nerve. Since there is little impairment of conduction, physical signs are slight and limited to a small patch of sensory loss in the periphery of the skin area involved where long sensory fibres have been affected. *Larger protrusions* cause less pain because they lock the root

Root Involved	*Pain and Sensory Loss*	*Motor Weakness*	*Reflex Change*
1st sacral root	Back of leg. Sole and side of foot.	Gastrocnemius, weak plantar flexion.	Absent A.-J.
5th lumbar	Back of thigh. Most of lateral aspect of leg. Dorsum of foot to big toe.	Anterior tibials. Weak dorsiflexion.	Nil.
4th ,,	Side of thigh. Front of inner aspect of leg.	Quadriceps and anterior tibial, weak dorsiflexion and extension of knee.	Diminished K.-J.
3rd ,,	Front of lower thigh.	Quadriceps.	Diminished K.-J.
2nd ,,	Front of mid-thigh.	Quadriceps.	Diminished K.-J.
1st ,,	Groin.	Nil.	Nil.

Often combined in L.4/5 disc.

Often combined in L.5/S.1 disc.

more firmly so that there is less friction, while the conduction of the nerve is diminished. Since conduction is diminished the physical signs are more marked and sensory loss is extensive owing to involvement of short sensory fibres. *Massive protrusions* completely fix the root and cause no pain whatsoever, but produce the maximum sensory loss and motor paralysis (obstetric paralysis).

Treatment consists of confinement to bed until symptoms abate, which is usually a matter of two to four weeks. A plaster jacket is applied for two to three months, after which a spinal brace is worn for a further period. The majority of cases are cured by this routine. Operation is indicated if symptoms persist, if severe pain recurs, or if motor weakness is developing.

The disc is removed by hemilaminectomy, removing the adjacent margins of two laminæ and the subflavian ligament which obscures the view of the nerve root and disc protrusion until it has been excised (fig. 488). After removal of the subflavian ligament, the swollen root is seen overlying the protrusion by which it is displaced inwards. Loose disc material (looking like crab meat) is then grasped with forceps and extracted.

A full laminectomy is contraindicated as it encourages further weakness of the spine. Even after satisfactory removal of a prolapsed disc, symptoms or physical signs are not always alleviated. Two conditions may persist. Prolonged compression of the nerve may have resulted in interstitial neuritis producing sensory loss or motor weakness, or the intervertebral joint may be unstable or become affected by osteoarthritis. Intervertebral arthritis or instability is to be suspected when pain is not relieved by rest, but persists at night and wakes the patient when he turns over in bed. If this condition is present, spinal fusion should be performed when the disc is removed so as to afford complete rest to the affected segment of the spine.

FIG. 488.—A typical portion of protruded disc removed at operation.

Cervical disc protrusions are relatively uncommon. In many cases a diagnosis of cervical disc lesion is mistakenly applied in patients who are really suffering from referred pain arising from a cervical strain which has affected the interspinous ligaments, or lateral articulations following a flexion strain, or who are suffering from cervical spondylosis. As true cervical disc protrusions are uncommon, this diagnosis must only be accepted when there is definite evidence of compression of the spinal cord or nerve roots, and when the presence of a disc protrusion has been confirmed by demonstrating a lateral filling defect at myelography. In the cervical region the nerve roots run transversely and come into relation with one intervertebral disc only. Each nerve issues above the level of the corresponding vertebra.

Lateral protrusions press on the corresponding nerve and half the spinal cord, producing cervical rhizalgia with half-cord compression, a Brown-Séquard syndrome. They are never situated sufficiently far laterally to press upon the nerve root alone. Symptoms of brachial rhizalgia without cord compression are produced by intraforaminal osteophytes occurring in association with cervical spondylosis, which may, unlike a disc, affect multiple nerve roots. Each spinal root emerges above the corresponding vertebra, hence C.5/6 protrusion compresses the C.6 nerve root and half the cord (see Table below).

Mid-line protrusions compress the anterior part of the cord and affect the anterior spinal artery and the anterior spinal veins. Compression of the anterior spinal artery may affect the pyramidal and spinothalamic tracts and anterior horns. Compression of the veins which ascend on the front face of the cord produces stasis in the anterior horns below the level of the lesion.

The symptomatology varies on repeated examination as pressure on the vessels varies, and may simulate syringomyelia, disseminated sclerosis or primary lateral sclerosis. In time secondary atrophic changes produce an atrophy of the cord substance with permanent spastic paresis.

Acute midline protrusions occurring in cervical injuries may produce symptoms

Charles Edouard Brown-Séquard, 1817–1894. Physician, National Hospital for Nervous Diseases, London (1860–1864), Professor of Medicine, Harvard University, Boston, Mass. (1864–1878) and College de France, Paris (1878–1894). Described this syndrome in 1851. He was a native of Mauritius.

Root	*Pain, Sensory Loss*	*Sensory Loss*	*Muscle Weakness and Reflex Change*
C.6	Trapezius. Shoulder tip, outer border of upper arm. Dorsum of forearm.	Lateral border of upper arm. Dorsum of forearm and thumb.	Weakness of biceps, diminished biceps and supinator jerk.
C.7	Trapezius. Shoulder tip, back of upper arm, and dorsum of forearm.	Dorsum of forearm and all fingers except thumb.	Weakness of trapezius and extensors of the fingers. Diminished triceps jerk.

of a partial or complete cord lesion without bone displacement. High protrusions at the 4th cervical level may cause serious respiratory embarrassment.

Treatment.—If neurological symptoms and signs do not improve as the result of conservative treatment by support and immobilisation operation is required. Lateral protrusions are exposed by hemilaminectomy and removed by an extradural approach. Midline protrusions are treated by a full laminectomy of sufficient length to permit one to open the dura and divide the dentate ligaments for several segments above and below the protrusion in order to allow the cord to fall back and thus relieve pressure on the arteries and veins. Attempts to remove a midline protrusion may damage the anterior spinal artery and cause permanent paralysis.

Cervical Spondylosis is a degenerative condition characterised by (1) degeneration of the intervertebral discs with the formation of bony ridges running across the anterior surface of the neural canal and (2) the formation of osteophytes from the neurocentral joints of Luschka which project back-

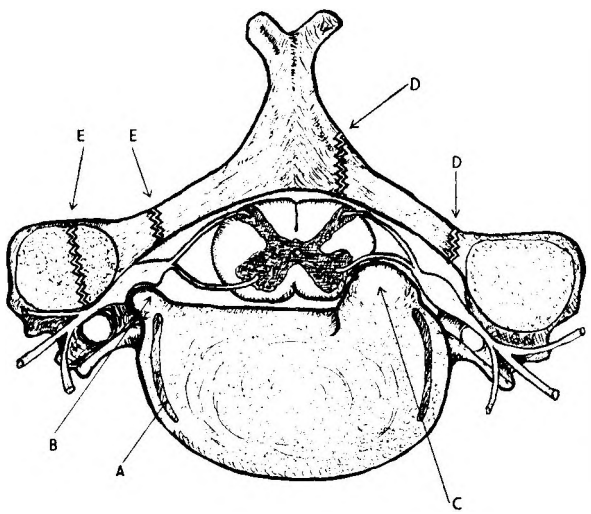


FIG. 489. — Diagrammatic cross-section of the cervical spine and cord showing the position of (A) the neurocentral joint of Luschka, (B) an intraforaminal osteophyte developing from that joint compressing the nerve root within the foramen, (C) a lateral disc protrusion compressing the cord and nerve root. Projection into the intervertebral foramen is prevented by the position of the joint of Luschka. (D–D) the section of bone removed in hemi-laminectomy giving access to a lateral disc protrusion. (E–E) The section of bone removed in hemifacetectomy giving access to an intraforaminal osteophyte. (*G. B. Northcroft, F.R.C.S., London.*)

wards into the intervertebral foramen (fig. 489). The condition may be quite symptomless or may cause neurological symptoms.

(*a*) Sudden strains inflicted on the affected joints as, for example, in the violent forward lurch sustained in road traffic accidents, so called ' whip-lash ' injuries, may give rise to referred pain in the occipital or post-auricular regions and in the upper portion of the trapezius, and between the shoulder blades.

Hubert von Luschka, 1820–1875. Professor of Anatomy, Tübingen, Germany.

Persistent occipital and posterior auricular pain can be relieved by excision of the corresponding nerves. Since referred pain and muscle spasm in the lower cervical region is the result of movement in the painful joints, these symptoms are often relieved by wearing a collar, but if they become chronic they may be permanently relieved by the insertion of a paraspinal inlay to immobilise the affected joints or by anterior spinal fusion in which the vertebrae are approached anteriorly internal to the carotid sheath. A portion of the upper and lower surfaces of adjacent vertebrae are channelled out to form a prepared bed into which a small bone graft is inserted from in front. Great care is required to avoid penetration of the dura and damage to the closely adjacent spinal cord. (*b*) Bony ridges on the anterior surface of the cord may compress arteries and veins with consequent neurological symptoms and spasticity and ultimate cord atrophy simulating a mid-line disc lesion. This should be treated by division of the dentate ligaments. (*c*) intraforaminal compression by osteophytes may affect multiple nerve roots. The nerve roots may be decompressed by excision of the back of the intervertebral foramina by hemifacetectomy.

Spondylolisthesis.—This is a condition in which a lower lumbar vertebra, usually the fifth, slips forward through the plane of the intervertebral disc below it and so carries with it the whole of the upper portion of the spine. The essential lesion is a separation of the body of the vertebra from the posterior articulation, lamina, and spinous process as a result of a defect in the pedicles which hold these two parts of the vertebræ together (fig. 490). Because the lamina is left behind in its normal position, the forward displacement of the vertebral body does not narrow the spinal canal. The deformity can, however, cause root pressure which may manifest itself as sciatica.

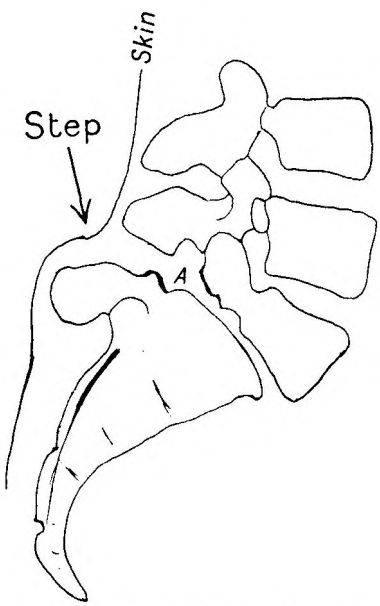


FIG. 490.—Spondylolisthesis at the level of L5 — S1 disc. Note the defect at A and the step over S1 spine.

In general, the main symptoms are usually those of long-standing low-back pain. On examination, in a mild case nothing will be found other than some excessive prominence of the first sacral spinous process. In a severe case, where the lumbar vertebra has become dislocated in front of the sacrum, there will be a severe lordosis and a rather characteristic shortening of the trunk, so that the lower ribs seem to rest on the brim of the pelvis.

Though trauma was frequently regarded in the past as a common cause of spondylolisthesis, accurate X-ray studies now show that a congenital defect in the development of the pedicles is much the commonest explanation. X-ray studies often reveal a condition sometimes known as ' prespondylolis-

thesis', or 'spondylolysis', where slipping of the vertebræ is absent, or minimal, in the presence of defects in the pedicles.

The majority of these cases never require any special treatment other than simple placebos for backache, such as local heat and rest from time to time. A stiff lumbosacral corset will give relief to the majority, but a few will require lumbosacral fusion to abolish pain. As in the management of any case of 'low-back pain', it is unwise to alarm the patient with injudicious reports about the X-ray appearance. Many cases of severe spondylolisthesis go through life with nothing more than an occasional attack of 'lumbago'. Injudicious remarks can convert a mild case into a complete invalid!

LUMBAR PUNCTURE

This procedure is adopted for the following reasons:

1. **Diagnostic,** e.g. meningitis, Wassermann reaction or gold test for syphilis; and to disclose intradural hæmorrhage. Also for the injection of pantopaque, or to assess the protein content, if a tumour is suspected. Manometric readings are essential to assess the intradural pressure (p. 499).

2. **Therapeutic,** in order to introduce some drug, e.g. penicillin for meningitis.

3. **Anæsthesia.**—Nupercaine, Novocaine.

Nupercaine is used in a 1:1,500 solution in 0·5 per cent. saline. The specific gravity of this solution is 1003, which is lighter than cerebrospinal fluid (sp. gr. 1006). The average dose is 14 ml., which gives complete abdominal anæsthesia, but 10 ml. is usually sufficient for operations below the umbilicus. After injection the patient lies on his face for five minutes so that the posterior roots are chiefly affected. He is then turned on his back, and slight tilting downwards of the head of the table will help to fix the Nupercaine caudalwards and counteract any fall of blood pressure.

Catastrophes, such as incontinence of urine or damage to spinal nerves, occasionally occur. These calamities are possibly due to an invisible crack in a phial, which, if submerged in spirit or other antiseptic, may allow some of the fluid to percolate into the interior. *Phials should always be submerged in coloured fluid*, and any phial which contains coloured contents is rejected. Otherwise autoclave all ampoules.

Some premedication, such as omnopon and scopolamine, is safe and humane in the absence of respiratory depression.

Technique.—At birth the cord extends to the third lumbar vertebra. In the adult it terminates opposite the lower border of the body of the first lumbar vertebra, below this level the dural sac can be safely punctured. A space of 2·5 mm. in width exists between the caudal equina nerve roots on either side and hence, if the lumbar puncture needle is kept strictly in the mid-line, it will avoid contact with the roots. The patient lies on his side with the spine well flexed in order to open up the spaces between the neural arches. Scrupulous asepsis is required. The skin must be fully prepared and isolated with sterile towels, gloves and gown being worn in order to perform the puncture. A line connecting the highest parts of the iliac crests passes over the fourth lumbar spine (fig. 491). The interspace above or below this spine is selected, and this area is infiltrated deeply by injecting local anæsthetic into the interspinous ligaments. The depth to which the needle will need to pass varies according to the build of the patient, from a

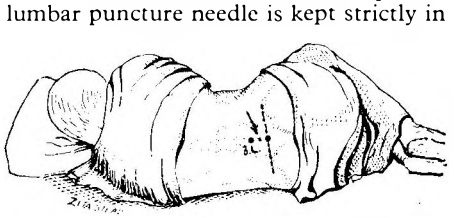


FIG. 491.—Lumbar puncture. (The site of puncture is usually between the third and fourth lumbar spines.)

few cm in a small child to 10 cm in a fat adult. In the case of some nervous children or patients afflicted with tetanus, general anæsthesia is necessary. The needle used should be of medium bore (about 18 to 20 gauge) as the aperture resulting from puncture with a wide needle will allow continuing escape of cerebrospinal fluid from the dura, with consequent persistent headache, produced by intracranial hypotension. The bevel of the needle must be short. As the puncture is commenced, the position of the needle should be checked, making sure that it is introduced at right angles to the skin and is not sloping to one side. This is best judged by looking along the skin of the patient's back; it should also be slanted slightly towards the patient's head so that it will enter obliquely beneath the lumbar spines. As the needle and stylet are introduced, the resistance of the tough ligamentum flavum is encountered, a little extra pressure perforates this structure, and on withdrawal of the stylet cerebrospinal fluid should flow from the needle. If only a few drops of blood escape, in all probability the point of the needle is lying in the spinal canal outside the dural sheath and the blood is escaping from a spinal vein, in which case the needle should be advanced slightly further. When the theca is entered, a manometer is attached to the needle in order to estimate the cerebrospinal fluid pressure, the normal being 120 mm of water in the recumbent position. In cases of raised pressure only a small quantity of fluid must be removed for investigation, and the pressure should not be reduced, even as low as the normal 120 mm, owing to the risk of causing midbrain pressure cone, or a medullary cone in the foramen magnum with fatal consequence.

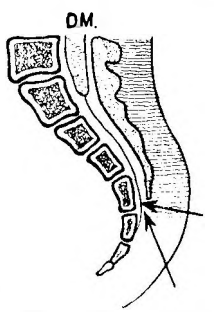


FIG. 492. — Indicating direction of needle.

SACRAL PUNCTURE (EPIDURAL)

The sacral epidural space may be entered through the large triangular opening on the dorsum of the bone, and sacral anæsthesia obtained by the injection into the space of 20 ml. of 1 per cent. Novocaine. Operations on the lower bowel and anal region can then be performed painlessly, and some obstetricians practise this method in order to relieve the pains of child-birth, an additional advantage being that the infant is unaffected.

To perform sacral puncture the median crest of the sacrum is palpated until the depression caused by the sacral opening is recognised. A lumbar-puncture needle is introduced directly forwards until the ligaments covering the opening are pierced, after which the direction of the needle is changed so that it passes vertically upwards within the sacral canal (fig. 492). In order to ensure that the point of the needle is within the epidural space, it is wise to inject a few millilitres of normal saline; if the needle has not penetrated the canal, infiltration of the subcutaneous tissues will be evident, in which case it is withdrawn and a fresh attempt is made.

CHAPTER 28

NERVES

CRANIAL NERVES

THE **Olfactory Nerve** may be injured by fractures of the cribriform plate, resulting in partial loss of smell (hyposmia) or anosmia on the corresponding side. Olfactory filaments may be ruptured as a result of anterior displacement following blows on the back of the head (*contre-coup*, p. 425).

The **Optic Nerve** may be damaged by fractures involving the optic foramen or by contusion against the margin of the foramen. Involvement by tumours or aneurysms is not uncommon. Primary optic atrophy with partial or complete blindness results in the affected eye, but contraction of the pupil will occur if the opposite retina is stimulated. The optic nerve is an outgrowth of the brain and gliomas arise in its substance, particularly in young children.

The **Third Nerve** is involved by tumours, trauma, or aneurysm in the skull, sphenoidal fissure, or orbit. Pressure on the nerve above the tentorium occurs in the early stages of midbrain pressure cone formation causing a dilated pupil. Partial lesions may only produce a dilated pupil, but in complete lesions the following features are noted.

(*a*) Ptosis of the upper eyelid, owing to paralysis of the levator palpebræ superioris.

(*b*) Proptosis, owing to paralysis of the majority of the ocular muscles, which normally exercise traction on the eyeball ; this will be increased if an intraorbital tumour is also present.

(*c*) Mydriasis, as the sympathetic fibres are unopposed, and cause unhampered dilatation of the pupil.

(*d*) Loss of accommodation, owing to paralysis of the ciliary muscle.

(*e*) Diplopia and external strabismus, with a slight downward inclination of the eye-ball due to unopposed action of the external rectus and superior oblique muscles. Owing to their proximity, other nerves passing to the orbit are often affected.

The **Fourth Nerve** supplies the superior oblique muscle, and is rarely involved alone. Diplopia and deficient movement of the eye in a downward and outward direction may be observed.

The **Fifth Nerve** or its branches are sometimes injured, and sensory disturbances follow.

Trigeminal Neuralgia.—This distressing condition occurs predominantly in the middle-aged and elderly, in women more than men, and is characterised by sudden very severe pain in the distribution of the trigeminal nerve; it is usually restricted to one or two divisions. The pain is often precipitated by exposure to cold, touching certain points on the face (trigger points), eat-

ing, talking and even walking. The pain is so severe that the patient ' screws up ' the affected side of the face and the hand comes up in a protective gesture; hence the condition acquired the name ' tic douloureux '.

In the majority of patients no cause is found. Very rarely it may be symptomatic of a cerebello-pontine angle lesion (e.g. acoustic neuroma), and onset at a younger age is suggestive of multiple sclerosis.

Treatment is initially by drugs, of which Carbamezapine (Tegretol) is currently the most effective. However if this fails surgery may be indicated. Prior to surgery it is essential to establish that the symptoms are those of true trigeminal neuralgia; in other types of facial pain surgery may well aggravate the condition. The methods used are:—

1. *Peripheral neurectomy*—supra-orbital or infra-orbital.—These procedures rarely bring lasting relief.

2. *Open surgery.*—(*a*) *Middle fossa approach* (fig. 493). The Gasserian Ganglion is approached subtemporally either extra or intradurally. By differential section it is possible to spare the ophthalmic division and therefore avoid the complications of anæsthesia affecting the surface of the eye.

(*b*) *Posterior fossa approach.*—By this approach the sensory root of the fifth nerve is divided in the cerebello-pontine angle as it runs towards Meckels cave. By micro-surgical techniques it may be possible to preserve touch sensation but abolish pain.

Both these operative approaches are major procedures which will carry some element of risk particularly as many of these patients are very elderly.

3. *Trigeminal Ganglion Alcohol Injection.*—This long-established method involves the insertion of a needle 1 cm below the zygomatic notch upwards, backwards and inwards for 5–6 cm through the foramen ovale to enter the ganglion or the CSF surrounding the ganglion. The position of the needle is checked by radiography and a trial injection of local anæsthetic (resistance being felt if the needle is in the nerve and ganglion) causing loss of sensation in one or more of the sensory divisions. Then 4–5 ml absolute alcohol is injected. This method is effective, provided the patient will accept the consequent numbness and is able to take precautions for the care of eye, skin and mouth.

4. *Electro-coagulation of the trigeminal ganglion.*—In recent years the technique of inserting an electro-coagulating probe through the foramen ovale into the trigeminal ganglion has been developed. With this it is possible to produce a staged or restricted lesion in terms of the distribution of the sensory loss and the modalities involved.

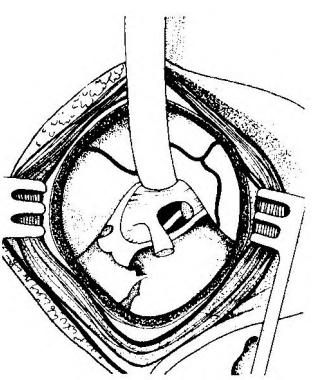


FIG. 493.—Fractional section of the lower and outer two-thirds of the sensory root at the apex of the petrous. The motor root is visible behind the cut portion of the sensory root.

The **Sixth Nerve** is slender and has a long intracranial course. It may be affected by fractures of the base of the skull or displacement of intracranial structures, or it may be involved in association with other ocular motor nerves by lesions in the cavernous sinus, sphenoid fissure, or orbit. The external rectus muscle is paralysed, and internal strabismus results.

The **Seventh Nerve** is involved by a variety of causes.

1. *Intracranial.*—Lesions within the brain are supranuclear, nuclear, or infranuclear. Supranuclear lesions involve only the lower half of the face, as the occipito-frontalis and orbicularis palpebrarum muscles enjoy bilateral innervation. In nuclear lesions the whole face and the sixth nerve on the same side are affected and also the opposite arm and leg, as the motor decus-

Johann Ludwig Gasser, died 1765. Professor of Anatomy, Vienna. His pupil, Raymund Balthasar Hirsch, discovered the ganglion and named it after his teacher.
Johann Friedrich Meckel (the elder) 1724–1774. Professor of Anatomy, Botany and Gynæcology, Berlin.

sation takes place at a lower level. An infranuclear lesion occasionally results from pressure of a tumour, e.g. of the cerebello-pontine angle, in which case involvement of the auditory nerve is also evident.

2. *Cranial.*—The intraosseous portion of the facial nerve may be affected by fractures of the base (fig. 494) and middle ear disease. Following fracture paralysis may appear immediately as a result of direct injury to the nerve or within hours or days from hæmorrhage within the nerve sheath, in which latter case recovery is possible. Paralysis occurring after some weeks is produced by the pressure of callus and is unlikely to recover. Facial paralysis is a complication of middle ear disease and may follow injury at operation on the mastoid antrum. Compression within the aqueduct of Fallopius occasionally follows chronic inflammation.

3. *Extracranial.*—The facial nerve or its branches may be injured outside the skull. The nerve is commonly involved by Bell's palsy, which is due to herpetic neuritis and follows exposure to cold or draught. Swelling within the sheath of the nerve extends into stylomastoid foramen, and the nerve is compressed within the bony canal. Absorption of exudate usually occurs before the pressure has damaged the nerve permanently, but in about 3 per cent. of cases complete paralysis remains, and in 5 to 10 per cent. some degree of paralysis persists. Tetanus arising from a wound in the distribution of the facial nerve sometimes causes paralysis, the cause being similar to that described above. Malignant tumours of the parotid gland involve the facial nerve, and paralysis is an important diagnostic sign distinguishing simple from malignant tumours.

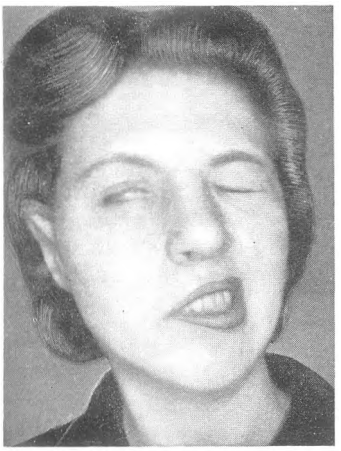


FIG. 494.—Right-sided facial paralysis following fracture through the middle fossa.

Branches of the facial nerve are injured either accidentally, e.g. by broken windscreens, or by ill-placed operation incisions. For operating on the parotid, the main trunk of the nerve must first be located and then branches followed forward (p. 583).

The paralysed face is flat and expressionless. The eye cannot be closed, and attempts to do so result in the eyeball being turned upwards and outwards (fig. 494). Corneal ulceration may follow from exposure. Epiphora occurs owing to drooping of the lower eyelids. Whistling is impossible, as the cheek merely flaps, and food collects between the gums and cheek.

Treatment is directed to the cause. To promote recovery the angle of the mouth is supported by a malleable rod covered with rubber tubing, which is bent like an 'S' so that the upper curve will hook around the ear and the lower will elevate the angle of the mouth. Small strips of adhesive strapping applied under tension also form a very convenient method of preventing-overstretching of the facial muscles. If considered advisable, an intra-oral

Gabriel Fallopio, 1523–1563. Professor of Anatomy and Surgery, Padua, Italy.
Sir Charles Bell, 1774–1842. Surgeon, Middlesex Hospital, London (1812–1835). Professor of Surgery, Edinburgh (1836–1842).

splint can be fashioned from plastic material. Electrical treatment and massage are prescribed, and during recovery the patient should practise facial movements with the aid of a mirror. Hypoglossal anastomosis may be considered in hopeless cases (p. 510). Repeated facial tic with spasmodic contracture of the facial muscles may be relieved by fractional division of the branches of the facial nerves which supply the affected muscles, these branches being exposed through a curved incision anterior to the lobe of the ear.

In cases of Bell's palsy early decompression of the nerve may be achieved by cortisone (60 mg. daily for 3 days, then 20 mg. for 3 days).

Facial tic.—An embarrassing condition in which repeated spasm of the facial muscles produces repeated winking or twitching of the face according to the muscles affected; may be relieved by partial section of branches of the facial nerve supplying the affected areas.

The **Eighth Nerve** may be involved in fractures of the middle fossa, or compressed by a tumour, e.g. of the auditory nerve sheath, causing unilateral deafness (p. 558). Vestibular functions are sometimes impaired (see Meniere's disease, p. 558).

The **Ninth Nerve** is occasionally injured by a fractured base. Some dysphagia may occur from paresis of the constrictor muscles.

Glosso-pharyngeal neuralgia is characterised by severe explosions of pain either in the region of the tonsil or deeply in the ear. The ' trigger ' zone is in the tonsillar area, and the diagnosis is clinched by the fact that cocainisation of the zone temporarily relieves the condition. In genuine cases the nerve must be divided. It is approached either through the posterior fossa and severed as it enters the jugular foramen, or as it crosses the tonsillar fossa after tonsillectomy.

The **Tenth Nerve** may be damaged in association with a fractured base, or crushed by a ligature which includes it as well as the internal jugular vein or common carotid artery. In doubtful cases examination of the laryngeal muscles will reveal recurrent laryngeal nerve involvement.

The recurrent laryngeal nerve may be damaged in operations on the thyroid gland (p. 633) or ductus-arteriosus by traction or post-operative extravasation. Complete paralysis may follow division or inclusion by a ligature at operation, infiltration by neoplasm of the thyroid gland or secondary lymph nodes from carcinoma of the lung, and on the left side by pressure of an aneurysm of the aortic arch. Partial involvement affects the abduction fibres, which are more susceptible than those which supply the adductor muscles, and thus if partial involvement is bilateral, stridor results, and tracheostomy may be required. Complete involvement results in paralysis of both abductors and adductors, and consequent paralysis of the corresponding vocal cord in the half-way or ' cadaveric ' position. The opposite vocal cord increases its range of mobility, so that it reaches across the mid-line and closes the glottis. The voice is adequate but somewhat monotonous.

Hysterical aphonia is emotional in origin and is distinguished from true paralysis by the fact that, although the patient can only whisper, coughing is readily performed on request.

The **Eleventh Nerve** is rarely damaged by fractures but is more commonly

injured during high cervical dissections, particularly in the removal of tuberculous lymph nodes which often entirely surround it. The nerve passes downwards and backwards at right angles to the centre of a line connecting the angle of the jaw and the mastoid process, and emerges from the posterior border of the sternomastoid muscle at the junction of the upper third and lower two-thirds; it then passes across the posterior triangle, and disappears under cover of the trapezius muscle. Division of the nerve in the anterior triangle usually produces complete paralysis of the sternomastoid muscle, but the motor supply contributed from the second and third cervical roots is sufficient in a third of the cases to prevent a complete paralysis of the sternomastoid. Division will result in only a partial paralysis of the trapezius as the trapezius receives a sufficient additional supply from the third and fourth cervical nerves. If injury occurs in the posterior triangle, the trapezius alone is affected. On inspection, drooping of the shoulder is seen, and wasting of the trapezius is obvious (fig. 495). Contraction of the sternomastoid muscle is tested by asking the patient to turn his head to the side opposite to the muscle. Palpation detects the rigid band of muscle if contraction is normal. Injury of the nerves to the trapezius results in inability to continue elevation of the arm after it is abducted to a right angle by the deltoid muscle. If the branches to the muscle from the third and fourth cervical nerves are intact, about 20 degrees elevation from the right-angled position is possible.

FIG. 495.—Drooping of the left shoulder and wasting of the trapezius muscle following division of the spinal accessory nerve. The swellings on forehead and face are soft fibromas—a common condition after middle age.

If division of the spinal accessory nerve is recognised at operation, primary suture should be performed. Secondary suture is not successful on account of retraction of the ends and difficulty in identifying them in scar tissue.

The **Twelfth Nerve** escapes in fractures of the base of the skull, as the anterior condyloid foramen is protected by a bony ridge which diverts a fissure towards the foramen magnum. It may be injured in removal of the submandibular salivary gland. Hemiatrophy of the tongue occurs, the corresponding side of the tongue being shrivelled and wrinkled, the tongue being pushed towards the paralysed side on protrusion (fig. 496).

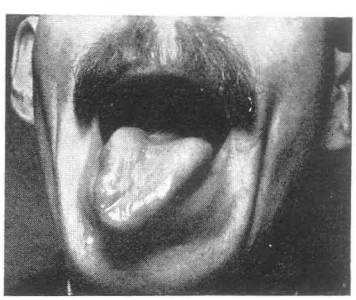


FIG. 496.—Hemiatrophy of the right side of the tongue following involvement of the hypoglossal nerve by syphilitic basal meningitis.

PERIPHERAL NERVES

Injuries of nerves are classified according to the extent of the damage to the nerve fibres and sheath, as neurapraxia, axonotmesis, and neurotmesis.

NEURAPRAXIA

is the equivalent of concussion. There is physiological paralysis of conduction in the intact nerve fibres as the result of stretching or distortion without any organic rupture. Neurapraxia is produced by minor stretch injuries, or by the concussion and vibratory effect of a high-velocity missile passing near a nerve without contacting it. Fibres remain intact within the intact sheath. It produces sensory loss, paraesthesiæ, and weakness of muscle groups lasting for days. During this time there is no reaction of degeneration in the muscles. There is no degeneration of the axons. When the power of conduction returns, all functions, motor and sensory, return together, sensation recovering in the whole limb within a period of hours. Recovery is complete.

Treatment consists of splinting the limb in a position of relaxation of the paralysed muscle groups until spontaneous recovery occurs.

AXONOTMESIS

consists of the intrathecal rupture of nerve fibres within an intact sheath. According to the cause, the nerve fibres are damaged to a variable degree (see below). Wallerian degeneration occurs in the distal portion of the broken axons, leaving an empty tubule. Intraneural fibrosis occurs at the sites of axonal rupture and minute intraneural hæmorrhages. Recovery takes place slowly by the proliferation and down-growth of axons into the distal tubules, the combined bulk of the proliferating axons and intraneural fibrosis producing a fusiform neuroma on the course of the nerve. There is some loss of nerve fibres owing to the blockage of the down-growing axons by the intraneural fibrosis, but since the relative position of axon and distal tubule is preserved by the intact sheath there is little maldistribution, hence the quality of regeneration is often good except in the case of progressive fibrosis. Recovery is delayed until the down-growing fibres reach their appropriate endings, and occurs first in the muscle groups nearest to the site of division and lastly in the peripheral skin areas, where the anæsthetic area begins to decrease steadily from the margin inwards. The length of time required for the recovery depends upon the level of the lesion. After an initial delay of approximately ten days, down-growing axons proceed distally at a rate of approximately 1 mm. a day, and on arrival at their endings there is a further delay of three weeks before the end organs become activated. Partial or complete intrathecal rupture of fibres may complicate a single stress occurring in association with fractures or dislocations, or excessive vigour during attempted reduction of such injuries; it also accompanies traction injuries at birth and may be produced by clutching for support when falling, as in brachial plexus lesions. It may follow contusion of a nerve with extensive hæmorrhage into its sheath or be the result of severe and acute compression by tourniquets, splints, or incorrect posture on the operating table.

Gradually increasing lesions are produced by progressive compression from crutches, splints, callus, and scar tissue or by repeated minor stretching of a nerve which becomes fixed near a mobile joint. The latter is seen

Augustus Volney Waller, 1816–1870. General Practitioner, Kensington, London (1842–1851). Subsequently worked as a physiologist at Bonn, Paris, Birmingham and Geneva.

when a nerve such as the external popliteal becomes fixed at the head of the fibula, or the ulnar nerve becomes fixed behind an arthritic elbow or stretched by cubitus valgus; in such cases movements of the joint pulling on the fixed nerve produce repeated minor ruptures at different levels, each of which is succeeded by a small degree of fibrosis, which gradually induces a progressive fibrosis and loss of function within the nerve.

Clinical Features.—Following a severe stretch, there is an initial picture of neurapraxia with widespread loss of sensation, tone, power, and reflex activity in the limb; this is followed by an incomplete recovery. The resolution of concussion will restore sensation and movement to certain areas of the limb to which the nerve fibres remain intact. The numbness and paralysis will persist in those areas to which the fibres have actually ruptured. Usually the total area affected is less than the known anatomical distribution of the nerve as a proportion of fibres have escaped.

Treatment of closed injuries of nerves consists in maintaining the nutrition of the limb and combating, by suitable means, the secondary pathological changes which would otherwise follow nerve injury, in order to maintain the muscles and joints in good working order pending the arrival of down-growing axons.

Secondary pathological changes accompanying nerve injury affect the skin, muscles, and joints. Impaired circulation consequent on disuse renders the part blue and cold. The skin becomes thin, the nails brittle, and so-called trophic changes occur as a consequence of minor unrecognised trauma. Those muscles which are paralysed and flaccid will be overstretched by unopposed action of antagonist groups. Within three weeks the reaction of degeneration appears. The muscle fibres no longer respond to the rapid make-and-break of faradic stimulation owing to an increase in the duration of chronaxie which accompanies paralysis, but they will still respond to the slow make-and-break of galvanism. The polarity also changes so that A.C.C.[1] becomes greater than K.C.C.[2] If the muscle nutrition is not maintained by galvanic stimulation, muscle fibres degenerate and progressive fibrosis replaces inert fibres. Recovery is then impossible. Periarticular adhesions eventually form around the immobile joints and fix them in a contracted position. To combat these effects the part must be protected by warm padding to prevent injury, and splinted in a position of relaxation of the paralysed groups. The electrical reactions of the muscles are recorded and muscle movement is maintained by regular galvanic stimulation of all paralysed groups. All joints are put through full passive movements daily to prevent contraction. In the case of the hands, joint movements and exercise of the paralysed muscles are best maintained by the use of elastic splints in which strands of catapult elastic are attached to leather thimbles, which are inserted over the finger-tips and mounted on wire frames in positions corresponding to the paralysed tendons, so as to provide an elastic counter-pull to replace the normal contraction of the paralysed muscles against which the opponents can act. This not only ensures full normal movement of all joints, but will also permit partial use of the hand during recovery.

Considerable encouragement must be given to the patient during the long period of time required for restoration of function. Progress is carefully checked by taking regular records of the electrical reactions and skin sensitivity. Electrical reactions change back to normal in the proximal groups as the downgrowth of fibres proceeds. These changes must be recorded, together with diagrams of the skin sensation, in order to determine whether

[1] A.C.C. = Anodal Closing Contraction. [2] K.C.C. = Kathodal Closing Contraction.

normal recovery is taking place. Failure to recover, or regression after the initial recovery, is an indication for the local exploration of the injured site. It is occasionally necessary to deal with a perineural scar or remove an intraneural fibroma.

Movements produced by unaffected muscles must not mislead the observer. Thus, in a case of radial paralysis, the fingers can be extended by the interosseous and lumbrical muscles, provided that the hand is supported. Also vicarious movements may be performed by adjacent muscles, e.g. in the case of division of the median nerve, the adductors of the thumb, acting in conjunction with the extensor ossis metacarpi pollicis, can produce opposition of the thumb.

H. J. Seddon *et al.* have endeavoured to calculate the rates of nerve regeneration, basing their results on the following clinical data:

(1) Tinel's sign—the course of the nerve is lightly percussed with a patella hammer, from below upwards. A tingling sensation is experienced when the level of regeneration is reached.

(2) Measurement of the rate at which pain and touch sensibility return.

(3) Observation of the times at which the function of muscles returns at different levels from the injury.

It appears that regeneration occurs initially at about 2 mm. a day, but the rate diminishes as time passes, so that after about three months it has slowed down to about 1 mm. a day. (Other factors which influence the results of suture are discussed on p. 509.)

Compression of a nerve.—The first symptoms usually consist of paræsthesiæ, numbness, and tingling along the distribution of the nerve, associated with neuralgic pain. Complete paralysis and wasting of muscles are uncommon unless the initial compression has been prolonged and severe, but when the compressing cause is progressive, paralysis can occur later and may then necessitate operative measures.

Treatment is by removal of the cause, usually a tight splint or plaster, but may necessitate exploration of the nerve and the excision of perineural scar tissue, or mobilisation of the nerve from callus, following which a new bed is prepared for the nerve in adjacent muscles, the nerve itself being wrapped in a layer of tantalum foil.

Sensory symptoms are satisfactorily relieved by these steps, but if intervention is delayed too long intraneural fibrosis may lead to persistent motor disability.

Progressive fibrosis produced by fixation of the nerve has to be treated by nerve transplantation (p. 517); recovery may be complete or incomplete, according to the degree of fibrosis.

NEUROTMESIS

is usually produced by penetrating wounds and results in partial or complete division of the nerve sheath and fibres. Very rarely, complete rupture of nerves may occur in major brachial plexus injuries. In war and severe industrial injuries extensive soft tissue damage to muscle and skin, and associated fractures, greatly complicate the problems of repair.

Partial lesions produce a lateral neuroma of the nerve. Complete division produces a terminal neuroma on the end of the proximal segment. In the proximal portion of the divided axons, retrograde degeneration occurs as high as the first node of Ranvier. After an interval of ten days the axons

Sir Herbert John Seddon, 1903–1977. Professor of Orthopædic Surgery, Royal National Orthopædic Hospital, London.
Jules Tinel, 1879–1952. Physician, Hôpital Beaujon, Paris.
Louis Antoine Ranvier, 1835–1922. French histologist and pathologist.

begin to subdivide to produce an excess of end bulbs, which then commence to grow downwards. By this time, however, the gap between the divided nerve ends, which was at first filled with blood, has now been replaced by organising clot and fibrous tissue, which presents an almost impenetrable barrier to the down-growing axons. In the distal segment of the divided nerve, Wallerian degeneration of the axons occurs, but the cells of the sheaths of Schwann proliferate, forming a slight bulb at the commencement of the distal end from which sprouts of Schwann cells grow proximally into the plane of division, being drawn by chemotaxis towards the down-growing axons. A few axons may by this means succeed in entering the distal segment, producing there a deceptively encouraging Tinel's sign, but any spontaneous healing is minimal.

Even after accurate nerve suture the quality of regeneration is less perfect than in cases of axonotmesis owing to the wastage of axons in the scar tissue at the suture line, and mal-distribution of those fibres which reach the distal segment. The density of scar tissue at the suture line is increased by local sepsis and inflammation, and by tension at the suture site.

Mal-distribution of fibres is greatest in the case of mixed motor and sensory nerves, motor for fibres may then unite with sensory endings. It is also greatest in those motor nerves which supply a large number of small muscles; hence the quality of recovery is best in the case of a pure motor nerve, such as the radial, which supplies a few groups of large muscles concerned in coarse movement, and is worst in mixed sensory and motor nerves supplying a large number of small muscles concerned in fine movement, such as the ulnar or median nerves at the wrist.

Treatment is by suture under suitable conditions, in the absence of sepsis and in the absence of tension, when the nerve can be placed in a suitable bed. In war injuries where there has been considerable loss of surface tissue, preliminary plastic operations such as pedicle grafting must precede attempted repair of the nerve in order to provide a suitable covering and bed at the suture site.

Open wounds (p. 4). In tidy clean incised wounds immediate primary suture is the ideal treatment for a divided nerve. In untidy contaminated wounds, the nerve suture should be postponed until three or four weeks after the injury. If a divided nerve is encountered during excision of such a wound, the ends are approximated by one stitch of fine silk, which prevents retraction during the period of delay. On no account should an attempt be made to identify or scrutinise a nerve in the vicinity. If only one end of a nerve is seen, then a suture should be inserted so as to fix it to adjacent muscle or fascia. The advantages of early secondary suture over immediate primary suture in untidy wounds are as follows :

(i) Primary suture usually requires enlargement of the wound by further incisions, so as to mobilise the nerve in order to allow approximation of the ends without tension. As the wound is potentially infected, exposure of previously uncontaminated tissues should be avoided.

(ii) The normal nerve sheath is a delicate structure, which is easily torn

Theodor Schwann, 1810–1882. Professor of Anatomy, Louvain, 1839–48, and, thereafter at Liège. Original researches before the age of twenty-seven laid the foundation of Physiology of Nerve and Muscle. The first to deal with problems related to living matter on a purely physical and chemical basis, and to recognise the cell as the unit of living matter. Discoverer of pepsin, and role of living organisms in fermentation.

by the slightest tension, and accurate suturing is essential for the success of the operation. In addition, the sheath is often further weakened by longitudinal slits or tears. After about three weeks of the injury, epineural fibrosis occurs and the sheath becomes thicker and tougher, consequently the insertion of sutures and accurate coaptation of the nerve ends are greatly facilitated.

NERVE SUTURE

The two ends of the nerve are identified, the incision being prolonged sufficiently to expose the nerve well above and below the seat of injury, in such a position that its normal anatomical relations are not obscured by scar tissue. The two ends of the nerve are freed and ' freshened ' by means of a scalpel or a Bard-Parker knife. Scissors should not be used, as the nerve is crushed thereby. Slices are removed from the ends of the nerve until the projecting fibres are seen, and blood freely oozes from the cut surface. Apposition of the two ends is accomplished as follows:

(*a*) *Mobilisation.*—The two ends are dissected from surrounding structures, care being taken to preserve important motor branches. Branches can often be stripped from the parent nerve in order to facilitate mobilisation.

(*b*) *Posture.*—The limb being held in a relaxed position.

(*c*) *Transposition.*—The radial nerve is brought in front of the humerus, or the ulnar nerve in front of the internal condyle (p. 517).

(*d*) *Nerve Anchoring.*—If it is obvious that the two ends of a divided nerve cannot be brought together, on account of excessive loss of tissue or retraction, then the two untrimmed ends are approximated as closely as possibly by tension stitches, the position of the limb being such that approximation is facilitated. Subsequently the nerve is stretched by gradually straightening the limb, and at a second operation the two ends are brought together. In some cases adequate suture can be performed while the limb is flexed, and extension is gradually regained.

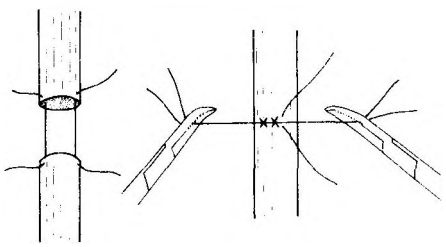


FIG. 497.—Primary nerve suture. Two sutures are passed through the sheath of the nerve in order to approximate the cut ends. The sheath is then sutured.

(*e*) *Resection of Bone.*—This extensive procedure may be justifiable if nerve injury is associated with an ununited fracture, which also needs operative measures, e.g. in the case of the radial nerve and a fractured humerus.

Approximation having been obtained by one of the above procedures, sutures are introduced through the nerve sheath (fig. 497). Non-irritating material is used, such as fine black silk or tantalum wire. Catgut encourages fibrosis of the nerve. The suture line can be further supported by painting with fibrin glue. Torsion of the nerve ends must be avoided, so that, as far as possible, proximal nerve fibres will join with their corresponding distal fibres. ' Shunting ' is thus avoided, and delay due to re-education of groups of muscles is obviated. If adjacent tissues are fibrosed, a new path can be constructed for the sutured nerve by opening a muscle sheath, and embedding the nerve among the muscle fibres. Finally, a layer of tantalum foil is always wrapped round the nerve to prevent epineural fibrosis and the out-sprouting of axons from the suture line, which may produce a painful local lesion. The limb is placed in a suitable position to prevent any strain on the sutured nerve, and a plaster cast may be advisable for a few weeks to immobilise the limb.

RESULTS OF NERVE SUTURE (see also Chapter 21)

This depends on many factors:

1. **Pre-operative.**—(*a*) *The Nerve Affected.*—Mixed motor and sensory nerves, e.g. ulnar and median, are subject to a greater degree of mal-distribution of downgrowing fibres than purely motor nerves supplying a small number of large muscles, e.g. radial.

Bard-Parker Inc., Danbury, Connecticut, U.S.A.

(*b*) *Infection* may cause delay in performing suture at the appropriate time and increases scar tissue formation following suture.

(*c*) *Time.*—Early secondary suture (p. 509) yields the best results, and further delay is detrimental.

(*d*) *Pre-operative Management.*—If muscles and tendons have been allowed to stretch or if the tone of the muscles has not been maintained, then the chances of recovery are correspondingly diminished.

2. **Operative.**—This consists of attention to the details already mentioned, e.g. hæmostasis, prevention of torsion and tension, preparation of suitable bed, and the use of non-irritating suture material.

3. **Post-operative.**—(*a*) *Absence of Infection.*—If the wound is already infected, or if infection supervenes, then little improvement is likely.

(*b*) *After-treatment* consists in continuance of the relaxation of paralysed muscles, massage, electrical treatment, and muscular effort gradually increased as muscles recover their power.

(*c*) *Co-operation of the patient.*—This important factor must receive due consideration and the patient given every encouragement during his rehabilitation.

(*d*) *Vicarious Movements.*—Although the physiological results of nerve suture may be poor, yet adjacent muscles often take upon themselves some of the functions of those which are paralysed, e.g. if the hamstrings are paralysed and flexion of the knee thereby affected, the sartorious and gracilis muscles hypertrophy, and partially compensate for this deficiency. Thus the functional result of a nerve injury is often more satisfactory than the physiological recovery would suggest.

Irremediable Injury.—If suture is impossible on account of loss of tissue or wide separation of the ends of a divided nerve, the following procedures may be considered :

(*a*) *Nerve anastomosis*, e.g. part of the hypoglossal nerve is united to the distal end of the facial nerve. This method often results in improvement, but ' successful ' cases are sometimes associated with uncontrolled grimaces on movement of the tongue.

(*b*) *Nerve grafting* has produced encouraging results in selected cases, e.g. a gap in the facial nerve within its body channel may be bridged by insertion of an autograft from the external cutaneous nerve of the thigh. Grafting of peripheral nerves is disappointing.

(*c*) *Tendon transplantation*, e.g. in the case of radial paralysis, tendons and muscles of the forearm may be transplanted into the extensor group. However, if proper relaxation of the extensor muscles has been consistently maintained, drop wrist should not occur, as the extensor tendons will not be over-stretched.

(*d*) *Arthrodesis*, e.g. in the case of injury to the sciatic nerve, arthrodesis of the flail ankle joint will render it stable and rigid.

(*e*) *Amputation*, for persistent sores and ulcers on the foot, particularly if growth is impaired. Sympathetic ganglionectomy might first be tried (p. 165).

Incomplete division of a nerve gives rise to a central or lateral neuroma. Effects vary according to the extent of the injury. Fibres supplying certain muscles are often constant in position, and hence are more liable to injury if their position exposes them to trauma. Thus partial division of the great sciatic nerve affects the lateral popliteal portion nine times more commonly than the medial popliteal, which passes down on the inner and deeper aspect of the great sciatic nerve.

Partial lesion of the median or medial popliteal nerves, or injury to their branches, may give rise to the distressing condition of *causalgia* (p. 519).

Injection lesions are due to accidental injection of therapeutic agents. They are more common in tropical countries where amœbiasis, schistosomiasis, and malaria are treated by intramuscular injections. The sciatic and radial nerves are usually involved. Injections in the buttock should always be given into the upper and outer quadrant (fig. 17) and in the case of the arm into the upper half of the deltoid muscle.

SPINAL NERVES

Injuries of the **Cervical Plexus** are uncommon, although muscular branches, e.g. to the trapezius and sternomastoid muscles, are occasionally damaged (p. 504).

The **Phrenic Nerve,** which arises from the third, fourth, and fifth cervical nerves, may be crushed in order to cause a temporary diaphragmatic paralysis and so reduce the size of the pleural cavity in such conditions as bronchiectasis or tuberculosis, and also to diminish movement after repair of the diaphragm for a hernia. Should the temporary paralysis produced by crushing prove to be insufficient, it is not difficult to recrush the nerve.

Paralysis of the diaphragm secondary to malignant disease in the thorax is equally frequent on the right and left sides and is often due to direct invasion of the phrenic nerve by adjacent growth. When phrenic paralysis is due to lymph node metastases, it is almost always due to involvement of a node which lies alongside the phrenic nerve at the level of the pulmonary artery at the upper region of the hilum.

Brachial plexus lesions are either complete or partial (fig. 498).

Complete lesions are rare, as an injury of sufficient severity to damage all the roots of the plexus will probably inflict fatal injuries on adjacent important structures.

In the event of a complete lesion, anæsthesia of the upper limb occurs except over areas supplied by the supra-acromial branches of the cervical plexus and the intercosto-humeral nerve. Complete paralysis of arm and scapular muscles occurs, except that in the case of tears of the plexus the lesion is usually distal to the site at which the nerve of Bell and the nerve to the rhomboids arise, and consequently the serratus magnus and rhomboid mucles escape.

Incomplete lesions, if due to stabs or cuts, are liable to affect any of the roots, the clinical features depending on the nerves divided. The commonest type of injury is due to traction or pressure, and affects either the upper or lower portions of the plexus.

Although the segmental innervation of the arm muscles is somewhat inconsistent, the following table summarises a distribution which is commonly accepted :

Nerve	*Muscles*
C.v . .	Rhomboids, spinati, deltoid, teres minor, biceps, brachialis, brachio-radialis, supinator brevis.
C.vi . .	Pectoralis major (clavicular head) and minor, subscapularis, coraco-brachialis, latissimus dorsi, teres major, serratus anterior, triceps, pronator teres, pronator quadratus.
C.vii .	The extensors of the fingers, extensor carpi ulnaris, and sternal part of the pectoralis major.
C.viii .	The flexors of the wrist and fingers.
D.i . .	The small muscles of the hand.

Upper Brachial Plexus Lesion (Erb-Duchenne, fig. 498 (1)). This injury is due to excessive displacement of the head, depression of the shoulder, or a combination of these two conditions. It affects infants after a difficult confinement or adults following blows or falls on the shoulder. The fifth and sometimes the sixth cervical roots are involved. In the former case the muscles affected are the biceps, brachialis, brachio-radialis, supinator brevis, spinati and deltoid, and thus the limb, internally rotated by the unopposed subscapularis, hangs by the side with the forearm pronated, in the well known 'tip' position (fig. 499). Sensory changes are absent if the fifth nerve only is involved, but if the sixth nerve also suffers, an area of anæsthesia is present

Wilhelm Erb, 1840–1921. Professor of Medicine, Heidelberg, Germany.
Guillaume Benjamin Amand Duchenne (Duchenne de Boulogne), 1806–1875. Neurologist, successively in Boulogne and Paris, but never held a hospital appointment.

over the outer side of the arm. Since the innervation of the hand is preserved, functional improvement is obtained by arthrodesis of the shoulder and elbow joints in suitable cases, together with transplantation of muscles from the pectoral groups to the humerus or scapula.

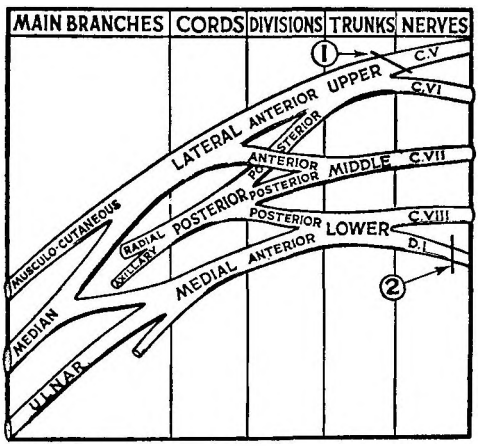


FIG. 498.—The brachial plexus, showing two classical sites for injury—see text.

Lower Brachial Plexus Lesion (Klumpke, fig. 498 (2)).—The lower nerve trunk or the inner cord are injured either above the clavicle, classically by inclusion with the subclavian artery in a ligature, or in the axilla, as by an unreduced dislocation of the humerus. In either case, the inner portion of the plexus is involved, and wasting of all the small muscles of the hand occurs, together with sensory loss along the inner side of the forearm and the inner three and a half fingers, owing to involvement of fibres passing to the ulnar and inner head of the median nerves.

FIG. 499.—Erb's paralysis.

Nerve roots may be avulsed from the spinal cord, as in the case of a falling person clutching at an object and hyper-abducting his arm, e.g. failing to obtain a foothold on a passing bus. The first dorsal root is usually affected. Paralysis of the intrinsic muscles of the hand results, with anæsthesia of the inner three and a half fingers in front, and inner one and a half behind. In addition, the oculopupillary fibres, which pass along the first dorsal nerve to the rami communicantes, and so to the cervical sympathetic, are also affected, and Horner's syndrome follows (fig. 146). Hæmorrhage sometimes occurs in the spinal cord following avulsion of the nerve root, and results in damage to the pyramidal tract and consequent spasticity of the leg on the same side.

Treatment.—Subcutaneous injuries are at first treated on expectant lines, paralysed muscles being relaxed. Thus, in the case of an Erb's paralysis, the arm should be fixed in a position of right-angled abduction and eversion, to relax the deltoid and spinati, the forearm being flexed and supinated in order to relax the biceps, brachialis, and supinators. In serious brachial plexus lesions, physiotherapy must be continued for two years before recovery is assessed.

Some minor improvement usually occurs during this time, especially if the initial loss of function has been due to neurapraxia and stretching rather than rupture of nerve fibres. When fibres have been ruptured, a great deal of

Madame Augusta Marie Dejerine-Klumpke, 1859–1927. Neurologist, Paris.
Friedrich Horner, 1831–1886. Professor of Ophthalmology, Zürich, described his syndrome in 1869.

mal-distribution of down-growing fibres among the complex plexus branches is bound to result in considerable reduction of functional efficiency. Where the nerve tracts are torn across (neurotmesis) little recovery is to be expected, and such cases are rarely improved by exploration of the brachial plexus.

BRACHIAL NEURALGIA

This condition (*syn.* brachial neuritis) is comparable to sciatica in the leg. The cutaneous distribution of the cervical nerves is illustrated in fig. 500, and the muscular innervation is summarised on p. 511. Some of the more important causes of brachial neuralgia are as follows:—

Prolapsed intervertebral disc (p. 493) is often difficult to distinguish from osteo-arthritis, but localised pain in the neck, which often appears suddenly and which is aggravated by coughing, suggests a disc lesion.

Osteoarthritis of the cervical spine (*syn.* spondylitis) causes neuralgia either as a result of pressure on a spinal nerve by an osteophyte or from absorption of the disc and consequent compression of the spinal roots. The onset is gradual and symptoms intermittent, and radiography usually establishes the diagnosis.

Costo-clavicular syndromes are due to many conditions. Sagging of the shoulder girdle, especially in middle-aged females, is a common cause, quite apart from cervical rib. Exercises and physiotherapy directed to improving the tone of muscles which elevate the shoulder may relieve the symptoms in such cases.

Spinal Tumour (p. 487).—Evidence of pressure on the cord is usually present, and Queckenstedt's test is commonly positive.

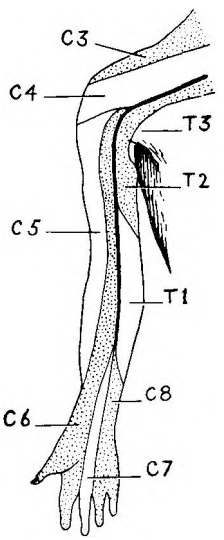


FIG. 500.—The dermatomes of the upper limb.

The **Circumflex or Axillary Nerve** passes through the quadrilateral space, and winds around the shaft of the humerus about one finger's-breadth below the centre of the deltoid muscle. It is sometimes injured by a direct blow, or involved by a fracture or dislocation of the humerus.

The deltoid muscle is paralysed and wastes rapidly, and a patch of anæsthesia over the outer side of the arm distinguishes this condition from a partial lesion of the fifth cervical nerve. Paralysis of the teres minor is unrecognisable clinically.

Recovery commences in a few weeks if the cause of the compression is removed, provided that the arm has been supported in right-angled abduction.

The **Nerve of Bell** (to serratus anterior) arises from the fifth, sixth and seventh cervical nerve roots. It may be injured by blows or carrying a heavy object on the shoulder, or during operations on the breast or chest wall, as it lies on the inner wall of the axilla. Paralysis of the serratus anterior allows 'winging' of the scapula, i.e. the vertebral border and inferior angle are unduly prominent (fig. 501). The 'lunge' stroke of fencing is dependent on the serratus anterior, and this and similar movements, such as pushing forward with the arm, are deficient. Owing to inability to rotate the scapula on the chest wall, difficulty is experienced in raising the arm above

Hans Queckenstedt, 1876–1918. (*See footnote p. 485.*)
Sir Charles Bell, 1774–1842. *See footnote p. 502.*

FIG. 501.—Winging of the scapula; the patient is pushing against a wall.

a right angle from a position in front of the body. Suture of the nerve is sometimes possible.

The **Radial** or musculospiral nerve is commonly injured. The classical sites are the axilla and the radial groove.

1. **Injury** in the **axilla** follows: (i) Crutch palsy: all crutches should have hand-grips, and their length should be carefully adjusted, especially if use is likely to be prolonged. Paresis has occurred after only four hours' use of crutches unsupplied with hand-grips.

(ii) Fractures and dislocations of the upper end of the humerus, or by attempts at their reduction.

(iii) Rarely by pressure of an aneurysm or new-growth.

Clinical Features.—(*a*) *Motor.*—The triceps and extensors of the wrist and fingers are paralysed, and consequently inability to extend the elbow, wrist, and fingers results, and wrist drop is present. If the hand is supported, as by supporting it upon the table, extension of the fingers can be produced by the action of the lumbricals and interossei, which are inserted into the extensor expansions. The supinator and brachio-radialis are also paralysed, but supination is ably performed by the biceps. The brachio-radialis muscle is tested readily by endeavouring to flex the semi-prone forearm against resistance. If the muscle is active, the contraction is visible, and the rigid muscle is easily palpable.

(*b*) *Sensory.*—Anæsthesia is present over the dorsum of the forearm and back of the hand where anæsthesia is reduced to a patch over the base of the thumb and first interosseous space owing to overlap of the musculocutaneous and other adjacent nerves (fig. 502). In peripheral nerve injury when sensory function is not restored, the final sensory loss is less extensive than the anatomical distribution of the nerve owing to the overlap of adjacent sensory areas.

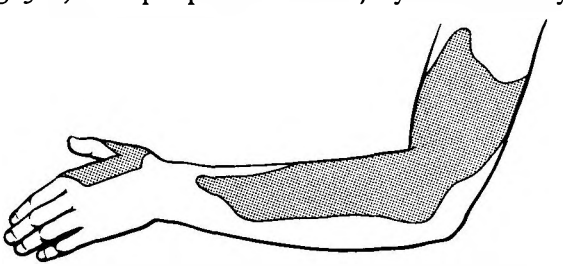


FIG. 502.—Areas of anæsthesia and anhidrosis following complete division of the radial nerve above the dorsal cutaneous nerve of the forearm and lower lateral cutaneous nerve of the arm. In lesions below the origin of the dorsal cutaneous nerve of the forearm the sensory loss is limited to the lower segment depicted over the dorsum of the base of the thumb and first interosseous space.

(*c*) *Trophic.*—These are usually trivial.

2. **Injury** in the **radial groove** is due to:

(i) Pressure, e.g. of the arm on the edge of the operating table, especially in Trendelenburg's position, or as in 'Saturday night' paralysis, due to the enjoyment of a heavy sleep with the arm over the sharp back of a kitchen chair. Prolonged application of a tourniquet is

Friedrich Trendelenburg, 1844–1924. See footnote p. 75. Described this position for pelvic operations in 1890.

especially liable to compress the radial nerve, as it lies close to the bone, and possibly the median and ulnar nerves as well. For this reason a sphygmomanometer should preferably be used on the arm.

(ii) Fracture of the shaft of the humerus when immediate injury of the nerve occurs in about 8 per cent. of cases. It is often overlooked owing to the more obvious fracture overshadowing the nerve injury.

(iii) The nerve being overstretched during operations on the humerus, e.g. in dealing with an ununited fracture.

(iv) ' Intramuscular ' injections of drugs being given into the radial nerve.

Clinical Features.—(*a*) *Motor.*—These are similar to those following injury in the axilla, except that the triceps and anconeus muscles escape.

(*b*) *Sensory.*—If the external cutaneous branch escapes, anæsthesia will be limited to a patch over the ball of the thumb. Division of the radial nerve in the upper third of the forearm is symptomless. Below this position the musculo-cutaneous nerve joins the radial, and division then causes anæsthesia over the ball of the thumb.

(*c*) *Trophic.*—These are slight.

The *posterior interosseous nerve* may be injured as a result of fracture or dislocation of the upper end of the radius, or in operations performed to deal with these conditions. Paralysis of the long extensors of the fingers results. The upper end of the nerve has been sutured with excellent results. If nerve repair is impracticable, good results are obtained by a tendon and muscle transplant.

The **Median Nerve** is classically injured at the elbow or wrist.

1. Injuries at the **elbow** are due to fractures of the lower end of the humerus or dislocations of the elbow joint. A tourniquet endangers the nerve at any level in the arm, in which case other nerves, particularly the radial, will also be involved.

Clinical Features.—(*a*) *Motor.*—The pronators of the forearm and flexors of the wrist and fingers, with the exception of the flexor carpi ulnaris and the inner part of the flexor profundus digitorum, will be paralysed. As a result of paralysis of the flexor carpi radialis, the hand deviates to the ulnar side when flexed against resistance. The index fingers cannot be flexed at the phalangeal joints—the ' pointing index '—but flexion of the other fingers is performed by that portion of the flexor profundus digitorum which is supplied by the ulnar nerve. Flexion of the terminal phalanx of the thumb is impossible, owing to paralysis of the flexor longus pollicis. The muscles of the thenar eminence are wasted and paralysed, and on inspection the eminence is flattened, so that the metacarpal bone of the thumb is apparently on the same plane as the other metacarpal bones—the so-called 'simian' or 'ape-like' hand. Paralysis of the two outer lumbricals is unrecognisable.

(*b*) *Sensory.*—Appreciation of touch is at first lost over the thumb and radial two and a half fingers in front, and posteriorly as far proximally as the middle of the proximal phalanges. Loss of response to pin-prick affects the terminal phalanges of the index and middle fingers, but sometimes a larger area is involved. Deep sensibility is lost over the terminal phalanges of the

index and middle fingers. Later this area is reduced by overlap from adjacent nerves.

(*c*) *Trophic.*—Obvious trophic changes are usually seen in the hand and affected fingers, particularly the index finger. Causalgia may complicate partial injuries (p. 519).

2. Injuries at the **wrist** are comparatively common, and are due to cuts from a variety of causes. Fractures of the lower end of the radius or dislocation of the semilunar bone sometimes cause injury to the median nerve.

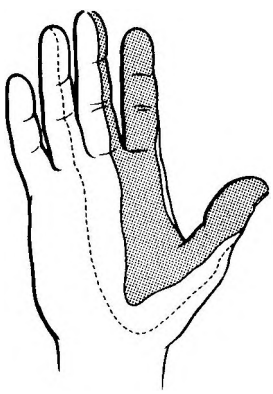


FIG. 503.—Area of persistent sensory loss in a complete median nerve injury above the wrist showing, in broken line, the distribution of the median nerve and, in stipple, the reduced area of anæsthesia, anhidrosis and analgesia finally persisting after injury owing to overlap of adjacent nerves. The ulnar nerve overlaps the ulnar side of the third finger.

Clinical Features.—(*a*) *Motor.*—The muscles of the thenar eminence are paralysed and wasted. The hand is 'simian', and abduction and opposition of the thumb are lost. Attempts to oppose the tip of the thumb to the tip of the little finger result in flexion and adduction of the thumb, as the patient is unable to swing it across the palm.

(*b*) *Sensory.*—Sensory losses resemble those following an injury at the elbow. Muscular sense is not impaired if tendons are not severed. Thus, as no striking muscular deficiency occurs, and as no part of the hand is completely anæsthetic, a divided median nerve at the wrist is readily overlooked, particularly in those who use refinements of sensation but little, e.g. a horny-handed labourer.

(*c*) *Trophic.*—These occur, as with an injury at the elbow.

The poor prognosis of a divided median nerve is rendered even more gloomy if tendons are also severed.

Median compression occasionally occurs as the nerve passes through the carpal tunnel (*entrapment neuropathy*). Paræsthesia, followed by wasting of the thenar muscles, suggests the diagnosis. A description to be found on p. 310.

The **Ulnar Nerve** is also classically injured at the elbow and wrist.

1. Injuries at the **elbow** are due to the following causes :

(i) Fractures in the region of the internal condyle.

(ii) Excision of the elbow joint.

(iii) Cubitus valgus, due to old injury of the humerus and increase of the 'carrying angle'. Hence the nerve is unduly stretched, and friction occurs as the groove on the internal condyle becomes a pulley, and this continuous friction results in interstitial neuritis. This condition may occur many years after the original injury, and transposition of the nerve is required.

(iv) Fixation of the nerve in the groove due to adhesions complicating osteoarthritis may lead to traction with resulting rupture of axons and progressive interstitial neuritis requiring transposition of the nerve (*entrapment neuropathy*).

Anterior transposition of the ulnar nerve is sometimes required for friction (axonotmesis), following fracture of the internal condyle, pressure by an osteophyte, recurrent dislocation of the nerve, or injury which results in loss of substance, so that approximation is thus rendered possible. The nerve is exposed by a curved incision with the concavity forwards, and the humeral head of the flexor carpi ulnaris is divided. Careful dissection is necessary so as to avoid injury to motor branches, and the internal intermuscular septum should be divided or excised, otherwise the nerve may be kinked by this structure when it is displaced forwards. A bed is then prepared in the flexor group of muscles and the nerve buried therein.

Clinical Features.—(*a*) *Motor.*—The flexor carpi ulnaris and inner portion of the flexor profundus digitorum are paralysed. Normally, on flexion of the wrist, the tendon of the flexor carpi ulnaris is readily palpable just above its insertion into the pisiform bone, but when the muscle is paralysed the tendon is impalpable, and wasting causes flattening of the inner border of the forearm. Weakness of the flexor profundus digitorum results in hyper-extension of the little, ring, and slightly of the middle fingers at the metacarpophalangeal joints.

Paralysis of the small muscles of the hand also results, with the exception of the thenar muscles and outer two lumbricals. Inability to abduct and adduct the fingers results, and the patient cannot grip a piece of paper placed between the fingers (fig. 504). If the patient pinches a piece of paper between his thumb and fingers the terminal phalanx of the thumb assumes a flexed position, as weakness of the adductor pollicis permits over-action of the long flexor of the thumb (Froment's sign) (fig. 505). Consider-

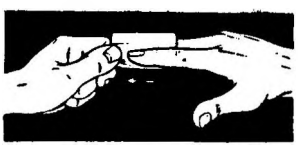


FIG. 504.—Test for weakness of the interosseous muscles.

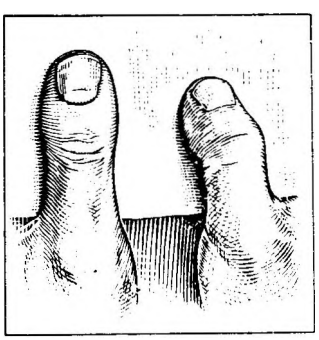


FIG. 505.—Froment's sign for right ulnar paresis.

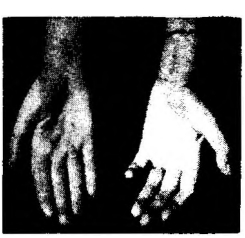


FIG. 506. — Wasting of the hypothenar eminence following injury to the left ulnar nerve.

able wasting occurs, which is obvious in the interosseous spaces and along the inner border of the hand, the normal curve being lost (fig. 506).

(*b*) *Sensory.*—The appreciation of light touch is at first lost over the inner one and a half fingers in front and behind. Response to pin-prick is lost over the little finger and ulnar border of the palm (fig. 507). Later this area will be reduced by overlap from adjacent nerves.

(*c*) *Trophic.*—These changes are usually well marked.

2. Injury at the **wrist** is due to the same causes as those enumerated in

Jules Froment, 1878–1946. Professor of Clinical Medicine, Lyons, France.

connection with the median nerve. The ulnar nerve passes in front of the anterior annular ligament, and is damaged by more superficial injuries.

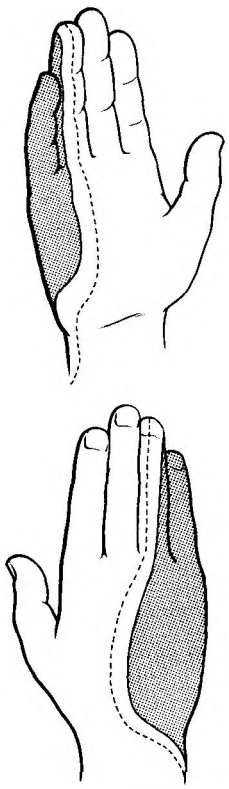


FIG. 507.—Persistent sensory loss in a complete ulnar nerve injury showing, in broken line, the anatomical distribution of the ulnar and, in stipple, the reduced area of persistent anæsthesia finally resulting.

Clinical Features.—(*a*) *Motor.*—Paralysis and wasting of small muscles of the hand, as described above.

(*b*) *Sensory.*—The dorsal cutaneous branch of the ulnar nerve leaves the main trunk about 6·3 cm above the styloid process of the ulna. Sensation is therefore lost only on the anterior aspect of the inner one and a half fingers.

(*c*) *Trophic.*—Correspond to the area of sensory loss.

The **Twelfth Dorsal Nerve,** as in the case of the intercostal nerves, is sometimes implicated by severe neuralgia, which may be associated with herpes zoster. More commonly the nerve is implicated by a suture during a kidney operation, or by subsequent scar tissue.

The **Ilio-inguinal Nerve** may be damaged on the right side in a gridiron incision for appendicectomy, although with care the nerve should be avoided. If drainage tubes are inserted through this incision, the resulting scar tissue may implicate the nerve. On the left side, injury to the nerve may follow iliac colostomy. Weakness of the conjoined tendon results, with consequent predisposition to the formation of an inguinal hernia.

The **External Cutaneous Nerve** is occasionally compressed as it passes through the deep fascia of the thigh, especially in muscular subjects, e.g. oarsmen. The condition is termed *meralgia paræsthetica*, and resection of part of the nerve is sometimes necessary to rid the patient of pain.

The **Sciatic Nerve** is occasionally injured by wounds, fractures, or 'intramuscular' injection of drugs. The component nerves in the pelvis may be involved by fracture, tumour, or aneurysm. Injury in the upper part of the thigh sometimes complicates deep wounds or posterior dislocation of the hip joint. If the lesion is above the origin of branches to the hamstrings, the following features will be present:

(*a*) *Motor.*—The flexors of the knee are paralysed, but some degree of flexion is possible owing to the action of the sartorius and gracilis muscles. Complete paralysis exists below the knee, and the pull of gravity therefore causes foot drop.

(*b*) *Sensory.*—Complete loss below the knee, with the exception of the skin supplied by the long saphenous nerve, i.e. a strip along the inner side of the leg extending along the inner border of the foot to the ball of the big toe.

(*c*) *Trophic.*—Especially on the sole of the foot and toes.

(*d*) Causalgia may complicate partial lesions (p. 519).

Partial involvement of the sciatic nerve affects the lateral popliteal portion nine times as commonly as the medial popliteal.

The **Lateral Popliteal Nerve** is injured as follows:

(i) Fracture or excision of the upper end of the fibula.

(ii) Pressure from plasters or splinters.

(iii) In operations for multiple ligation of varicose veins.

(iv) Pressure within the origin of the peroneus longus from the fibula (*entrapment neuropathy*).

(v) Subcutaneous tenotomy of the biceps tendon.

Clinical Features.—Complete lesions will cause:

(*a*) *Motor.*—Complete paralysis of the extensor and peroneal groups of muscles, with resulting talipes equino-varus.

(*b*) *Sensory.*—Anæsthesia of the outer side of the leg in its lower two-thirds, and of the dorsal aspects of all the toes, with the exception of the outer side of the little toe, which is supplied by the external saphenous (sural) nerve, as one contributory branch—the sural communicating from the medial popliteal nerve—escapes.

(*c*) *Trophic.*—Corresponding to the sensory loss. Moderate degrees of pressure will cause ill defined pain and variable degree of weakness or sensory loss.

The **Medial Popliteal Nerve** is but rarely injured on account of its protected position. The calf muscles and muscles of the sole are paralysed, and talipes calcaneo-valgus may result. The sole is anæsthetic, and trophic changes are usually severe. Causalgia (see below) occasionally follows a partial injury of the nerve.

AUTONOMIC NERVOUS SYSTEM

Indications for Operations.

1. **To Improve Circulation** (Sympathectomy, pp. 143, 146 and 165).
2. **To Relieve Secretory Disturbances.**

Hyperhidrosis (excessive sweating) can be very distressing both at work and socially. Sweating of the hands responds promptly to cervico-dorsal sympathectomy (p. 165). Sodden, offensive feet, the skin of which is cracked and painful, may be a genuine disability for which lumbar sympathectomy is justified. If the axilla is troublesome, relief follows the excision of that area of axillary skin which contains apocrine sweat glands. The area can be mapped out by applying a sweat-sensitive starch and iodine dusting powder. Alternatively these glands can be destroyed by curettage or excision by careful snipping away of all subcutaneous fat, inverting the skin through axillary incisions.

A **parotid fistula** (p. 589) may be encouraged to heal by avulsion of the auriculo-temporal nerve.

Vagotomy to decrease gastric secretion is described in Chapter 43.

Auriculo-temporal syndrome (p. 596).

3. **To Relieve Painful Conditions**

Causalgia is a condition in which paroxysmal attacks of pain follow an incomplete nerve injury, especially of the brachial plexus, sciatic or median nerves. In more than half the cases symptoms supervene immediately after the injury, in the remainder symptoms are deferred for any length of time up to two or three months. The incomplete lesion on the nerve, usually a lateral neuroma, gives rise to antidromic impulses which pass peripherally to the sensory nerve endings where stimuli give rise by the production of histamine-like substance (H substance) similar to that liberated to the posterior nerve root vasodilators. The accumulation of H substance causes vasodilation and renders the part red and engorged, the affected area also sweats profusely and is exquisitely painful and hyperæsthetic owing to the increased pressure. The skin is often thin as the result of trophic changes. Sooner or later the patient loses his morale and becomes introspective and unco-operative. The condition is often so distressing that surgical alleviation is desirable.

Treatment.—Paravertebral block (either D.2 and 3, or L.1, 2 and 3) relieves the pain for a short time, and confirms the necessity for operation (p. 143). Thoracic sympathetic trunk section or lumbar sympathectomy is the treatment of choice.

Visceral Pain.—*Pancreas.*—The wearying pain from recurring pancreatitis and carcinoma may respond to injection (see paravertebral injection, p. 143) of the cœliac and related sympathetic ganglia.

Uterus.—Certain cases of spasmodic dysmenorrhœa can be relieved by presacral neurectomy. No untoward effects on uterine (including parturition) or bladder function have been recorded. The bifurcation of the aorta is exposed by an incision through the overlying peritoneum. The presacral nerves are usually represented by a plexus, and in order to ensure complete removal, all the nervous, fatty, and areolar tissues lying over the lower inch of the aorta and the sacral promontory are excised.

CHAPTER 29

EYE AND ORBIT

PERIORBITAL AND ORBITAL SWELLINGS

A. Swellings Related to the Supraorbital Margin

External angular dermoid cyst.—The commonest situation for a dermoid cyst (fig. 508), and a swelling in this position is almost pathognomonic. The cyst often lies within a bony depression, caused by the constant pressure.

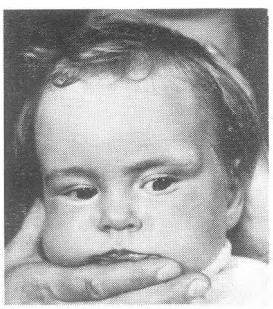


FIG. 508.—External angular dermoid.

FIG. 509. — Dermoid cyst at the root of the nose.

FIG. 510.—A mucocele of the lachrymal sac. (*Dr. Peter Hansell, London.*)

Some dermoid cysts may have a dumb-bell extension into the orbit, and may erode the orbital plate of the frontal bone to become attached to dura. X-ray is therefore important before excision.

Midline subcutaneous cyst.—If this does not vary on pressure, then it is almost certainly a dermoid cyst (fig. 509), but if it can be emptied on pressure, a meningocele or a communication with a dural venous sinus must be considered.

B. Swellings of the Lids or Lachrymal Drainage Apparatus

Meibomian cyst (chalazion) is a retention cyst of a tarsal gland, and is visible as a swelling on both skin and conjunctival surfaces of the lid; it may become secondarily infected. Treatment is by a vertical incision on the conjunctival surface, and curettage of the contents. This condition must be distinguished from a *Hordeolum* (stye), which is a pyogenic infection of an eyelash follicle and is self-limiting (p. 113).

Mucocele of the lachrymal sac is due to obstruction of the nasolachrymal duct, and gives rise to a swelling just below and medial to the inner canthus of the eyelid (fig. 510). Pressure on the swelling may cause mucous regurgitation back into the conjunctival sac. It is common in infants from delayed canalisation, and spontaneous resolution may be expected by one

year, but in adults surgical drainage into the nose is necessary; unless this is performed, infection may result in acute abscess formation requiring preliminary surgical drainage via a vertical skin incision.

C. Swellings Arising from within the Orbit: Proptosis

A localised or diffuse swelling arising within the orbit causes space-occupation, resulting in displacement of the eyeball. This is usually associated with some limitation of ocular movement, causing double vision. The commonest cause of proptosis is not however due to *neoplasia*, but due to *hyperthyroidism*, *vascular* and *inflammatory* conditions.

Hyperthyroidism.—Chapter 35.

Vascular causes of proptosis.—There are two conditions of the cavernous sinus which present with abrupt proptosis due to intense venous congestion of the orbital tissues. 1. An *arteriovenous fistula* may develop between the cavernous sinus and the internal carotid artery, either spontaneously or following a head injury, associated with a pulsating noise in the head. Vision may be jeopardised in both eyes from poor intraocular perfusion, but unless blindness threatens, conservative treatment is the safest procedure. 2. A *cavernous sinus thrombosis* presents with abrupt proptosis, visual impairment and oculomotor pareses and the patient is extremely ill. This condition may arise by septic emboli from a focus of infection in the territory of the facial vein, or may complicate meningitis. Intensive parenteral antibiotic therapy is required, but the prognosis is grave.

Hæmorrhage into the orbit may follow blunt trauma, but the history and associated lid bruising makes the diagnosis obvious.

Inflammatory causes of proptosis.—1. *Orbital cellulitis* is a common complication of acute sinusitis; this is accompanied by severe pain, discharge and lid swelling, and proptosis and double vision develop; marked constitutional symptoms are present (fig. 511). Initially the cellulitis must be controlled by intensive parenteral antibiotic therapy, but subsequently the underlying sinusitis may require surgical drainage. 2. In more chronic sinus infections, a localised abscess or mucocele may extend into the orbit, causing displacement of the eyeball. Surgical drainage is required. 3. In *granulomatous* disease, such as sarcoidosis or Wegner's granulomatosis, involvement of the lachrymal gland may cause proptosis, associated with other general manifestations (p. 196).

FIG. 511.—Orbital cellulitis.

Tumours of the Orbit.—These present with slowly developing proptosis, and the direction of the displacement of the eyeball gives some indication of the origin of the tumour.

Commonly, the orbit is secondarily invaded by *tumours arising from adjacent structures*, such as antral or ethmoidal carcinomas, sphenoidal ridge

meningiomas, or invasive rodent ulcers from the skin. *Orbital metastases* are rare, the commonest being from a neuroblastoma of the adrenal gland. Proptosis may be the presenting feature of *leukæmia* in children. *Primary orbital tumours* are rare; in children, the principal tumours are a *glioma* of the optic nerve (fig. 512), or an *osteogenic sarcoma* (fig. 513); the latter may follow irradiation to this region. In adults, a *mixed tumour* may arise in the lachrymal gland (fig. 514), resembling its counterpart in the parotid gland.

Treatment.—In all conditions presenting with proptosis, treatment is directed not only to the cause, but also to prevent damage to the cornea from

FIG. 512.—Glioma of the optic chiasma. (*J. Minton F.R.C.S., London.*)

FIG. 513.—Sarcoma of the orbit. (*Professor A. A. McConnell. Dublin.*)

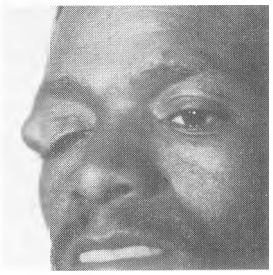


FIG. 514.—Mixed tumour of the lachrymal gland. (*Professor Charles Saint, Cape Town.*)

exposure, by liberal application of antibiotic ointment or artificial tear drops.

The treatment of orbital tumours is by excision where feasible, followed by irradiation for malignant lesions.

INTRA-OCULAR TUMOURS
- **Occurring in Children = Retinoblastoma**
- **Occurring in Adults = Melanoma**

Retinoblastoma.—This is a highly malignant undifferentiated tumour arising in the retina; the growth fills the eye, and becomes visible as a white pupil. It may spread by invasion of the optic nerve and meninges, or by bloodborne metastases. Most retinoblastomas are unilateral and sporadic, but in the less common hereditary form, both eyes are affected. Treatment is by enucleation or by radiotherapy.

Malignant melanoma.—This tumour commonly originates in the choroid, but it may arise from the ciliary body or iris; it presents as an elevated pigmented growth. Although initially intraocular, direct extension through the sclera may occur, or it may metastasise through the veins. However, growth of these metastases may be extremely variable, and can be delayed many years, hence the adage ' beware the patient with a large liver and a glass eye '. (fig. 515). The prognosis for an intraocular melanoma is more benign in the elderly, and for the more anteriorly sited lesion. In most patients, however, an intraocular melanoma is an indication for enucleation.

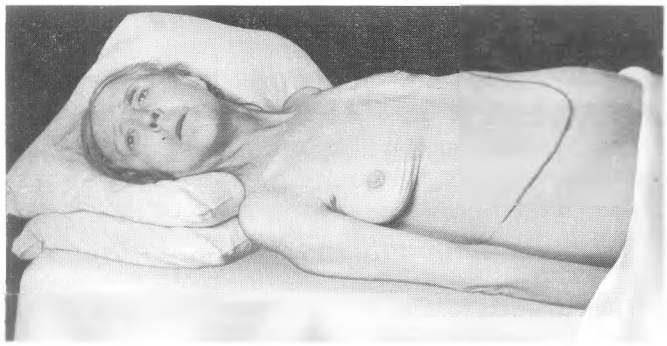


FIG. 515.—Patient with a greatly enlarged liver, who for many years had worn a glass eye after excision of the eyeball for melanoma.

INJURIES INVOLVING THE EYE AND ADJACENT STRUCTURES

Burns.—The commonest ocular burn is *radiational*, after an arc-welding flash; symptoms of intense pain and photophobia develop in both eyes some hours after exposure to the ultraviolet radiation, but healing rapidly occurs.

In *thermal* burns, rapid lid closure usually protects the eye itself, but full-thickness burns involving the eyelids will lead to scarring and deformity, resulting in corneal exposure, and early reconstructive surgery is indicated. A direct thermal burn of the eye follows a splash of molten metal, causing intense localised necrosis. Treatment consists of prevention of infection with antibiotic ointment, combined with atropine drops, and prevention of adhesions developing between the burnt eye and conjunctiva by daily separation with a glass rod. Scarring of the cornea may permanently impair vision.

Chemical burns are the most serious, particularly alkali burns, which can penetrate deeply into the eye and cause ischæmic necrosis. The most important treatment is immediate irrigation, which should be continued for 30 minutes before seeking expert advice. In lime burns, particulate matter may be trapped under the lids, requiring manual removal, followed by irrigation with a chelating solution. Subsequent treatment of chemical burns is along the lines indicated for molten metal burns, but the prognosis is worse.

FIG. 516.—Facial lacerations from windscreen injury: beware of a perforating eye injury.

Blunt Injuries.—A blunt injury may either cause hæmorrhage into the orbit, or the force may be transmitted to the orbital floor, resulting in a *blowout fracture* (fig. 516). Orbital contents may be trapped in the fracture, causing double vision. Crepitus of the eyelids from surgical emphysema confirms the communication of the orbit with the maxillary antrum, and X-rays may demonstrate blood and other structures trapped in the fracture. Spontaneous improvement may occur, but if double vision persists, the floor of the orbit requires exploration via a subperiosteal approach along the infra orbital margin.

Direct contusion of the eye may follow blunt injury, and the commonest manifestation of this is an *hyphaema*, blood in the anterior chamber (fig. 517). There is a risk of serious secondary bleeding within the first four days after injury, and complete bedrest for this period is mandatory. More serious damage to the intraocular structures may permanently affect vision.

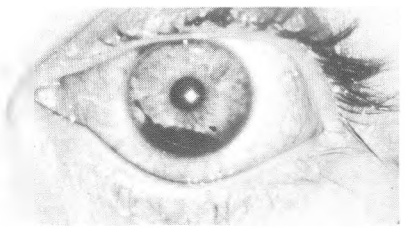


FIG. 517.—Hyphæma—blood in anterior chamber after blunt injury.

Lacerations.—It is important in any laceration involving the eyelids to exclude an underlying perforation of the globe: this is particularly true of multiple facial lacerations following an injury from broken glass, and any bleeding from beneath the lids should suggest the possibility of a perforating eye injury. In such an injury, prolapse of intraocular contents occurs through the wound, resulting in distortion of the pupil, loss of the anterior chamber, and intraocular hæmorrhage; damage to other structures may be present. This requires expert repair, and there is a risk of developing an autoimmune reaction in the uninjured eye that may lead to blindness; this is known as **Sympathetic Ophthalmia**. Prevention of this complication is by enucleating any injured eye that fails to improve rapidly after surgical repair, and this should be performed within two weeks of injury. Danger signs in the injured eye are failing vision, increasing irritability, and loss of intraocular pressure.

Note.—A commonly missed serious injury involves falling on a sharp object, such as a stick; the entry wound may not be very obvious, and the point may bypass the eyeball, and penetrate the orbital roof or superior orbital fissure, with ensuing brain damage, abscess formation or meningitis. Any such injury should be X-rayed and carefully observed. Similarly, a patient presenting with a history of a foreign body in the eye after using a hammer and chisel, or after an explosion, should always be X-rayed, as a minute steel or copper foreign body may penetrate into the eye without obvious external signs, and may lead to blindness if not removed.

EXCISION OF AN EYEBALL

Indications include a painful blind eye, intraocular neoplasms, and the risk of Sympathetic Ophthalmia.

The Operation.—The speculum is introduced between the lids, and opened. The conjunctiva is picked up with toothed forceps and divided completely all round as near as possible to the cornea. Tenon's capsule is entered, and each of the rectus tendons is hooked up on a strabismus hook and divided close to the sclera (fig. 518). The speculum is now pressed backwards and the eyeball projects forward. Blunt scissors, curved on the flat, are insinuated on the inner side of the globe, and these are used to sever the optic nerve. The eyeball can now be drawn forward with the forceps, and the oblique muscles, together with any other strands of tissue which are still attaching the globe to the orbit, are divided. A swab moistened with hot water and pressed into the orbit will control the hæmorrhage.

Evisceration of the Eyeball.—Owing to the danger of opening up lymphatic spaces at the back of the globe and thus favouring meningitis, evisceration is to be

Jacques René Tenon, 1724–1816. Surgeon, La Salpêtrière, Paris.

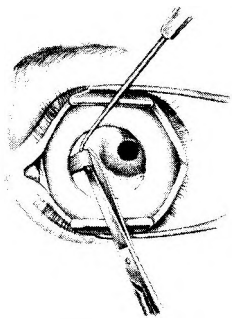


FIG. 518.—Excision of an eyeball. Medial rectus tendon being divided, aided by a strabismus hook.

preferred to excision in panophthalmitis. The sclera is transfixed with a pointed knife a little behind the corneo-sclerotic junction, and the cornea is removed entirely by completing the encircling incision in the sclera. The contents of the globe are then removed with a curette, care being exercised to remove all the uveal tract. At the end of the operation the interior must appear perfectly white.

DIFFERENTIAL DIAGNOSIS OF THE ACUTE RED EYE

The importance of this is in the recognition and treatment of minor ocular complaints, and those requiring expert attention, particularly acute glaucoma.

The four conditions presenting as an acute red eye are:—

1. Conjunctivitis
2. Keratitis (corneal ulceration)
3. Uveitis (inflammation within the eye)
4. Acute glaucoma (acutely raised intraocular pressure)

Conjunctivitis.—The symptoms are grittiness and discharge, and the conjunctiva is diffusely infected. It is serious in the newborn, when gonococcal infection may be present, but otherwise it is usually self-limiting, and will respond to antibiotic drops Only conjunctivitis is not painful and does not affect vision.

The following other conditions are all painful and may affect vision; both pain and visual loss are most severe in acute glaucoma, which should be suspected in an elderly patient. All are characterised by circumcorneal injection.

Keratitis.—In keratitis, minor conditions, such as inturned eyelashes or a subtarsal or corneal foreign body may be dealt with by removing the offending cause and applying antibiotic ointment, but *corneal ulceration*, which may be demonstrated by staining with a drop of fluorescein, is serious. Steroid drops should never be used, as a virus keratitis may be induced and aggravated.

Uveitis.—Uveitis may be recognised by the presence of an exudate in the anterior chamber and by irregularity of the pupil.

Acute Glaucoma.—In acute glaucoma, the cornea becomes hazy, and the eye feels rock-hard. The severe pain may be accompanied by such vomiting and prostration as to be mistaken for an acute abdominal emergency.

Corneal ulceration, uveitis and acute glaucoma require immediate referral for expert treatment, and the reader is referred to a specialist text.

CHAPTER 30

FACE. LIPS. PALATE. FACIO-MAXILLARY INJURIES

EMBRYOLOGY OF THE FACE

ABOUT the sixth week of fœtal life a depression appears at the front of the head. Around this depression, called the stomodæum or primitive mouth, five processes appear: a single one at the cephalic end, the frontonasal process, and on each side a maxillary and a mandibular process (fig. 519). Soon the frontonasal process becomes subdivided, by the appearance of the olfactory pits, into a solitary median nasal process and two lateral nasal processes. The median nasal process becomes bluntly bifurcated, forming the processus globularis. Maxillary mesoderm extensions grow medially beneath the olfactory pits to join in the mid-line, thereby forming the primitive palate and excluding the placodes (future nostrils) from the upper lip. Every congenital deformity of the face has existed from the ninth week of foetal life. The pinna is developed from six tubercles situated around the posterior end of the first branchial cleft.

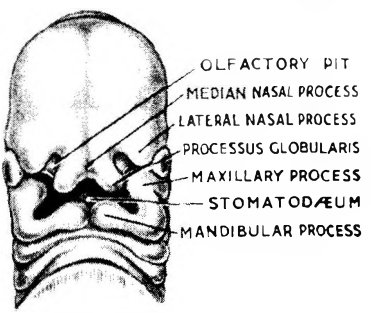


FIG. 519.—The head of an eight-week embryo.

CONGENITAL ABNORMALITIES

Cleft-lip and Cleft-palate[1]

Failure of coalescence of the developing processes (fig. 519) results in either (*a*) a cleft of the lip alone, (*b*) a cleft of the lip and the alveolus, (*c*) a cleft of the lip, alveolus, and palate, or (*d*) cleft-plate alone (figs. 520 and 521).

Possible causative factors.—Exposure to X-rays, cortisone intoxication, vitamin deficiency, and certain virus infections (e.g. rubella) during the first trimester of pregnancy, are all associated with an increased incidence of these abnormalities in the

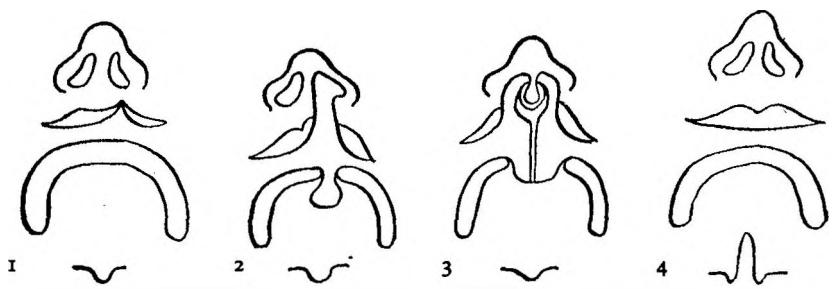


FIG. 520.—1. Partial cleft-lip. 2. Complete cleft of lip and alveolus (see fig. 523). 3. Double cleft of lip and alveolus. 4. Cleft soft palate. (*After Kernahan and Starke.*)

[1] Cleft-lip is also commonly known as hare-lip.

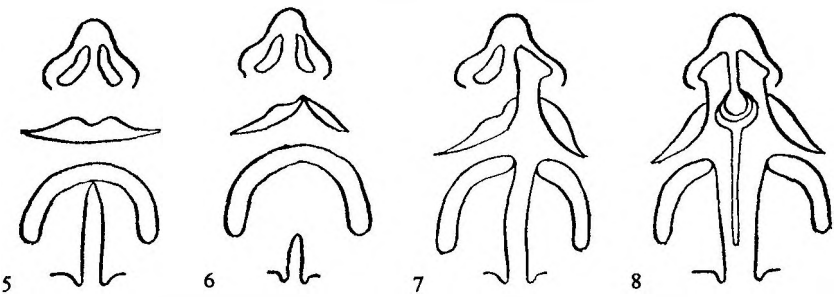


FIG. 521.—5. Cleft soft and hard palate. 6. Partial cleft of palate and lip. 7. Complete cleft of lip, alveolus, and palate. 8. Double complete cleft of lip, alveolus, and palate (see fig. 527). (*After Kernahan and Starke.*)

fœtus. In about 12% of cases the condition is familial. In 10% of cases other abnormalities are present, such as hydrocephalus, congenital blindness and mental deficiency. For a reason that has never been explained, clefts on the left side greatly outnumber those on the right side and bilateral clefts.

Relative Distributions of the Various Types of Cleft.—Based on a study of the records of 703 Danish patients operated upon for one or other of these anomalies, P. Fogh-Andersen found that the incidence was as follows:

Cleft-lip alone	25 per cent. (60% males)
Cleft-palate alone	25 per cent. (59% females)
Cleft-lip and cleft-palate combined . .	50 per cent. (70% males)

In 75 per cent. of the patients the cleft was unilateral.

Effects on Function.—Swallowing, speech, dentition, nose development, and even hearing are affected.

Ability to Suck.—Cleft lip does *not* interfere with feeding. A special teat may be required for bottle feeding but, with breast feeding, a mother invariably acquires a technique to feed the baby adequately.

With cleft palate, however, the infant may be unable to suck properly as negative pressure cannot be established. In this case feeding may be assisted with special bottles and teats, or alternatively spoon feeding may be employed. In some cases an obturator is needed which facilitates sucking.

Speech.—A person with a cleft-palate is unable to make consonants and has a typical cleft-palate voice. To make consonants (e.g. B, D, K, P, T, and the hard G) air must not escape into the nose via the naso-pharyngeal sphincter (the levator sling pulls the soft palate backwards and into contact with the posterior wall of the pharynx). Eighty per cent. of patients may develop favourable speech, but usually require extensive speech therapy to achieve this.

Orthodontics.—The child will require very careful supervision of the growth of the alveolus and teeth, and corrective splinting where indicated.

The nose may remain misshapen (fig. 524), particularly if the upper lip does not develop to its full width and dimensions, and lack of development of the alveolar arch results in malpositioning of the teeth. Much depends upon the reconstitution of, and therefore the moulding action from a properly functioning orbicularis oris muscle.

Hearing.—About 50 per cent. of children with cleft-palate have an audiometrically detectable hearing loss of about ten decibels, due largely to œdema of the orifice of the Eustachian tube consequent upon pharyngeal inflammation from regurgitated food.

Treatment

Aims of Treatment.—*Cleft-lip*—the aim is solely to improve appearances. (Cleft-lip does not interfere with feeding—see above.)

Cleft-palate—the aim is to achieve adequate speech and dentition. The length of the palate is not important, but mobility and muscularity are.

Poul Fogh-Andersen. Plastic Surgeon, Deaconess Hospital, Copenhagen, Denmark.
Bartolommeo Eustachi (Eustachius), ?1520–1574. Professor of Anatomy, Rome.

Timing of Treatment.—*Cleft-lip*—repair is usually undertaken at an age of three months or when the infant weighs 5–6 kg.

Cleft-palate—repair is undertaken at the age of 1–1½ years.

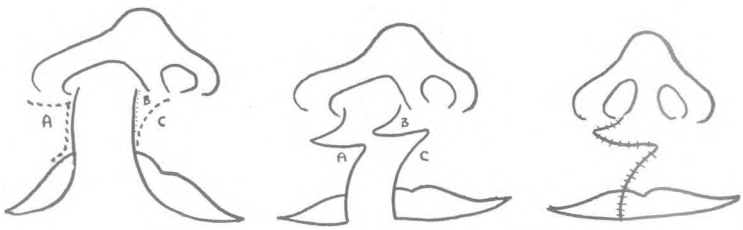


FIG. 522.—Repair of cleft-lip (after Millard).

Methods of Repair.—*Unilateral Cleft-lip:* There are several different operations described for repair of the unilateral cleft-lip. Fig. 522 illustrates the method of repair described by Millard. Fig. 523 shows a cleft-lip before operation and fig. 524 the child at the age of four. Note the residual nasal defect.

Cleft-palate: The aim of repair of cleft-palate is to achieve adequate speech and dentition.

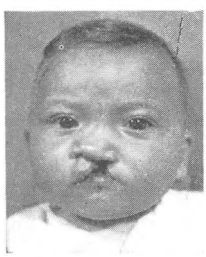


FIG. 523.—Cleft lip.

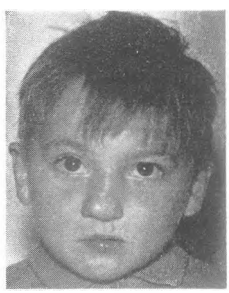


FIG. 524.—Same case as fig. 523 four years after repair.

Operation.—Endotracheal anæsthesia is employed and the head is well extended. The principle of the repair is to achieve firm union of the muscle of the palate in the mid-line. In order to achieve this, incisions are made laterally and the soft tissues extensively dissected to mobilise the palate. (This may involve fracturing of the hamulus). This repair is known as the V-Y repair (fig. 525). The repair is effected by suturing the cleft in the mid-line in two layers (nasal and pharyngeal), leaving a raw area laterally which re-epithelialises.

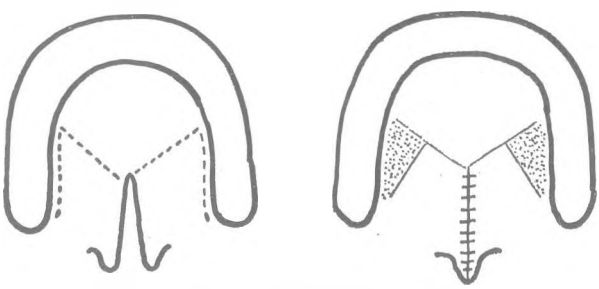


FIG. 525.—V-Y Repair of cleft-palate.

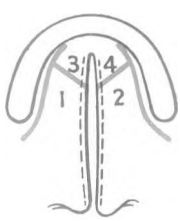


FIG. 526.—Four-flap method.

With clefts extending as far forward as the alveolar arch, the ' four-flap ' repair is used (fig. 526).

When severe alveolar defects exist, bone-grafting is used after corrective orthodontic alignment.

David Ralph Millard, Jr. Contemporary. Plastic Surgeon, Miami, Florida, U.S.A.

Double Cleft-lip with an Unfused Premaxilla (fig. 527).—The premaxilla must not on any account be removed, for the subsequent concavity is most difficult to remedy with a prosthesis. The best method of treatment of these difficult cases is still open to question, but if the premaxilla juts out considerably the second of the following two procedures should be chosen.

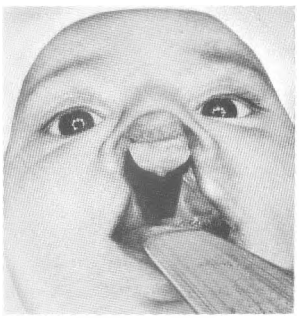


FIG. 527.—Bilateral cleft-lip associated with an unfused premaxilla and cleft-palate.

(*a*) The cleft on each side is united to the prolabium in one or two stages, and as a result of the continuous pull exerted by the reconstructed orbicularis oris, the premaxilla retreats between the halves of the maxilla.

(*b*) First the lip on one side is repaired, in order to ensure an adequate blood-supply to the adjacent bone. With this established, a few weeks later prevomerine bone can be resected safely and (at the same operation) the repair of the other side of the lip is performed (R. J. V. Battle). Again the reconstructed orbicularis oris proves an admirable agent for retaining the replaced premaxilla.

Other Congenital Abnormalities of the Face and Lips

Preauricular Sinus.—Imperfect fusion of the six tubercles that form the pinna results in a preauricular sinus, the opening of which is usually found at the root of the helix or on the tragus. The track run downwards, and ends blindly. A preauricular sinus gives rise to no symptoms unless the tiny opening becomes occluded, when a cyst is prone to develop. If the cyst becomes infected and it bursts or is incised, a cutaneous preauricular ulcer may follow. This ulcer refuses to heal, for infection is maintained from the sinus (Stammers). Often the condition is mistaken for a tuberculous sinus associated with a preauricular or parotid lymph node. The only curative treatment is complete excision of the sinus.

Periauricular dermoid cyst is due to inclusion of epithelium during fusion of two contiguous embryological aural tubercles. Usually the cyst is posterior to the pinna (fig. 528).

FIG. 528.—Peri-auricular dermoid cyst.

Congenital Short Frænum[1] **of the Upper Lip.**—Both lips have a frænum, that of the upper lip being better developed. Sometimes it is congenitally short and is then associated with a wide gap between the permanent incisor teeth (fig. 529). If dental malposition, and a sibilant lisp in the speech are to be prevented, early in life the frænum must be incised transversely, the mucosa on each side of the incision mobilised by undercutting, after which the cut edges are sutured longitudinally.

Mucus sinuses of the lower lip (fig. 530) occur in certain families and as part of van der Woude's syndrome (with cleft lip or cleft palate and small premolar teeth). The sinuses are wide-open mucus-secreting labial glands, which are also found in certain quadrupeds. The treatment is excision.

Prolapse of the Mucous Membrane of the Lip (Bichelia).—The mucous membrane of the lip protrudes as an unsightly fold below the vermilion border. Usually congenital, it can be acquired by repeatedly sucking the lining of the lip through a gap between the natural teeth or an ill-fitting denture, in which case the prolapse is uni-

[1] Frænum—Latin = a bridle.

Richard John Vulliamy Battle, Contemporary. Formerly Plastic Surgeon, St. Thomas's Hospital, London.
Francis Alan Roland Stammers, Contemporary. Emeritus Professor of Surgery, Birmingham, England.
Anne van der Woude, Contemporary. Institute of Human Biology, University of Michigan, Ann Arbor, U.S.A. Described this syndrome in 1954.

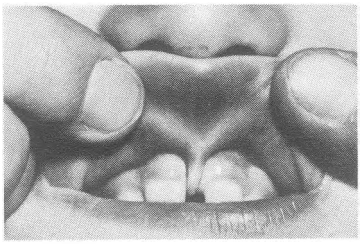


FIG. 529.—Congenitally short frænum labii. (*The late Patrick Clarkson, F.R.C.S., London.*)

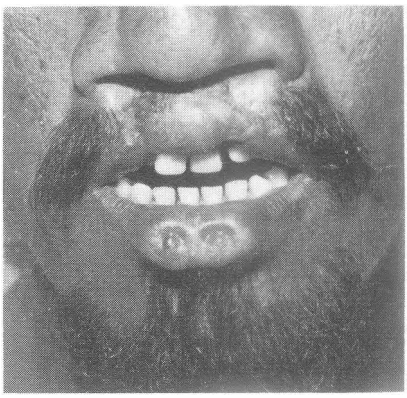


FIG. 530.—Mucus sinuses of the lower lip (van der Woude's syndrome). (*John C. Nicholl, F.R.C.S., London.*)

lateral and mostly affects the lower lip. *Treatment* consists of excision of redundant mucosa. In acquired cases orthodontic corrections must also be made.

Micrognathia (fig. 531).—This is often associated with cleft palate. The infant is born with a mandible the horizontal ramus of which is foreshortened. This renders the tongue unduly mobile, and results in tongue-swallowing with frequent attacks of dyspnœa, cyanosis, and feeding difficulties, especially if a cleft-palate is present as well. *Treatment*—is conservative with attention to nursing (face down), the maintenance of an airway, and feeding. In young adulthood, cosmetic bone-grafting may vastly improve the patient's appearance.

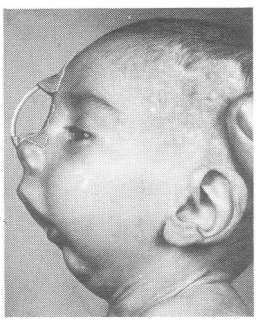


FIG. 531.—Micrognathia. An endotracheal tube has been passed. (*The late Sir Alan Moncrieff, London.*)

FIG. 532.—Mandibular prognathism. (*J. H. Hovell, F.D.S., R.C.S., London.*)

Mandibular Prognathism (fig. 532).—This occurs as a developmental abnormality and may be genetically determined. The unopposed incisor teeth suffer early periodontal disease as a result of disuse. Faulty speech occurs on account of incorrect relationship of the tongue to the incisor teeth, and temporo-mandibular joint dysfunction is favoured. *Treatment.*—After freeing the periosteum from the bone, an open sliding osteotomy of the ascending ramus or the body, with preservation of nerves, and wire fixation of the fragments, gives satisfactory correction of the deformity. If the periosteum is not freed from the bone, muscle pull on the original attachment causes recurrence of the deformity.

LESIONS OF THE PALATE

Swellings.—(*a*) A tumour of the palatal mucosa or bone; (*b*) ectopic salivary tumour (fig. 614); (*c*) carcinoma of the maxillary antrum extending downwards (p. 549); (*d*) an alveolar abscess of the incisors (p. 541); (*e*) chronic infections, e.g. gumma.

Perforation.—A hole in the *mid-line* of the hard palate may be due to friction from a malfitting dental plate. Rarely nowadays is it the result of a gumma. A hole *to one side of the mid-line* is due either to the bursting of an

empyema through the floor of the antrum, or a palatal antrostomy. *Treatment.*—A well-fitting dental plate is all that is required.

LESIONS OF THE LIPS

Pigmented Lips and Buccal Mucous Membrane.—Brown pigmented spots on the lips, inside the cheeks, and on the palate occur in familial intestinal polyposis (Peutz-Jeghers syndrome (fig. 1111)). Addison's disease must be excluded.

Macrocheilia.—True macrocheilia is due to a lymphangioma, akin to lymphangiomatous macroglossia (p. 545). Treatment is similar. Chronic inflammation also causes enlargement.

Cracked Lip.—Chapping is common, and a definite crack in the middle of the lower lip, which bleeds readily, is a frequent complaint in cold weather. *Treatment* includes the local application of glycerin. In chronic cases simple excision may be indicated.

Cracks at the corners of the mouth (p. 564).

Chancre of the Lip.—Unlike a similar lesion on the genitals, the neighbouring lymph nodes become *greatly* enlarged.

Herpes Simplex Infections (see p. 562).

NEOPLASMS OF THE LIP

Lymphangioma (macrocheilia), and cavernous hæmangioma are innocent neoplasms (p. 118).

Ectopic Salivary Tumour.—Usually it is the upper lip of a young adult male which is affected, the tumour commencing on one or other side at the site of the right or left fusion lines (Kuber Dhar Sharma).

Carcinoma of the lip is usually seen in men between sixty and seventy years of age who have followed an outdoor occupation, and it has been known colloquially as 'countryman's lip'. Only comparatively recently has the importance of exposure to sunlight, wind and driving rain been stressed in the ætiology of this neoplasm. In particular, bright sunlight causes exfoliation of the lips—more especially the lower lip. Actinic cheilitis should be considered as a precarcinomatous condition, and 40 per cent. of patients with carcinoma of the lip give a history of recurrent blistering cheilitis.

This is a squamous-celled carcinoma (known for short as an 'S.C.C.' or an 'epithelioma') which presents as a shallow ulcer (fig. 67) with or without the typical everted edge. Thus, any ulcer of the lip which does not readily heal should be viewed with suspicion and a biopsy obtained. With small lesions, diagnosis and treatment can be combined as an excision-biopsy. The lower lip is affected in 93 per cent. of cases, the upper lip in 5 per cent., and 2 per cent. occur at one of the angles of the mouth. Rutherford Morison was the first to point out that a carcinoma occurring at the angle of the mouth and involving *both* lips, however slightly, is far more malignant than the more usual varieties. The reason for this is that the angle of the mouth has a double lymphatic drainage.

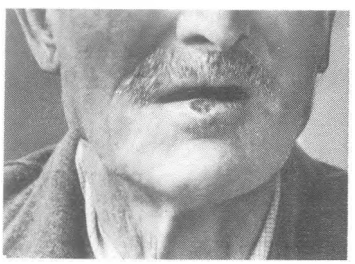


FIG. 533.—Carcinoma of the lower lip.

Carcinoma of the lip must be distinguished from Molluscum sebaceum (Kerato-acanthoma), described on p. 116.

Treatment.—In its typical form (fig. 533) the carcinoma is comparatively slow-growing and it is eminently curable by

John Law Augustine Peutz, 1886–1957. Chief Specialist for Internal Medicine, St. John's Hospital, The Hague, Holland.
Harold Jos Jeghers. Professor of Internal Medicine, New Jersey College of Medicine and Dentistry, Jersey City, U.S.A.
Thomas Addison, 1793–1860. Physician, Guy's Hospital, London.
Kuber Dhar Sharma, Contemporary. Professor of Pathology and Bacteriology, Medical College, Aurangabad, India.
James Rutherford Morison, 1853–1939. Professor of Surgery, University of Durham, Newcastle-upon-Tyne.

surgery or radiotherapy, the five-year survival being 70 per cent. If the lesion is advanced, or has recurred after radiotherapy, adequate surgical treatment necessitates plastic reconstruction.

Radiotherapy.—After confirmatory biopsy, conventional deep therapy voltage (250 kV) is used. Ten doses are given over a period of two weeks to a total dose of 5,000 rad, the mouth, alveolus, and tongue being protected by lead backing. Radium needles or a mould may be used for buccal or commissural lesions (7,000 rad in seven days).

Treatment of Lymph Nodes.—Involvement of the lymph nodes (submental or deep cervical) is relatively late. If the nodes are mobile, the present practice is to perform a classical block dissection (p. 610). If the nodes are fixed, or the patient is elderly and feeble, operation is contra-indicated. Telecobalt therapy may be of use in these circumstances.

Careful follow-up of cases is essential.

FACIAL INJURIES

Injuries to the face are extremely common. Minor injuries such as black-eyes and small lacerations form a large number of casualty attendances especially at weekends. These are inevitably due to accidents, sport injuries or fights. In more severe trauma such as that resulting from motor accidents, the face and underlying bones are involved in at least 30% of the cases. The victims are usually motor car passengers who are thrown forward against the windscreen or dashboard. The nature and severity of the injuries vary enormously with the force and striking surface involved. The wearing of safety harness, now compulsory equipment in new cars, reduces the incidence of this mutilation.

First Aid.—The immediate danger to patients with severe injuries of the face is respiratory obstruction due either to inhaled blood, or of the tongue falling back as a result of a bilateral fracture of the jaw. The patient should therefore be immediately placed in a horizontal and lateral (tonsil) position (p. 690) or right over on to his face with his forehead supported on his hands. If he has to be transported by ambulance he should remain in this position and not be turned on to his back. Local firm pressure with pads will help to control bleeding (p. 74).

Examination of the injury.—These wounds often appear to be more horrifying than they really are (fig. 3) especially as bleeding is so profuse. No examination is complete until carried out in the operating theatre with the patient appropriately anæsthetised.

N.B. If the patient is suffering from shock, one should always look for other injuries. The shock is seldom due to facial injuries alone.

Soft Tissue Injuries.—Because of the good blood supply, wounds of the face heal well. Excision of dead tissue can be reduced to a minimum, and nearly all wounds can be treated by primary suture even when treatment has been delayed for as long as 24 hours. The procedure for operation on facial wounds is similar to that for wounds in general (p. 4).

If the laceration follows a car accident in which the victim has been thrown against the windscreen, the wound is very likely to contain pieces of glass. These and any

dirt must be carefully searched for and removed. Dirt may be removed by persistent gentle scrubbing, any left behind may cause permanent tattooing. All layers must be carefully reconstituted, mucosa and muscle with 4/0 chromic catgut sutures and skin with 4/0 or 6/0 silk or nylon sutures. Numerous small sutures are stronger than a few widely spaced thick sutures. On the nose, sutures remaining for more than 5 days may leave a permanent mark. After 24 hours skin wounds need no dressing at all and should be left open (fig. 4).

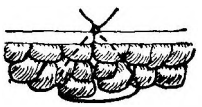


FIG. 534.—Method of avoiding inversion of the skin edges. (*After T. P. Kilner, F.R.C.S., Oxford.*)

Facial Nerve.—Primary repair may be indicated (depending on other injuries and the state of the overlying skin etc.), but this can be a major task. Secondary repair is not usually very satisfactory. Re-innervation after trauma occurs in a sufficient percentage of cases to warrant waiting 12 months.

Parotid Duct.—Suturing is often easier than expected and should be done where ever possible. It is advisable to anastomose the duct over a fine ' polythene ' catheter or a broad nylon thread which remains *in situ* for 2-3 weeks. Parotid fistulae often close spontaneously but may take many months to do so (p. 590).

Lachrymal Apparatus.—Suture of the lid is performed in layers after cannulating both ends of the injured canniliculus with a fine nylon thread, the distal end of which is passed into the lachrymal sac. This thread is kept in as long as possible (up to 3 months). Secondary procedures for scarred duct systems are best referred to plastic or ophthalmic surgeons.

Bone Injuries of the Face and Jaws

Fractured Nose.—This is the simplest type of ' facio-maxillary ' fracture, most often missed and often the most difficult to treat. The 2 nasal bones, plus their lateral cartilages, form a tent over the vertical midline septum. The injuries may be (i) *lateral* (fig. 540) resulting in impaction of one lateral bone under the other. Reduction cannot be effected until the side away from the impact is elevated first, thus freeing the bone on the side of the blow, (ii) *frontal*—this results in telescoping of the nasal structures, and is often associated with maxillary fractures.

Treatment.—Reduction is best undertaken using general anaesthesia and a pharyngeal pack. However, it may be done in a co-operative patient using local anaesthesia with adrenaline. (1) *Lateral injury*—Using a Walsham's forceps (fig. 535) covered with rubber tubing, the deformity is increased, thus freeing the impacted bone. Both sides are then aligned using the same forceps. The corrected position can be maintained by a plaster of Paris cast (fig. 536) but this is not essential. (ii) *Frontal injury*—The septum is gripped with Asch's forceps (fig. 537) and the whole nose is elevated. It may be necessary to hold the position with trans-nasal wires over lead plates. The septum must always be carefully aligned with Asch's forceps.

Fractures of the maxilla are classified according to Le Fort[1] (fig. 538). The likelihood of a fracture being present is suggested by the type of accident, the facial injury (dish-face deformity, split-open face, or swelling), the inability to close the jaws (open-bite deformity), or diplopia due to upward displacement of the floor of the orbit. Injuries to the eye and mandible are present in over 40% of cases. High fractures across the root of the nose may involve the cribriform plate and result in C.S.F. rhinorrhoea. An X-ray confirms the type of fracture. *Treatment* is best carried out in a facio-

[1] René Le Fort, Surgeon, Paris, defined these fractures as early as 1901, by macabre research, in which he dropped rocks and other heavy objects on the faces of cadavers.

William Johnson Walsham, 1847–1903. Surgeon, St. Bartholomew's Hospital, London.
Morris Joseph Asch, 1833–1902. Surgeon, New York Eye and Ear Hospital, New York, U.S.A.

maxillary centre. A tracheostomy may be necessary. In addition to the repair of the face (p. 533), the displaced maxilla is freed, by rocking with bone forceps, and restored to its normal position. According to the type of fracture, stability is maintained by either (1) wiring the upper and lower jaws together in proper occlusion, (2) by wiring a pliable metal bar to the teeth or the edentulous alveolus, and fixing this by a system of extra-oral rods (scaffolding) (fig. 539), (3) by internal wire sutures (maxilla to malar bone).

FIG. 535.—Walsham's forceps.

FIG. 537.—Asch's forceps.

FIG. 536.—Reduced nasal fracture. Position may be maintained by P.O.P. cast (see text). (*E. Melmed, F.R.C.S., Dallas, U.S.A.*)

Le Fort I. Fracture involving the maxilla only. The fracture line passes through the base of the antrum on either side and across the nasal floor through the septum.

Le Fort II. Posteriorly the fracture line passes up behind the nose in the direction of the malar-maxillary junction, across the posterior wall of the antrum, and across the pterygoid process in the middle or upper third. The anterior fracture line passes into the orbit in the area of the lachrymal bone, and soon emerges again to break across the fronto-nasal process of the maxilla and the bridge of the nose to meet a similar fracture line on the opposite side. The posterior wall of the orbit is not fractured in this type.

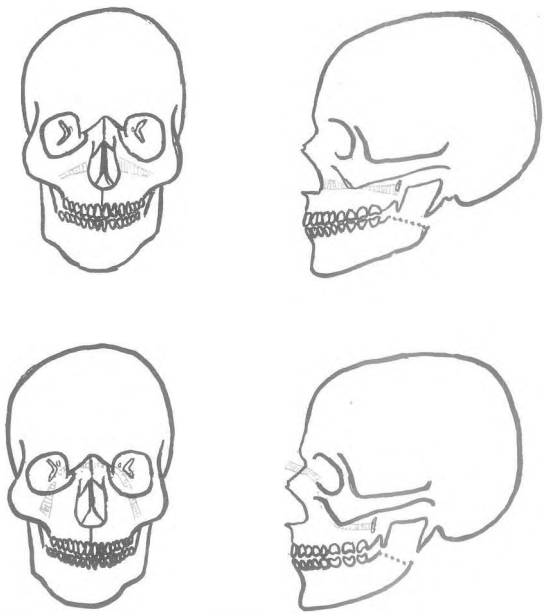


FIG. 538.—Naso-maxillary fractures, Le Fort classification.

Le Fort III. There is a cranio-facial disjunction, the fracture line passing through both zygomatic arches, both external angular processes of the malar bones, across the

back of both orbits, and the back of the nose in the ethmoid area. There is a separation of the nasal bones from the frontal bone. Posteriorly the septum is fractured high up and likewise the pterygoid processes. (i.e. a complete separation of the maxilla, nose and ethmoids and the palatal bones from the base of the skull.)

The **Malar bone and Zygomatic Arch** may be fractured and depressed by a direct blow. The important physical signs indicating the necessity for reduction are diplopia and obstruction to opening of the mouth. Unsightly deformity results (fig. 540), and anæsthesia of the cheek may occur due to pressure on the infra-orbital nerve. Pain on mastication is commonly noticed. Fracture of the orbital floor may result in trapping or prolapse of the inferior oblique muscle and gives rise to diplopia when looking upwards and outwards to the affected side. A palpable notch may be felt on gentle palpation of the infra-orbital margin.

Treatment.—(i) *Elevation:* The simplest approach is to make an incision in the temple hair margin through the temporalis fascia. A Bristow's elevator is then passed behind the zygomatic arch and the malar is replaced. Sometimes an audible click is heard as it locks back into position. (ii) *Fixation:* In simple fractures (e.g. rugby injuries) often no fixation is necessary if the reduction is stable. If unstable, it is best to wire the fronto-zygomatic suture and the malar-maxillary infra-orbital margins using 0.35 mm. stainless steel wire. If the malar is collapsed, it may be necessary to pack the maxillary antrum to maintain contour. This is approached via a Caldwell-Luc antrostomy (p. 552) and the cavity packed with ribbon gauze soaked in antiseptic varnish (Whitehead's). The pack is kept in position a minimum of one month.

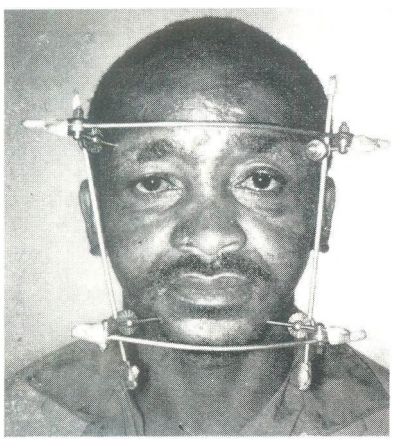


FIG. 539.—Extra-oral rods maintaining position of jaw in facio-maxillary injury (Le Fort II). (*E. Melmed, F.R.C.S., Dallas, U.S.A.*)

FIG. 540.—Depressed fracture of left zygomatic arch. Motor cycle accident. (*T. McNair, F.R.C.S., Edinburgh.*)

The **Mandible** is usually fractured in one of three situations (fig. 541):

(1) The *neck of the condyle* is occasionally fractured, in which case it is displaced forwards and inwards by the pull of the attached external pterygoid muscle. Localised pain occurs on movements of the jaw, and crepitus is detected by the patient or surgeon.

(2) The *ascending ramus* may be fractured, usually in the region of the angle of the jaw. The opposite condyle may be fractured at the same time. Little displacement occurs, as the masseter on the outside and internal pterygoid muscle on the inner aspect sandwich the fragments between them. The injury is suspected on account of persistent localised pain, and is confirmed by an X-ray. Mastication is limited to soft food for three weeks.

Walter Rowley Bristow, 1882–1947. Orthopædic Surgeon, St. Thomas's Hospital, London.
Walter Whitehead, 1840–1913. Surgeon, Manchester Royal Infirmary, Manchester, England.

(3) The *body of the jaw* is the part most commonly fractured, sometimes as the result of a blow with a fist. The fracture frequently occurs at the site of the socket of the canine tooth, the cavity of which weakens the bone, and also because this region is the junction of two curves. Occasionally the fracture is bilateral, in which case the central portion of the jaw is displaced downwards by the anterior bellies of the digastric muscles and the muscles attached to the genial tubercles. Owing to the firm attachment of the mucoperiosteum to the bone, the fracture is nearly always compound.

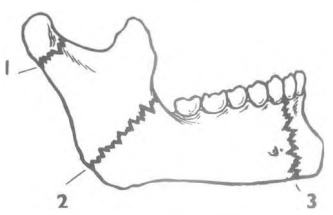


FIG. 541.—Lower Jaw.
1. Neck of condyle.
2. Through the angle or ascending ramus.
3. Anterior to the mental foramen, through the canine fossa.

Diagnosis is usually obvious, as speech and swallowing are impaired. Blood-stained saliva trickles from the mouth, and irregularity of the line of the teeth is apparent. Crepitus can be elicited with ease.

Principles of Treatment

As soon as possible the patient is referred to the dental surgeon for fixation of the fracture. The simplest method is to wire the jaws together in occlusion by interdental wiring (fig. 542). An alternative is an interdental splint which fits over the teeth adjacent to the fracture. A corresponding splint is fitted to the upper jaw, and the two splints are fixed together, in occlusion, by wire or elastic bands. Any obviously infected teeth, or an interposed tooth, are removed during the fitting. In edentulous patients, either dentures or acrylic moulds are wired to the alveolus and thence together.

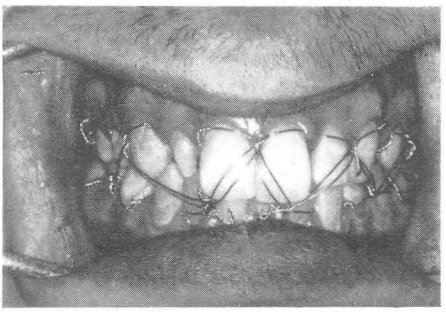


FIG. 542.—Interdental wiring for fractured mandible. (*E. Melmed, F.R.C.S., Dallas, U.S.A.*)

Post-operative Care.—As the fracture is compound, infection from the mouth is likely, particularly if pyorrhœa or infected teeth are present. Warm antiseptic mouth-washes are therefore used almost continuously. The diet is confined to fluids, which are taken temporarily through a rubber tube. Antibiotic cover is necessary.

Complications are mainly due to infection. Thus necrosis of bone and delayed union are not uncommon, while submandibular cellulitis may require operative intervention. Aspiration pneumonia is a grave danger, particularly in old or alcoholic patients with pyorrhœa.

SPECIAL INFECTIONS OF THE FACE

Boils and 'Pimples' in the region of the 'danger' (mask) area of the face (fig. 543) should never be squeezed, pricked, or incised, for by so doing the infection

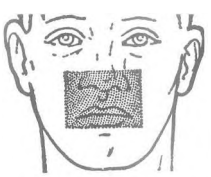


FIG. 543.—The 'danger' area of the face.

can reach the cavernous sinus by venous connections and cause cavernous sinus thrombosis, a fatal condition in the pre-antibiotic days. The local treatment of boils and 'pimples' include dressings with magnesium sulphate and glycerin paste, or short-wave diathermy. A broad-spectrum antibiotic is given, with review following the culture of any pus. Massive doses of antibiotics (e.g. 25 mega units of penicillin daily) are commenced immediately there are any local signs or general symptoms (rigors) suggestive of cavernous sinus thrombosis, and they are continued (in reduced dosage) for a prolonged period.

FIG. 544.—Chronic infected facial 'cyst'. Inset, the causative lesion, showing a chronic abscess attached to a root. (*D.C. Bodenham, F.R.C.S.(Edin.), Bristol.*)

Infected Facial 'Cyst' of Dental Origin.—Silent dental infection can be responsible for producing a chronic subcutaneous abscess of the face (fig. 544). Almost always this is diagnosed as an infected epidermoid cyst and, alas, frequently the 'cyst' is excised, and a chronic discharging sinus results.

The patient must be subjected to dental radiography. When a root abscess is the cause of the facial lesion, extraction of the offending tooth or the responsible remnant thereof will rapidly render other treatment, other than perhaps a tiny incision, unnecessary.

Anthrax (p. 35).
Lupus (p. 114).
Leprosy (p. 40).

NEOPLASMS OF THE FACE

Benign and malignant neoplasms of the skin of the face are considered on pp. 114 to 125.

CHAPTER 31

THE TEETH AND GUMS. JAWS. NOSE. EAR

Congenital Anomalies.—The third molars and upper second incisors may be absent. Supernumerary teeth sometimes occur in clusters. The presence of a fourth molar may cause trigeminal neuralgia (Shah). Rarely seen now are the notched, peg-shaped incisors of Hutchinson, and turret molars of Moon, in congenital syphilis (p. 47). Small premolars exist in the van der Woude syndrome (fig. 530).

Impacted Wisdom Tooth.—The tooth is the third lower molar. It is prevented from normal eruption by an adjacent tooth. The unerupted portion of the crown is overhung by a pocket or flap of the gum, known as an operculum, viz. ——→

where food debris collects, resulting in infection, which tends to spread to the cheek and sometimes to the neck. Trismus[1] is often present.

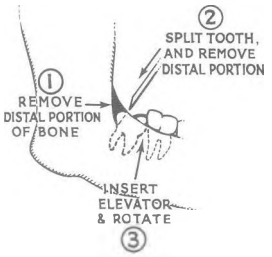


FIG. 545.—Steps in extracting a vertically impacted mandibular molar tooth. (*After Professor H. H. Stones.*)

Treatment.—If trismus is severe the patient should be given a general anæsthetic to release the spasm of the muscles of mastication and permit the insertion of a mouth-prop. If an abscess is present beneath the operculum, it is drained. When the acute symptoms have subsided, the impacted tooth should be extracted (fig. 545), unless one of the molar teeth anterior to it is carious. Extraction of the carious tooth may then afford room for the impacted molar to erupt.

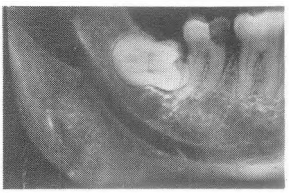


FIG. 546.— Radiograph showing horizontal impaction of third molar. (*Dr. Sydney Blackman, London.*)

In horizontal impaction (fig. 546), the second molar may be extracted first and the third impacted molar removed by (*a*) chiselling away the overlying alveolus; (*b*) dividing the tooth; (*c*) removing the fragments with an elevator, viz. ——→

Hæmorrhage after Extraction.—Often within the socket there is an open artery that in more accessible situations would have been ligated in the first instance. Quite often the bony alveolus has been fractured, and a loose fragment or a spicule of bone is present, either of which prevents firm clotting within the socket. Especially in males, it is necessary to exclude hæmophilia (p. 80).

Treatment.—Provided the blood pressure is satisfactory the patient should sit bolt upright. A mouth gag is placed in position, the edge of the socket is everted with a pair of dissecting forceps, and with another pair the cavity is mopped with suitable pledgets of cotton-wool. Loose bone or spicules, if present, are removed. Firm pressure with a pledget for a few minutes may stop the hæmorrhage. When the hæmorrhage comes from the depths of the socket many dental surgeons use liquor ferri perchlor. as a styptic, and having packed the cavity, a small roll of gauze, upon which the patient bites, is placed over the socket. Snake venom (Stypven) and absorbable gauze or gelatin sponge may also be used. Should the hæmorrhage come from the gum margin and is persistent, the flow can be arrested by a stitch passed through the gum on either side of the tooth socket.

[1] Trismus—Greek τρισμός = clenching.

C. S. Shah, Contemporary. Dental Surgeon, Rajkot, India.

Oro-Antral Fistula.—A hole between mouth and antrum via a tooth socket is attributed to the special use of narrow elevators for removing the apex of a tooth (Fickling). Air will escape when the patient blows his nose, or a sinogram will confirm the diagnosis. *Treatment.*—(1) Immediate suture of the socket, and cover (not pack) by ribbon gauze. (2) If seen later with sinusitis give saline irrigations and parenteral antibiotics. (3) Failure to heal in six to eight weeks requires the operation of inkwell inversion of freed circularly incised mucosa and cover by a long muco-periostal flap.

DENTAL CARIES

Ætiology.—*Diet.*—The teeth of those Esquimaux who live exclusively on fish, flesh, and fowl, and of African pygmies who consume uncooked foods only, are almost free from dental caries. The incidence rises steeply among civilised people whose diet includes carbohydrates in highly refined forms, e.g. white bread, and sweets eaten between meals. Dental caries is rife before the age of twenty-five, particularly in early childhood because the deciduous teeth are vulnerable to decay.

Acid-producing and Proteolytic Oral Bacteria.—Carbohydrate material clinging to the teeth is subjected to the activity of bacterial enzymes (sucrases), forming dental plaques of dextran, which is later broken down to lactic acid, which is capable of dissolving dental enamel. Once the enamel shell has been penetrated, proteolytic enzymes, generated by other bacteria, disintegrate dentine, which forms the bulk of the tooth (fig. 547).

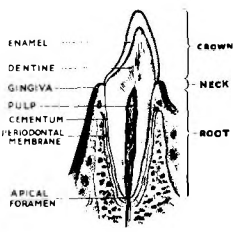


FIG. 547.—Sagittal section through an incisor tooth *in situ*.

Minor enamel hypoplasia resulting from lack of vitamins and possibly from fluoride deficiency in domestic water supplies, is a factor in producing an increased incidence of caries in children (Mellanby).

The Stages of Dental Decay

Stage 1.—*Erosion of the enamel* is symptomless, and will almost certainly be overlooked unless each tooth is examined systematically with a dental probe.

Stage 2.—*Dissolution of dentine* is also painless until it has progressed to near the pulp, when thermal changes (heat, and especially cold) and mastication of sweets may bring on the familiar pain (toothache).

Stage 3.—*Part of the crown collapses,* due to undermining. The cavity is now more easily seen. Pain may be referred to other teeth.

Stage 4.—*Acute Pulpitis.*—Pain is throbbing and intermittent, and thermal changes cause pain. Pressure of a piece of cotton-wool inserted into the cavity is painful. Pulpitis terminates in disintegration of the pulp. Untreated, acute pulpitis is liable to lead to an alveolar abscess. Chronic pulpitis is the harbinger of a periapical abscess.

Dental Caries and the Surgical Patient.—The culmination of caries, a mouth full of broken teeth, stumps and tartar, increases the risk of morbidity from surgical procedures. Pre- and post-operatively there is a likelihood of stomatitis, and, in conditions leading to dehydration (e.g. intestinal obstruction), of acute parotitis (p. 585). Mastication at mealtimes will massage bacteria from infected roots into the blood stream, so causing recurrent bacteriæmia which may be detrimental to recovery from any operation.

Thus, special attention must be given to oral hygiene in the pre- and post-operative periods. A dental opinion and the help of a dental hygienist is invaluable, and in non-emergencies dental extraction prior to surgery may be considered. This tends to delay operation by four to six weeks in order that the gums should be soundly healed, so dispersing all chances of bacteriæmia.

During anæsthesia, special care must be taken to prevent loosening or

Benjamin William Fickling, Contemporary. Consulting Dental Surgeon, St. George's Hospital, London.
Lady Mary Mellanby, Contemporary. Formerly Investigator for the Medical Research Council, London.

breaking of a tooth, which may be inhaled to cause collapse of a segment or lobe of a lung and then a lung abscess. Thus, prior to anæsthesia a note should be made of the state and numbers of teeth and, if possible, the patient should be warned that a loose tooth is in jeopardy and may require removal for safety. In the case of a missing tooth, believed inhaled, an X-ray of the chest, and possibly bronchoscopy, will be required.

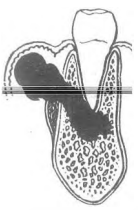


FIG. 548.—Alveolar abscess eroding the lateral plate of the alveolus. This is the most common course, because the roots point in this direction, and the lateral plate is about half the thickness of the medial plate.

ALVEOLAR ABSCESS

While an acute alveolar abscess can occur at any age, it does so most often during childhood and early adult life. Abscesses in the first dentition can occur in either jaw, while those connected with the second dentition concern particularly the teeth of the lower jaw, more especially a molar tooth.

Pathology.—As a result of acute pulpitis, the putrifying pulp generates gases that exert pressure through the root canal into the osseous tissue around the apex of the tooth. Localised osteitis and abscess formation (gumboil) ensue. Almost always the abscess points on the labial aspect (fig. 548). One abscess to point lingually is that in relation to the upper lateral incisor, which gives rise to a swelling on the palate (p. 531), and an abscess from an impacted wisdom tooth can burst through the medial wall of the alveolus and cause the dangerous Ludwig's angina (submandibular cellulitis, p. 604).

If early resolution is not brought about by antibiotic therapy, or if the abscess is not drained promptly and effectively, osteomyelitis of the jaw is to be expected (p. 544).

FIG. 549.—Extensive alveolar abscess.

Clinical Features.—The pain is dull and constant. Swelling of the cheek (fig. 549) and redness and œdema of the gum in the neighbourhood of the culpable tooth are characteristic signs. The general reaction to the infective process may be considerable, and a tender enlargement of the regional lymph nodes is usual.

Diagnosis and Radiography.—Every swelling of the jaw demands a careful dental and radiological examination. For alveolar abscess a dental cause must be found—caries, an impacted 'wisdom', retained roots, or a dead tooth (p. 542). Only when sufficient time (ten days or more) has elapsed for resorption of bone to occur will rarefaction around the apex of the affected tooth become apparent on X-ray.

Treatment. Drainage and Antibiotics.—Surgical principles must be followed. (1) An abscess must be drained. (2) Antibiotics are not used in place of a drainage operation: they are of value early, before pus has formed, i.e. in the stage of indurated cellulitis, and they are used in support of the drainage operation. Here penicillin is usually employed, to be changed if necessary when the antibiotic sensitivities of the organisms grown from the pus are known.

Physical Agents.—Cold applications externally and hot intra-oral irri-

FIG. 550.—Sinus following bursting of an alveolar abscess externally.

gations are useful adjuncts. Hot external poultices are avoided as they promote external pointing which, cosmetically, is to be avoided (fig. 550).

Drainage of an Alveolar Abscess:

(*a*) *Into the Mouth.*—When a cystic swelling in relation to the gum becomes apparent, the abscess should be opened into the mouth by incising the periosteum. If an alveolar abscess has been opened, ten days should elapse before the dental extraction.

(*b*) *Drainage by Extraction of the Offending Tooth.*—In the case of the primary dentition this is an accepted and generally successful procedure. In the case of the second dentition gentle and proper dentistry is vital. *General anæsthesia* must be used. Rough dentistry and local anæsthesia may engender osteomyelitis, in spite of antibiotic cover.

ROOT ABSCESSES

While an alveolar abscess is usually acute, a root abscess is nearly always chronic. It arises by extension of infection from the pulp through the apical canal. As a rule there are no symptoms, and no visible signs of a root abscess, which is only revealed by a dental radiograph (fig. 551). The absence of pain, or sensory response to hot or cold stimuli means a *dead tooth.* If so, it will look grey-blue and has an altered percussion note. Occasionally a chronic root abscess breaks its confines, and burrows to present externally in the form of a median mental abscess sinus (p. 546), or a facial 'cyst' (p. 538), which respond to drainage by extraction of the affected tooth.

FIG. 551.—Dental radiograph showing root abscess of the left lower lateral incisor tooth.

THE GUMS

GINGIVITIS AND PYORRHŒA ALVEOLARIS

The periodontal membrane is the periosteum lining the tooth socket. Its fibres are arranged to form a sling for the root (or roots) of a tooth in its bed of bone.

Clinico-pathological Features.—The fundamental predisposing cause of these conditions is gum recession and an excessive deposit of tartar. The tartar pushes the gingival margin away from the teeth with the result that (*a*) the periodontal membrane is broken, (*b*) the gum recedes, and (*c*) particles of food accumulate between the gum and the teeth. *In gingivitis,* the gums are swollen, spongy, and bleed readily. *In pyorrhœa,* pus can be expressed from the gingival pockets. Fetor oris may be extreme. The teeth become loose and fall out.

Gingivitis may be associated with general stomatitis (p. 560), Vincent's stomatitis (p. 563), or be in evidence in scurvy and in chronic mercurial poisoning. In chronic lead, and bismuth, poisoning there is a characteristic narrow line of blue-black dots on the gums near the dental margin.

Treatment.—Comparatively early cases are often benefited by regular scaling, daily massage of the gums with the fingers, removing debris with a wooden toothpick and the frequent use of an astringent mouth-wash (e.g. ferric chloride). If this treatment is unsuccessful and the majority of the remaining teeth are sound, gingivectomy (see below) is advised in order to eradicate the periodontal pockets. When the condition is fully established, cure is unlikely, if not impossible, without extracting the teeth implicated.

Dangers of Dental Extraction in Pyorrhœa Alveolaris.—Clearing the jaws of teeth is not to be undertaken lightly in cases of pyorrhœa alveolaris. The bacteriæmia that follows is liable to set up broncho-pneumonia, and also may infect any existing endocardial valvular lesion; frequently the onset of subacute bacterial endocarditis dates

from dental extraction. Therefore extraction in cases of pyorrhœa alveolaris should be carried out only under full antibiotic cover.

HYPERTROPHY OF THE GUMS

Hyperplastic Gingivitis.—The gums around the labial aspect of the upper incisors and their inter-dental papillæ are affected most. Bleeding is frequent. On pressure, pus usually oozes from the gingival pockets. In some long-standing cases the hypertrophied gum almost buries the teeth.

Gingivitis Gravidarum.—Consisting of granulation tissue, the hyperplastic 'tumours' may grow as large as currants and they may require cauterisation or diathermy.

In the treatment of epilepsy, the administration of sodium phenytoin leads to hyperplasia of the gums in susceptible individuals.

Treatment is gingivectomy or, if a radiograph shows the bone to be hypertrophied, alveolectomy in addition.

Localised hypertrophy of the gum due to pressure of an ill-fitting denture is common. If removal of the cause does not remedy matters in a fortnight, the hypertrophied area must be excised and examined histologically to see if the lesion is neoplastic.

GINGIVECTOMY

Indications.—(1) Hypertrophy of the gums; (2) to remove periodontal pockets around otherwise sound teeth.

Operation.—Under local anæsthesia, the excess of gum is excised *obliquely* (fig. 552) on both the labial and the lingual sides, together with the interdental papillæ. A cautery arrests bleeding, and a paste (zinc oxide and eugenol[1]) is applied to the raw surface and left for a week to prevent granulation and to allow epithelialisation. If both sides are affected there are three (fortnightly) stages of removal : (*a*) both left sides as far as the canine teeth ; (*b*) both right sides similarly; (*c*) the remainder.

FIG. 552.—Gingivectomy. The obliquity of the resection promotes drainage and prevents recurrence.

ALVEOLECTOMY

Indications.—(*a*) Hypertrophy of gums with hypertrophy of alveolus before fitting dentures. (*b*) Irregularities of alveolus, e.g. after extractions. (*c*) Sometimes in cases of fibrous epulis.

Operation.—Any remaining teeth are extracted. With a stout scalpel, an incision is made right down to the bone. Employing a periosteal elevator, a flap is raised on the labial aspect, and a similar one on the palatal aspect. The irregularities of the bone are removed with a hammer and chisel. The cut edges of the mucosa are sutured over the alveolus.

FIG. 553.—Fibrosarcomatous epulis.

TUMOURS OF THE ALVEOLUS (EPULIS)

'Epulis' is an ancient term with no pathological significance; it merely indicates that there is a solid swelling 'situated on the gum'. There are four varieties of epulides :

1. **Granulomatous Epulis.**—A mass of granulomatous tissue forms around a carious tooth, or at the site of irritation by a denture, requiring extraction of the tooth and scraping away the granulations. A portion must be examined histologically. *Gingivitis Gravidarum* is referred to above.

2. **Fibrous (fibroid) Epulis.**—The commonest form, is a simple whorled fibroma arising from the periodontal membrane, present under the gum. Rarely a soft, bluish-red, bleeding, and rapidly growing fibrosarcomatous type occurs (fig. 553). Both require removal of the adjacent tooth or teeth and resection of a wedge of bone, including the portion of the gum containing the growth.

3. **Giant-celled ('myeloid') Epulis** is an osteoclastoma, arising peripherally in

[1] Eugenol—an active principle extracted from oil of cloves.

the jaw and presenting under the gum. It is soft, and the over-lying gum is sessile and plum-coloured from vascularity. Ulceration and serious hæmorrhage can occur. X-rays show bone destruction with ridging of the walls (pseudo-trabeculation). The treatment is curettage for small tumours, but radical excision (even requiring a graft for a mandible) for large tumours.

4. **Carcinomatous Epulis.**—An epithelioma of the gum may start around a tooth, or in a socket, causing a painful, infected, and infiltrating lesion which later becomes frankly fungating and ulcerating, invading bone and spreading to cervical lymph nodes. A biopsy must be performed to confirm the diagnosis. Treatment depends upon adequate resection (grafting for mandible, excision of maxilla). Radiotherapy may be useful.

THE JAWS

A CLASSIFICATION OF JAW SWELLINGS

This is merely for guidance. The epulides, by definition ' swellings of the gum,' are excluded.

1. INFLAMMATORY.	2. ODONTOMES (epithelial).	3. BONE TUMOURS
		Benign.
(*a*) Alveolar abscess.	(*a*) Dental cyst.	(*a*) The fibro-osseus group.
(*b*) Osteomyelitis.	(*b*) Dentigerous cyst.	(*b*) Osteoclastoma, and the giant-celled reparative granuloma.
(*c*) Actinomycosis. (*Not specifically of the jaw but related to it.*)	(*c*) Adamantinoma.	Malignant.
		(*a*) Osteosarcoma.
		(*b*) Carcinoma of the maxillary antrum (destroying bone) (and see p. 549).

1. INFLAMMATORY SWELLINGS

(*a*) **Alveolar Abscess.**—This has been described (p. 541).

(*b*) **Osteomyelitis of the Jaws.**

This can be acute, subacute, or chronic.

Acute osteomyelitis is rare. In infants it may be a complication of acute specific fevers (measles or scarlet fever), and either the maxilla or mandible may be affected. In other cases infection may spread from the antrum, lachrymal sac, or a dental sac of the first molar tooth. If the maxilla is affected, puffiness of the eyelids occurs (fig. 554). The lateral aspect of the alveolus will be found swollen. A discharging sinus may be seen and pressure over the maxilla will cause pus to run from the nostril and sinus. Radiographs are of no value (Cavanagh). The condition is often mistaken for dacryocystitis.

Treatment.—Though the antrum is small, a polythene tube is introduced by trocar and cannula via the nose, and the cavity is irrigated with normal saline twice daily, the tube being strapped to the cheek. Appropriate antibiotics are given for seven to ten days.

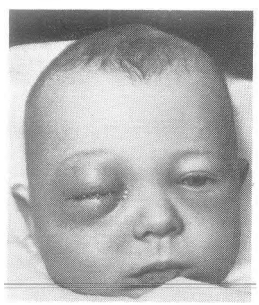


FIG. 554.—Acute osteomyelitis of the maxilla. (*Florence Cavanagh, F.R.C.S. (Edin.), Manchester.*)

Subacute Osteomyelitis.—The commonest type, it affects adults and is caused by the spread of apical dental infection or an alveolar abscess. The

Florence Cavanagh, Contemporary. Honorary Clinical Lecturer in Child Health, University of Manchester.

condition may follow a fractured jaw, which is almost invariably compound into the mouth. The organism is usually the *Staphylococcus aureus*. The mandible is usually affected, probably because it has a single, rather tenuous blood supply running in the long axis of the bone, which is easily obstructed by infection or trauma. Obstruction to the blood supply causes bone necrosis, which is the essence of osteomyelitis of the jaw. The maxilla is better protected by a series of vertical arteries with anastomoses. Injudicious extractions performed under a local anæsthetic, and poor general condition (anæmia and under-nutrition), are local and general factors (Wass).

Thus osteomyelitis must be suspected if there is pain, swelling, tenderness after dental extraction or a treated alveolar abscess. There may be a discharging internal or external sinus; this may relieve tension within the bone and so pain is relieved. Increased tension in the dental canal compresses the inferior dental nerve (as well as the vessels) so numbness of the chin in the distribution of the mental nerve is diagnostic. Radiological evidence of bone necrosis takes three weeks or longer to become evident.

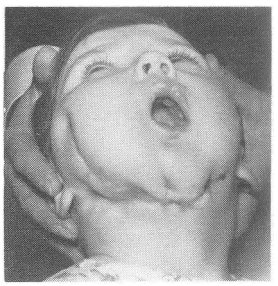


FIG. 555.—Osteomyelitis of the lower jaw shortly after a very large sequestrum had been removed.

Differential Diagnosis.—Osteomyelitis must be distinguished from the two other jaw infections—alveolar abscess (p. 541) and, in particular, actinomycosis (p. 38). Sarcoma, though clinically similar, has distinct X-ray appearances.

Treatment.—If osteomyelitis is suspected clinically but not confirmed radiologically, *weekly X-rays* must be taken. As soon as bone necrosis is seen, *operation* is required to relieve tension and remove diseased and dead bone (sequestrum) (fig. 555). *Antibiotics* are given: (a) during the waiting period, providing one is not thereby lulled into a false sense of security (weekly X-rays must continue); (b) as an aid to operative surgery. Bone recovery (involucrum) is poor. It occurs in young patients, but after middle life there is hardly any regeneration and pathological fracture may occur.

Chronic Osteomyelitis.—Again the mandible is affected more often than the maxilla, and necrosis occurs in a number of ways.

(i) **After Apical Dental Infection,** *Alveolar Abscess, or Peridental Suppuration.*—Most patients are over fifty years of age. In 50 per cent. of cases dental extraction precedes the onset of symptoms. Many months may elapse before the patient seeks advice. X-ray will show local osteitis with sequestrum formation or a localised abscess (a Brodie's abscess similar to chronic osteomyelitis in long bones).

(ii) **After fractures.**

(iii) **Radiation and Chemical Necrosis.**—Radium and irradiation necrosis, though seemingly non-bacterial, has a large infective element, probably due to the spread of apical dental infection after the vascular thrombosis caused by the irradia-

Samuel Hall Wass, 1907–1970. Surgeon, Guy's Hospital, London.
Sir Benjamin Collins Brodie, 1783–1862. Surgeon, St. George's Hospital, London.

tion of tumours in the area. There is also a danger of necrosis if extractions are performed after radiation. 'Phossy-jaw' necrosis due to phosphorus poisoning, is rarely seen nowadays, since the crude element is no longer used in the manufacture of matches.

Tuberculous, syphilitic, and actinomycotic necrosis are rare. A hole in the hard palate following a gumma is an example of syphilitic necrosis (p. 48).

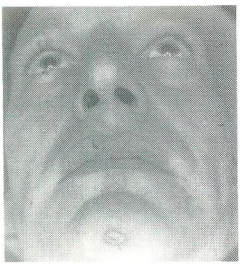


FIG. 556.—Median mental sinus.

Treatment.—Dependent drainage gives the best results. A suitable incision is made beneath the mandible, and a trough is chiselled in the bone, any sequestrum present being removed. The wound is left open and packed lightly with petroleum jelly gauze. Appropriate antibiotics are given.

Median mental sinus (fig. 556) is a clinical entity which is often diagnosed and treated incorrectly. It is produced by an apical abscess on one (or more) of the lower incisors which has penetrated the buccal cortical plate below the origin of the mentalis muscles. These muscles take origin from the labial surface of the alveolar process at or just above the bottom of the labial sulcus and diverge to be inserted into the skin of the chin. Thus pus which accumulated deep to mentalis muscles can escape to the surface only in the mid-line and consequently discharges through a sinus in the centre of the chin.

Often the offending tooth has become pulpless as a result of a blow so there is no obvious cause for necrosis of the pulp. The lower incisors should be tested for vitality to establish the causative tooth which preferably should be root filled as replacement of a single lower incisor is difficult. The sinus will close spontaneously once the infection is controlled.

(*c*) **Actinomycosis**

The cervico-facial type of actinomycosis (swelling of the soft tissues, p. 38) has clinical similarities to osteomyelitis of the mandible and must be considered in the differential diagnosis of inflammatory swellings of the jaw. There is swelling of the soft tissues with sinuses, with or without pain. The swelling is more brawny than in osteomyelitis, and the skin is bluish. External sinuses are common, being uncommon in osteomyelitis. 'Sulphur granules' may be seen in the discharge from the sinuses, but their absence, and the inability to grow the streptothrix actinomyces does not rule out this diagnosis. In fact, such a swelling with persistent negative X-ray appearances of the jaw, is considered to be actinomycosis and treated accordingly (p. 39).

2. THE ODONTOMES

Odontomes are cysts, tooth malformations, or tumours arising from the epithelial or mesothelial elements of the teeth, or sometimes from both. Only the three epithelial odontomes are common and important: (*a*) Dental cyst, (*b*) Dentigerous cyst, (*c*) Adamantinoma.

(*a*) **Dental cyst** occurs in connection with the root of a normally erupted, but chronically infected, usually pulpless, tooth, viz. :⟶ Epithelial cells, believed to be derived from the enamel-organ, proliferate and degenerate, forming a cyst. The cyst enlarges, causing expansion of the alveolus, and eventually most of the epithelial lining disappears. These cysts, which can appear at any age, are more frequent in the upper jaw, and when they attain a large size they encroach upon the antra or the nasal fossæ, but rarely open into these cavities. Usually the fluid in the cyst is clear, and it often contains cholesterol crystals ; as a

rule it is sterile, but secondary infection can occur. When not infected, the condition is painless.

(*b*) **Dentigerous cyst** (*syn.* follicular odontome) arises, usually in young adults, in connection with a non-erupted permanent tooth. The swelling consists of a cyst containing a tooth, most commonly an upper or a lower third molar, often well developed except for truncated roots, lying obliquely in a cavity filled with viscid fluid, viz:⟶

These cysts expand the outer table of the jaw even to causing egg-shell crackling. The stronger inner table prevents pathological fracture. Ridges of bone on the side walls cause a pseudo-trabecular or a soap-bubble appearance on X-ray.

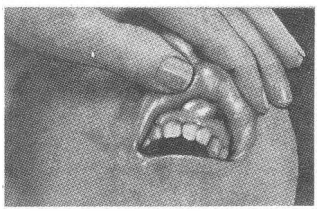


FIG. 557.—Naso-palatine cyst.

Treatment of Dental and Dentigerous Cysts.—The essential point in the operative treatment is the excision of the whole epithelial lining of the cyst. After preoperative dental hygiene, treatment of caries etc., cysts in the upper jaw or small cysts in the lower jaw are exposed through an intra-oral approach to the outer table by raising a muco-periosteal flap. An external approach is used for a large mandibular cyst. After excision the bone cavity is obliterated by a soft tissue 'push-in', or with bone chips, and the wound is sutured.

Naso-palatine Cyst (fig. 557).—A rare cyst, arising in connection with the naso-palatine canal and *not* with incisor teeth, which need not be sacrificed (Roper-Hall).

(*c*) **Adamantinoma** (*syn.* multilocular cystic disease; ameloblastoma) is an epithelial tumour possibly arising from ameloblasts (enamel-forming cells). It is more common in coloured races. It behaves pathologically and looks histologically like a basal-celled carcinoma (p. 120). (Willis believes it arises from the ectodermal epithelium of the stomadæum.) Unlike the other two odontomes just described, it is a locally invasive solid tumour, but undergoes multicentric cystic degeneration. Some of the loculi are separated by true bony trabeculæ. An adamantinoma will recur if simply curetted, but it does not metastasise to lymph nodes. It usually occurs in the third decade, nearly always affecting the mandible causing expansion, mostly of the outer table in the region of the molar teeth (fig. 558). It rarely causes pain. The cystic degeneration leads to areas of softening or egg-shell crackling.

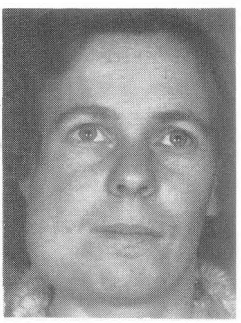


FIG. 558.—Adamantinoma of the mandible. (*A. H. Lendon, F.R.C.S., Adelaide.*)

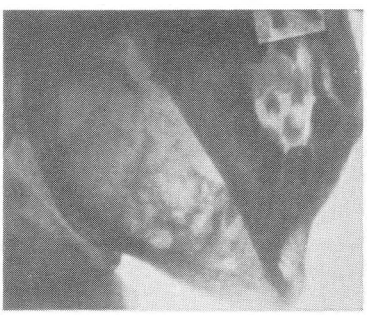


FIG. 559.—X-ray of adamantinoma (large loculi and small honey-combing). (*The late H. S. Wass, F.R.C.S., London.*)

An X-ray shows large loculi, but also a fine honey-comb appearance which is characteristic and distinguishes it from some dentigerous cysts or an osteoclastoma (fig. 559).

Treatment.—Evacuation and curettage of the cysts is followed invariably by recurrence. X-ray or radium therapy is without permanent value. Therefore resection of that portion of the jaw bearing the tumour, together with a margin of healthy bone, is essential. Before this is undertaken provision should be made for retention of the remaining portions of the bone in normal relationship by means of dental fixation. Provided no recurrence takes

Harry Thomas Roper-Hall, 1890–1969. Dental Surgeon, Birmingham and Midland Eye Hospital.
Rupert Allan Willis, 1898–1980. Professor of Pathology, Leeds, England.

place, after an interval of several months the mandibular defect can be bridged with a bone graft.

3. BONE TUMOURS OF THE JAWS (BENIGN)

(*a*) **The Fibro-osseus Group.**—The jaws are membrane bones; there is no cartilage. The ' tumours ' of this group are composed of varying proportions of fibrous tissue and bone, being hard if mainly bone, softer, if fibrous tissue, while myxomatous degeneration causes softening in parts. The condition can be localised or diffuse. Thus the variety of types and their descriptive names are many. For example, a mainly fibrous tumour is called *Monostotic fibrous dysplasia* if localised, or *Polyostotic fibrous dysplasia* if diffuse. If solely of bone and localised, it is an *Ivory osteoma* (fig. 560) or *Leontiasis ossea* if diffuse. Radiological appearances vary between uniform sclerosis and irregular mottling.

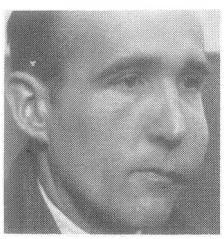


FIG. 560.—Ivory osteoma of the left maxilla. The tumour has been growing slowly for eight years.

The cause is obscure. Dental sepsis was once thought responsible (Leontiasis Ossea being due to a creeping periostitis). The behaviour of the ' tumours ' is unlike that of a true neoplasm, for they appear in the first or second decade and usually stop growing when the individual is fully grown. Thus Wass believed that they are aberrations of ossification in membrane. When growth has finished, the unsightly swellings of small local lesions can be removed under antibiotic cover.

Paget's Disease of the Jaws (fig. 561).—This condition can be part of a generalised Paget's disease, or confined mainly to the jaws. It may be confused with that diffuse bony variety of the fibro-osseus group, leontiasis ossea, but it occurs in older people, pain is often present, other bones are affected, and the radiological appearances of the teeth (if there are any) show apical club-shaped swellings (cementosis). As the need arises the alveolus and, if necessary, the anterior surface of the maxilla, is reduced in size by chiselling away the excessive bone formation.

Complication.—Sarcoma develops more often than is the case in other bones affected with Paget's disease.

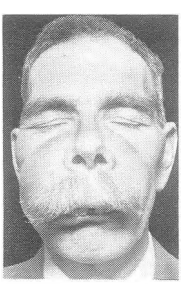


FIG. 561.—Paget's disease of the maxillæ. After partial resection of both upper jaws the patient emigrated to New Zealand.

(*b*) **Osteoclastoma and Giant-celled Reparative Granuloma.**—Both these conditions are rare. *Osteoclastoma*, besides occurring under the gum as a myeloid epulis (p. 543), can expand bone, though Wass encountered only one instance (in the maxilla) out of 350 jaw tumours.

Giant-celled reparative granuloma is often mistaken for an osteoclastoma (Jaffe). The lesion appears to be related to the occurrence of a hæmorrhage within the bone marrow. Somewhat rare, it occurs more frequently in females than in males, and nearly always between the ages of ten and twenty-five years, whereas a *bona fide* giant-celled tumour appears in patients between twenty-five and forty years of age. The lesion causes a painless swelling of the jaw, situated much more frequently in the mandible than in the maxilla.

Radiography.—There is a rounded or oval area of radiotranslucency (fig. 562). The granuloma thins and expands, but does not perforate the cortex.

Pathology.—Multinuclear cells are small, sparse, and distributed unevenly. Histologically it is difficult or impossible to distinguish this lesion from the so-called brown tumour of hyperparathyroidism.

Differential Diagnosis.—Hyperparathyroidism should be excluded by ascertaining the serum calcium level and by radiography of other parts of the skeleton (p. 647). It is also important to exclude an adamantinoma (p. 546), for the treatment

Sir James Paget, 1814–1899. Surgeon, St. Bartholomew's Hospital, London.
Henry Lewis Jaffe, Contemporary. Formerly Director of Laboratories, Hospital for Joint Diseases, New York, U.S.A.

of the latter condition must be more radical. At operation a giant-celled granuloma will be found to consist of opaque, semi-solid, dark red material, whereas an adamantinoma is filled with transparent fluid or jelly.

Treatment.—The treatment of choice is thorough curettage through an external incision without opening the bone cavity into the mouth. Should the condition recur, hyperparathyroidism must again be excluded.

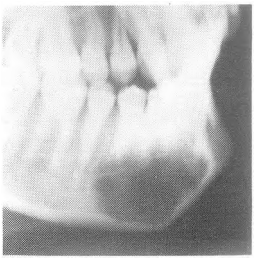


FIG. 562. — Radiograph showing a giant-celled reparative granuloma of the mandible. *(Dr. H. L. Jaffe, New York.)*

3. TUMOURS OF THE JAWS (MALIGNANT)

Malignant Neoplasms of the Maxilla.—The clinical diagnosis of 'malignant upper jaw' embraces a number of pathological conditions.

(*a*) **Osteogenic sarcoma** is usually of the round-celled variety; rarely it is a highly differentiated fibro-myxochondro-sarcoma.

(*b*) **Columnar-celled carcinoma**—of the maxillary antrum (fig. 563).

(*c*) **Squamous-celled carcinoma**—derived from the epithelium overlying the hard palate, from a tooth socket, or from the gum.

(*d*) **Invasion of the maxilla by a sarcoma of the ethmoid.**

(*e*) **Malignant Lymphoma of Africa (Burkitt Tumour)** (p. 195).

(*a*) **Osteogenic Sarcoma.**—While neither sex nor any age is exempt, this disease has a curious predilection for women about the age of forty. When of the periosteal variety—and this is the more usual—it is the *anterior* aspect of the jaw which is maximally affected, but the condition soon shows itself on the inferior or palatal wall as well. Pain, nasal obstruction, and epiphora[1] usually occur late in the disease.

(*b*) **Carcinoma commencing in the maxillary antrum** is much more frequent than the foregoing, and the age incidence of this group is similar to that of carcinomas elsewhere. These neoplasms are divided about equally between the sexes, and most cases are associated with secondary infection of the nasal sinuses. Growths that originate on the floor of the antrum frequently cause pain in the teeth, and a dental surgeon is consulted, but without relief of the pain; however, pain usually results in a comparatively early diagnosis. A foul, purulent and blood-stained discharge from the nose is common. Free bleeding associated with antral puncture should always suggest the possibility of carcinoma of the antrum. The main clinical features vary in accordance with the direction in which the tumour spreads:

FIG. 563.—Carcinoma commencing in the maxillary antrum.

If posteriorly, there is little if any alteration in contour of the face.

If the medial wall is implicated, there will be nasal obstruction and epiphora.

If the antero-lateral wall is involved, there will be evident swelling of the face (fig. 563).

If the floor of the antrum is transgressed, the hard palate will bulge.

If the roof is invaded, there will be proptosis and diplopia.

[1] Epiphora—Greek, *ἐπιφορά* = overflow. An abnormal overflow of tears down the cheek usually due to obstruction of the lachrymal duct.

Metastasis occurs relatively late, the upper jugular lymph nodes being the first to be affected, giving rise to a swelling beneath the angle of the jaw. A third of the cases have metastases when they are first seen.

Radiography.—In the early stages there is opacity and increase in size of the antrum. Later decalcification of the bony walls is visible.

Biopsy.—All nasal polypi should be submitted to microscopical examination. Unfortunately cases in which polypi are present are comparatively few. Fragments of the neoplasm may be washed out by antral lavage and solid matter, after centrifuging, should be examined microscopically. As a rule, a diagnostic Caldwell-Luc operation (p. 552) is necessary to obtain biopsy material, and as histological examination is a cardinal step in diagnosis, no effort should be spared in obtaining a representative specimen.

Treatment.—Collaboration between surgeon and radiotherapist is essential.

Combined Irradiation and Conservative Surgery for Carcinoma of the Maxilla.—In several clinics the following plan is adopted:

(*a*) A course of high-voltage irradiation or gamma rays from a teleradium unit is given.

(*b*) Six to eight weeks after the conclusion of the irradiation, by which time the inflammatory reaction has abated, palatal antrostomy is performed. The purpose is to remove any residual irradiated growth and to discover whether and where any viable malignant tissue remains by taking a series of geographic biopsies. It also allows easy follow-up inspection of the cavity.

The plate of bone forming the antro-nasal wall is nibbled away. The inferior and middle turbinate bones and the tissue involved in the growth are removed with a diathermy needle, if possible in one piece; doubtful areas are coagulated. A pack or plastic obturator is inserted. The patient is given antibiotic therapy for a week.

(*c*) In 30 per cent. of cases the tissue removed shows no evidence of malignancy, and nothing more is required. When the tissue removed at operation shows evidence of active malignancy a hollow plastic applicator, made by a dental surgeon, is filled with wax and drilled so as to take radium tubes. In this way further irradiation is given. If the post-ethmoidal or sphenoidal regions are involved, radium needles are inserted directly into those areas.

(*d*) A block dissection of the neck is undertaken if lymph nodes are palpable (p. 610).

(*e*) A permanent obturator bearing teeth is fitted at least three months after operation.

If recurrences occur they may be treated by diathermy excision, intracavity irradiation or infusion with *cytotoxic drugs* (p. 701). The overall five-year survival rate is 35 to 50 per cent.

Excision of the Maxilla.—Since the introduction of intratracheal anæsthesia the operative mortality is low. There is surprisingly little deformity after this formidable operation, especially if a prosthesis is constructed by a dental expert as soon as the wound has granulated. Such treatment offers hope of a cure. Whether surgical excision should precede or follow irradiation is still undecided, as comparable results are obtained by either method (Maxwell Ellis).

Maxwell Philip Ellis, Contemporary. Formerly Surgeon, Royal National Throat, Nose and Ear Hospital, London.

MALIGNANT NEOPLASMS OF THE MANDIBLE

The mandible is rarely the site of a primary malignant neoplasm or of a metastasis. However, it becomes directly involved by an advancing primary carcinoma of the tongue or of the floor of the mouth. Sometimes an advanced carcinoma of the lip spreads to this bone through the mental foramen. A third method of neoplastic involvement occurs when the facial lymph node (Stahr's gland) is the seat of a secondary deposit.

Treatment.—So regularly does irradiation cause necrosis of the mandible, that when the bone is involved by growth, it is usually necessary to resect a portion or perform hemi-mandibulectomy. If the chin itself can be spared, gross deformity is avoided.

SARCOMA OF THE ETHMOID

Sarcoma of the ethmoid is rare. As it expands it widens the space between the orbits and flattens the nasal bones, producing that clinical entity, the ' frog-faced man '. Still later the superior maxillæ are invaded.

Treatment.—The treatment by combined irradiation and operation does not differ in principle from that described for carcinoma of the maxillary antrum. When the air sinus is infected, drainage is essential before irradiation is commenced.

INFECTION OF THE MAXILLARY ANTRUM

Surgical Anatomy.—The maxillary antrum (of Highmore) is rudimentary at birth and attains full development by the age of twelve years. Lined with ciliated epithelium, it communicates with the middle meatus of the nose by a small ostium situated high on its medial wall (fig. 564). The apices of the roots of the second premolar and the first and second molar teeth are in close apposition to the floor of the antrum being separated only by periosteum and mucous membrane. Rarely, the first premolar and the canine teeth are related similarly.

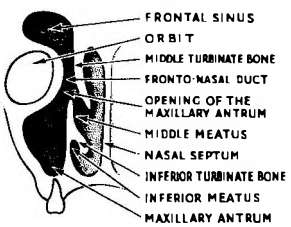


FIG. 564.—Coronal section of the maxillary antrum.

Maxillary sinusitis may be unilateral or, much less frequently, bilateral. Infection occurs as an extension from the nose. One epidemic of acute respiratory infection (common ' cold ') brings as an aftermath many cases of infection of the accessory nasal sinuses, of which the maxillary antrum always heads the list; while another epidemic, in all other respects similar, is free from this complication. A less frequent cause is penetration of the floor of the antrum by bacteria from a periodontal abscess connected with a carious tooth; also entry of infected water during swimming.

Acute Empyema Antri.—As a rule the general symptoms are severe, especially when the pus is confined by occlusion of the natural ostium. Pain and tenderness are present over the affected maxilla; sometimes the pain is referred along one of the divisions of the trigeminal nerve. Breathing through the nostril on the side of the lesion is impaired and often obstructed. If the ostium is patent, which is unusual in acute cases, a flow of pus can be obtained when the head is held downwards and forwards with the affected side uppermost.

Transillumination of the antrum and a radiograph (fig. 565) are likely to reveal a relative opacity of the affected antrum.

Treatment.—This includes bed rest, analgesics, ephedrine 1 per cent. in saline by a nebulizer, menthol inhalations, and systemic chemotherapy if

Hermann Stahr, 1869– . Director of Pathology Institute, Danzig, Germany. Described the inconstant lymph node on the facial artery as it crosses the mandible in 1899.
Nathaniel Highmore, 1613–1685. General Practitioner, Sherborne, Dorset, England. Published a treatise on anatomy in 1651.

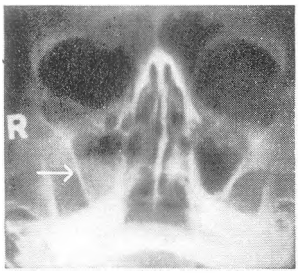


FIG. 565.—Radiograph showing pus in the right antrum of Highmore.

the temperature is over 38°C. Puncturing the antrum is for diagnostic and therapeutic purposes. An antral trocar and cannula punctures the antrum beneath the inferior turbinate bone after application of surface anæsthesia. Pus is aspirated and washouts are performed with normal saline.

Chronic Empyema Antri.—Pain and swelling are often absent, and frequently the only symptom is an intermittent flow of pus from the nostril on the affected side. When the ostium is patent, the information gained by an examination with a nasal speculum may be diagnostic.

Treatment.—Purulent maxillary sinusitis not responding to full medical treatment and antral lavage warrants adequate drainage:

Caldwell-Luc Operation.—This is a good standard procedure, though not advocated in children as dentition may be affected. Under general anæsthesia, with oral intubation and packing, an intra-oral incision 2·5 cm long is made in the buccolabial sulcus, centred over the canine fossa. The muco-periosteum is elevated and a gouge is used to make an opening about 12 mm in diameter through the anterior wall of the antrum (fig. 566). All the mucous membrane is removed with Luc's forceps (fig. 567) and any diseased ethmoid cells can be exenterated. An intranasal antrostomy completes the procedure, allowing post-operative washouts.

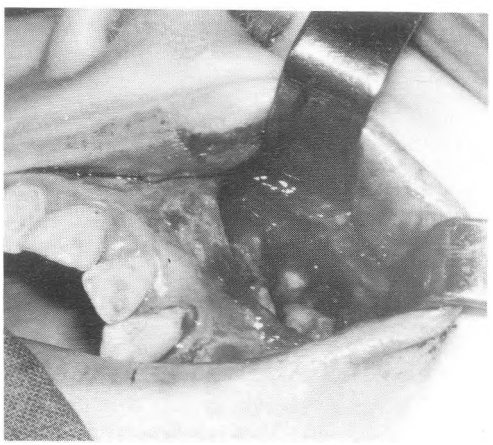


FIG. 566.—Caldwell-Luc operation (see text). *(H. Holden, F.R.C.S., London.)*

Intranasal Antrostomy is performed in children in order to avoid affecting dentition, and as an operation of compromise in some patients with recurrent sinusitis. Under a general anæsthetic, a piece of the medial wall of the antrum is removed to allow drainage into the nose.

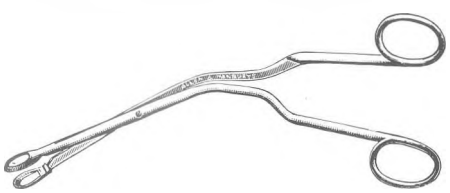


FIG. 567.—Luc's ethmoidal forceps.

Nasal Polypi. — Nasal polypi (œdematous ethmoid sinus mucosa) are usually multiple and are seen to prolapse from the ethmoid cells in the region of the middle turbinate (fig. 564). They are recognised by their glistening gelatinous appearance when light is focused upon them.

They are practically confined to adults, and patients complain of nasal obstruction, nasal discharge, and some loss of smell. Many of the sufferers

George Walter Caldwell, 1866–1946. Otolaryngologist who practised successively in New York, San Francisco and Los Angeles. Described his operation for treating suppuration of the maxillary antrum in 1893.
Henri Luc, 1855–1925. Otolaryngologist, Paris. Described his operation in 1889.

are allergic to dusts and pollens which may initiate attacks of sneezing and rhinorrhœa.

Treatment.—Single polypi are removed under local surface anæsthesia with a snare and appropriate forceps (fig. 567). Multiple polypi usually require a general anæsthetic for more complete removal. Associated ethmoidal or antral procedures can be then undertaken at the same time. Antihistamines should be used after removal, since recurrence is common.

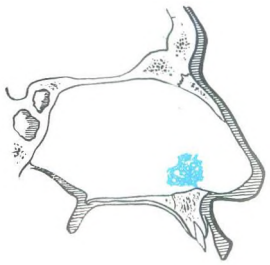


FIG. 568.—Little's area.

EPISTAXIS

Source and Cause of the Bleeding.—The bleeding may be arterial or venous. In 90 per cent. of cases it comes from a plexus of veins (sometimes varicose) situated in Little's area (fig. 568) on the antero-inferior portion of the septum. The most frequent cause is nose-picking—epistaxis digitorum. Other causes include trauma, nasal infection, neoplasms, hypertension, blood dyscrasias, acute specific fevers, nephritis and uræmia.

Treatment.—Sit the patient up so that blood does not run down the throat. Blow nose to remove clots. Pinch the nose for ten minutes. An ice-pack or cold sponge applied to the nose is often helpful.

Cauterising the bleeding-point with trichloracetic acid, 50 per cent., or electric cautery, is undertaken if the bleeding-point can be seen.

Sedation with diazepam or Omnopon and admission to hospital are necessary if bleeding is uncontrolled. Blood transfusion may be required.

Packing: *Anterior Packing.*—After anæsthetising the mucous membrane with 4% xylocaine, ribbon gauze coated with B.I.P.P. (Bismuth Iodoform Paraffin Paste) is inserted so as to fill the nasal cavity, and if the bleeding is from the septum the nasal cavity of the opposite side is packed also. Antibiotics are given to combat infection.

Posterior Packing.—The insertion of a cone-shaped gauze tampon (fig. 569) moistened with B.I.P.P. is the most satisfactory, although a quick method is to insert a Foley-type catheter as far as possible to the back of the nasal cavity and inflate the balloon. The pack can be left in for five days if an antibiotic is administered.

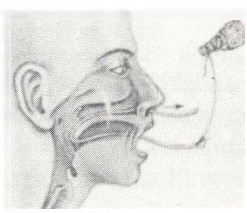


FIG. 569.—Method of inserting a posterior nasal tampon (Dickie's method).

Arterial Ligation.—When the above methods fail, or profuse hæmorrhage recurs, it is likely that the source of the bleeding is more posterior. Ligation of the anterior ethmoidal artery in the orbit is indicated, particularly in traumatic cases (fig. 570). Trans-antral ligation of the internal maxillary artery, and ligation of the external carotid artery are other methods which are sometimes called upon.

DEFLECTED NASAL SEPTUM

Although the majority of adults do not have a completely straight septum, it is only the grosser deflections causing symptoms which require correction. Septal deformities are caused either by trauma or by disproportionate growth during development. They may produce nasal obstruction or headaches in the region of the nasion.

The submucous resection (S.M.R.) operation consists of the removal of cartilaginous and bony spurs from between the two coverings of mucoperichondrium and mucoperiosteum. This allows the septum to take a more midline position.

Nasal Bone Fractures (p. 534).

FIG. 570.—Incision for the approach to the anterior ethmoidal artery.

James Lawrence Little, 1836–1885. Professor of Surgery, University of Vermont, Burlington, Vermont, U.S.A.

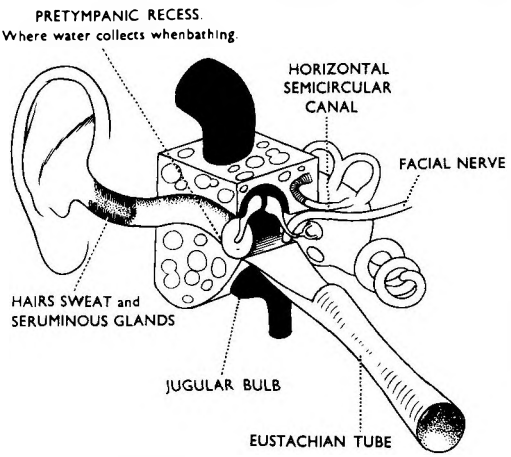


FIG. 571.—Diagram showing the surgical anatomy of the ear.

THE EAR

Now follows a brief presentation of well-known afflictions of the ear. The reader is referred to the appropriate text-books for fuller accounts and descriptions of operative surgery.

Surgical Anatomy (fig. 571).—The ear consists of four parts: (1) Pinna, (2) External auditory canal, (3) Middle ear, (4) Inner ear and Eighth nerve track.

The pinna is cartilaginous, covered by stratified squamous epithelium. The external auditory canal is in two parts: the outer cartilaginous part, which is lined by similar epithelium, tapers to its narrowest part where it joins the bony external auditory canal. This is lined by a thin stratified epithelium containing no glands or hair follicles, and widens medially until closed off by the tympanic membrane at an angle of 60° to the vertical.

The middle ear is part of the mastoid bone. Above, is the attic connected by the aditus to the mastoid antrum and variable air cells. Below, it communicates with the nasopharynx via the Eustachian tube. It contains (*a*) three ossicles: malleus, incus, and stapes; (*b*) two muscles—tensor tympani and stapedius; (*c*) two windows—oval and round; (*d*) one nerve—chorda tympani. The facial nerve traverses the superior and posterior wall in its bony canal. The horizontal semicircular canal lies in the attic.

Congenital Abnormalities.—These are: partial or complete absence of pinna; partial or complete occlusion of external auditory canal; partial or complete absence of middle ear—the stapes is nearly always present. Treatment is surgical reconstruction.

EXTERNAL EAR

Wax.—This is best removed by syringing with warm water and bicarbonate of soda solution (1 per cent.) after appropriate oily drops have been introduced for the previous two nights. An aural syringe with a small nozzle must be used, and directed upwards and backwards. An intact drum is unlikely to rupture if these rules are followed.

Foreign bodies easily become wedged in the narrow part of the meatus, whence they can be removed using a hook, sucker or fine forceps under indirect lighting or under the surgical microscope.[1] This should be done by someone familiar with such procedures, especially in children. In difficult cases, a general anæsthetic may be required.

Inflammation.—Inflammation of the external canal may be localised (furuncle) or widespread (external otitis).

Furuncles are staphylococcal in origin, and produce pain, pyrexia, periauricular swelling and, characteristically, a painful pinna on movement.

Treatment.—Decongestive drops or wicks (e.g. glycerine and icthyol or

[1] Celsus (see p. 26) recommended tying the patient on a wooden plank with the affected ear downwards and hitting the plank with a hammer.

antibiotic hydrocortisone cream). If symptoms are severe or generalised (pyrexia), broad spectrum antibiotics are required.

External otitis (*syn.* telephonist's ear, Singapore ear) is often fungal and secondary to dandruff or athlete's foot. Irritation, discharge, and minimal deafness are the symptoms—secondary bacterial infection introduced by scratching the ear leads to further discharge and pain. Antifungal preparations combined with a local antibiotic either as drops or cream is indicated. The primary cause, scalp or toes, must be treated at the same time and the ears kept dry. Mild sedation, e.g. Phenergan 25 mg may be advisable.

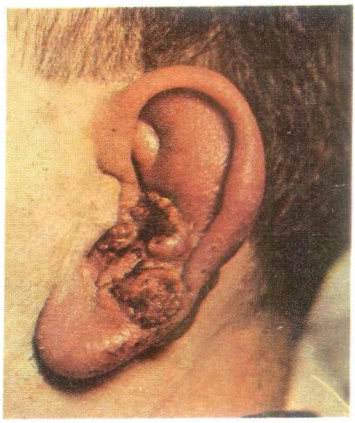


FIG. 572.—Carcinoma of the pinna.

Carcinoma of the Pinna (fig. 572) is of the squamous-celled type, and the condition is often comparatively advanced before the patient seeks relief. However, if treated early, favourable results accrue from excision of a part, or the whole of the pinna. Acrylic and rubber prostheses effectively replace the lost part.

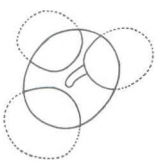


FIG. 573.—Bony exostoses of the meatus, as seen through the auroscope.

Bony Exostoses.[1]—Bony exostoses of the deep meatus produce a bizarre appearance (fig. 573). Only if the remaining chink past them is very small is removal indicated to alleviate deafness. They are removed by elevating the meatal skin and drilling away the bony exostosis. There is a potential danger to the facial nerve.

MIDDLE EAR

Note: glue ear and acute otitis media in children are conditions which are encountered very commonly.

'Glue ear', more correctly called seromucinous otitis media, has become an increasingly common disorder in children. It usually affects those with recurrent otitis media and often enlarged infected adenoids, presumably because of poor Eustachian tube function. It is especially common in children with cleft palate (even after repair) as the muscles which open the tube have lost their insertion into the middle of the palate and atrophied. Inadequate antibiotics for acute infections is another possible cause. 'Glue ear' is painless, and simply causes conduction deafness, of which it is the commonest cause in childhood. The appearances of the eardrum may be striking (gross retraction, yellow or grey/blue discoloration, increased vascularity), but more often are not. The condition must therefore be suspected from the history.

Treatment.—An antihistamine/decongestant mixture is usually tried first, but if this fails to clear the condition, adenoidectomy, myringotomy (fig. 574) and grommet insertion (fig. 575) are advised. In cleft palate cases the adenoids are never removed, however, lest nasal air escape during speech be worsened.

[1] Supposed to afflict those who indulge in under-water swimming.

Acute otitis media is secondary to an upper respiratory infection, and presents as severe prolonged earache with pyrexia and systemic illness. In some very acute cases, pus may discharge through a perforation before treatment can be started. More usually, the eardrum is reddened in appearance.

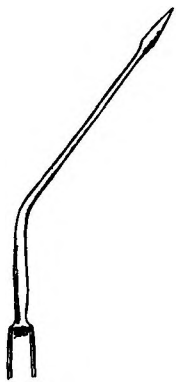


FIG. 574. — Myringotomy knife.

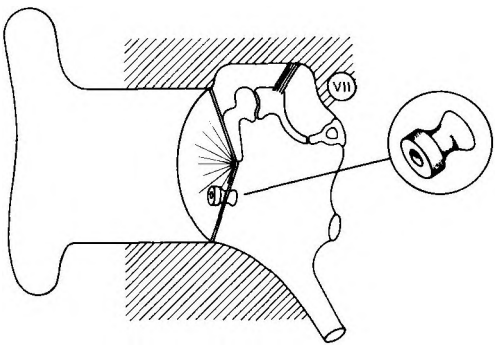


FIG. 575.—Grommet tube positioned in a anterior-inferior quadrant of drum.

Treatment.—Antibiotics are given in full dosage for five days; nasal decongestants may be added. Early antibiotic treatment has meant that myringotomy (incision of the eardrum) is rarely required nowadays.

Complications. Acute mastoiditis and intracranial complications have become rare since the widespread use of antibiotics.

Recurrent otitis media.—Frequent attacks of otitis media in childhood are an indication for adenoidectomy.

Acute Mastoiditis.—The symptoms are pain deep in the ear, periauricular swelling, pyrexia. The pinna is not painful on movement (as distinct from a furuncle). Conduction deafness is marked.

Treatment.—Pus must be let out. Therefore a simple mastoidectomy is performed, with an adequate course of antibiotics in support.

The operation entails the removal of the infected air-cells through a posterior auricular incision. The middle ear is not disturbed. The wound is drained for twenty-four hours.

Complications are lateral sinus thrombosis, extradural abscess, meningitis, cerebral or cerebellar abscess, facial palsy, and, rarely, labyrinthitis.

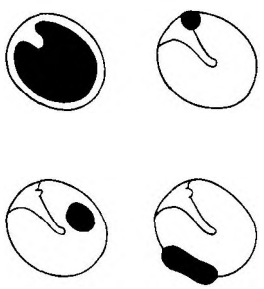


FIG. 576.—Types of perforation of tympanic membrane in chronic otitis media.

Chronic Otitis Media.—This term simply means that there is a perforation of the eardrum. If the ear is constantly discharging scanty offensive pus through a posterior or superior perforation (' unsafe type ', fig. 576 right upper and lower diagrams), modified radical mastoidectomy may be required, especially if cholesteatoma is present (see below).

A dry central perforation (' safe type ', fig. 576 left upper and lower dia-

grams) which discharges intermittently with colds or swimming, is treated by antibiotic/steroid drops when wet, and may be repaired when dry by grafting of the eardrum (myringoplasty, fig. 577) if necessary with reconstruction of the ossicular chain (tympanoplasty).

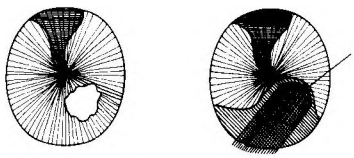


FIG. 577.—Myringoplasty. (*a*) Central perforation. (*b*) Epithelium elevated and fascial graft inserted to cover perforation. The epithelium is then replaced over fascia.

Tympanoplasty (fig. 578).—Operations and procedures under this group are designed to repair or reconstruct the tympanic membrane and ossicular chain of the

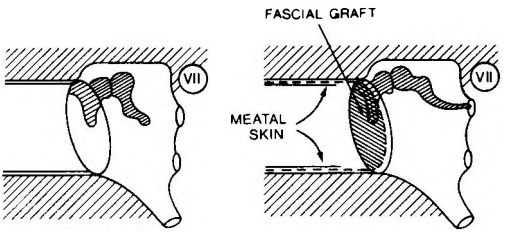


FIG. 578.—Tympanoplasty (a) Subtotal perforation with erosion of ossicles and loss of stapes. (b) Repair with fascia and ossicular continuity re-established by incus repositioning.

middle ear. Various tissues have been used including homografts from cadavers and animals but the most widely accepted are autogenous fascia with repositioned ossicles or shaped bone chips.

Complications. — Conduction deafness with ossicular chain destruction, labyrinthitis, facial palsy, cerebral meningitis, cerebral and cerebellar abscess. A *cholesteatoma* (skinball), whose origin is debatable, produces otorrhœa and the above complications by pressure necrosis. *Treatment* is as above, with the removal of all the lining membranes.

Ruptured drum is due to trauma, e.g. hard slap on ear or syringing. There is sudden pain and deafness, with tinnitus and bloodstained discharge.

Treatment.—Do not touch. No eardrops. No antibiotics required.

Otosclerosis produces bilateral conduction deafness. Common in females, it is often hereditary, and may be exacerbated by pregnancy.

Treatment by hearing aid or operation—stapedectomy with Teflon (or wire) strut and vein (or fat) graft (fig. 579). This produces improvement permanently in 90 per cent. and it has replaced the fenestration operation.

Serous Otitis Media is usually found in adults and, in contrast with ' glue ear ', the middle ear effusion is thin and watery; like ' glue ear ', however, it causes conduction deafness. It may result from viral upper respiratory infection or from nasopharyngeal carcinoma (causes of Eustachian tube obstruction), or from barotrauma (sudden change in atmospheric pressure produced by flying or diving). Treatment is by decongestants and, if persistent, by myringotomy and grommet insertion (fig. 575).

Malignant Tumours.—Excluding the pinna (fig. 572), **squamous-celled carcinoma**, though uncommon, can arise in the middle ear or mastoid cells. There is pain and otorrhœa (which may be long-standing), becoming bloodstained. Deafness is variable. Biopsy will confirm. Treatment is by combined surgery and radiotherapy, but the prognosis is poor.

Glomus jugulare tumours are rare but of special interest as they resemble carotid body tumours (p. 608). Slow growing and locally invasive, they are highly vascular, with nests of epithelioid cells and fibrous tissue. Early signs are seventh nerve palsy,

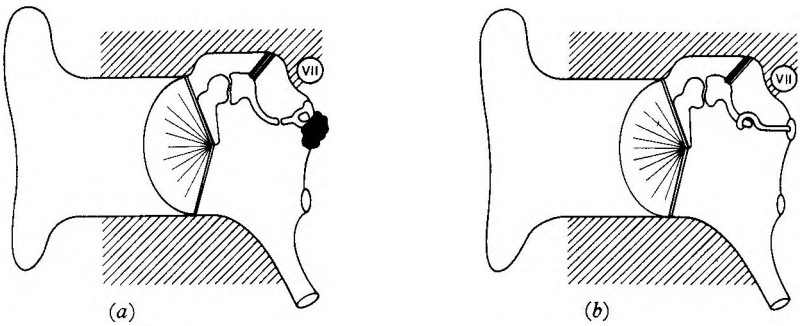


FIG. 579.—Stapedectomy. (*a*) Condition prior to removal of stapes and footplate. (*b*) After removal of stapes; sound conduction restored by a Teflon prosthesis.

pulsatile tinnitus, and free bleeding. Radiotherapy diminishes both size and vascularity. Cryosurgery is effective for small tumours.

THE INNER EAR

Presbyacusis[1].—High-tone bilateral perception deafness gradually affects lower tones with advancing years. It is often associated with atherosclerosis. Treatment is by a hearing aid.

Cochlear Concussion.—Hearing at 4,000 Hz is commonly affected, but if concussion is repeated daily (e.g. gunfire, road-driller, or aircraft pilot not using ear defender), the loss spreads up and down. Failure to hear the telephone bell is often the first symptom.

Menière's syndrome is unilateral perception deafness, intermittent true rotational vertigo associated with nausea and tinnitus. It may be associated with hyper- or hypo-tension, intracranial lesions, syphilis, or disseminated sclerosis. But if all these are excluded, and labyrinthine function tests show loss of function on one side, it is true Menière's disease.

Treatment may be (*a*) medical—Tab. nicotinic acid 100 mg, t.d.s. (vasodilator), Avomine, Stemetil, or (*b*) surgical—1. Operations to reduce endolymphatic pressure by a surgical approach to the endolymphatic sac for drainage can prove satisfactory. Also relatively minor procedures such as ultra-sonic or cryosurgery applied to the labyrinth may indirectly produce a similar result.

2. Labyrinthine destruction. A labyrinthectomy may be indicated in cases where the hearing is severely impaired and the patient is getting vertigo symptoms not controlled by other means.

Congenital Deafness.—This is due to intrauterine viral toxins in the first three months of pregnancy (e.g. rubella, influenza, etc.).

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[1]Presbys (Gr.) = old.

Prosper Menière, 1799–1862. Physician, Institute for the Deaf and Dumb, Paris. Described this condition in 1861.

RECENT ADVANCES

Otoneurosurgery has advanced temporal bone surgery towards the adjacent intracranial cavities; it involves teamwork between otologist and neurosurgeon in the treatment of:

Acoustic neuroma, presenting with unilateral perception deafness and unsteadiness; later, the Vth nerve is involved (numbness of the face, loss of the corneal reflex), and later still there is headache caused by raised intracranial pressure. Removal is by the translabyrinthine or posterior fossa approach.

Facial paralysis, which may follow temporal bone fractures; the facial nerve can be explored in its first portion, decompressed, and grafted if necessary.

Intractable vertigo which has not responded to the treatment outlined above may require vestibular nerve section. Both the facial and the vestibular nerve may be approached by the translabyrinthine route (if there is no cochlear function) or via the middle cranial fossa (if there is normal or near-normal hearing).

CHAPTER 32

THE MOUTH. THE CHEEK. THE TONGUE

Stomatitis

Stomatitis is a general term applied to inflammatory, erosive and ulcerative conditions widely affecting the mucous membranes which line the oral cavity. Gingivitis (p. 542) refers to inflammatory, erosive and ulcerative conditions which are confined to the mucoperiosteum covering the alveolar processes. Some inflammatory conditions of the gums may spread to involve other parts of the oral mucous membrane, in which case the term gingivostomatitis is used.

The mucous membrane which covers the alveolar process, the retromolar region and the hard palate is keratinised, firmly attached to the underlying periosteum and bone and is relatively resistant to injury. It is described as the masticatory mucosa. The papillated mucosa of the dorsum of the tongue is also keratinised and tough and firmly attached to the underlying muscle. Certain diseases have a characteristic distribution in relation to the different types of mucosa lining the oral cavity.

Infecting Organisms.—Many organisms are to be found in the oral cavity where, under normal circumstances, the majority are harmless to the individual. The number of such organisms is controlled, and their ability to cause harm is reduced, by a variety of factors such as:—

1. The regular desquamation and replacement of surface cells. By this means microscopic damage to the surface is repaired and many organisms are carried away on the shed cells.
2. The constant washing of the oral cavity by saliva so that organisms are swallowed and destroyed in the stomach.
3. The mild antibacterial activity of the saliva.
4. The health and integrity of the lining epithelium. Certain oral commensals are *facultative pathogens* and will take advantage of any weakness in the defences of the oral mucosa to produce a localised or generalised infection of the mouth. Certain other organisms produce specific infections and are *true pathogens*.

The surface of an ulcer in the oral cavity, irrespective of its cause, is soon colonised by facultative pathogens such as oral streptococci, staphylococci and occasionally Vincent's organisms. There is a resultant non-specific, acute, inflammatory reaction in the tissues forming the floor of the ulcer. For this reason a microbiological examination of swabs from oral ulcers and a biopsy is not often helpful as a means of establishing the diagnosis. Where necessary, secondary infection of oral ulcers may be treated by an 0·2% aqueous solution of chlorhexidine gluconate which is used as a mouthwash four times a day.

Predisposing Factors.—1. *The patient's general health and nutrition* are reflected in the state of the oral mucosa. Anaemia associated with vitamin B_{12}, folic acid and iron deficiency all lead to thin, atrophic, epithelia and loss of the papillae from the dorsum of the tongue. The mucosæ are easily damaged during mastication so that the patient suffers from recurrent ulceration of the insides of the lips and cheek. Lack of several other B vitamins can lead to loss of the papillae on the dorsum of the tongue. All such patients may present with a complaint of a red, burning tongue

Jean Hyacinthe Vincent, 1862–1950. Professor of Epidemiology, Val-de-Grace Military Hospital, Paris.

and angular cheilitis. Both the sore tongue and the angular cheilitis are due to secondary infection by candida albicans. A similar condition of the mouth is seen in severe protein deficiency (kwashiorkor[1]) and in sprue. Cancrum oris is another serious condition associated with malnutrition.

2. *Other factors affecting the health and efficiency of the epithelium* may lead to recurrent ulceration, or chronic candida infection. Several inherited conditions, such as epidermolysis bullosa result in abnormalities of both skin and oral mucous membrane. Epidermolysis bullosa leads to bullæ which, in the moist environment of the mouth, soon rupture to form ulcers. In severe forms, gross scarring occurs with adhesions which obliterate the sulci. Hyperkeratotic patches, due to any cause, may be invaded by candida albicans. Cytotoxic drugs affect the ability of the epithelial cells to reproduce so that the epithelium is abnormally thin and easily damaged. Repair is also slow because epithelialisation of the resultant ulcers is delayed.

3. *A reduced ability to deal with secondary infection* may lead to recurrent ulceration or the persistence of ulceration of the oral mucosa which is the result of minor trauma. Cyclical leucopænia, agranulocytosis, aplastic anæmia and hypogammaglobulinæmia are conditions which predispose to recurrent oral ulceration in this way. Patients who are taking adrenal cortical steroid drugs tend to develop thrush.

4. *An immune or auto-immune mechanism* is believed to underlie a variety of conditions affecting the oral mucous membranes. Damage to epithelial cells can result in a slough which separates to form an ulcer. Such a mechanism has been invoked to explain recurrent aphthous ulcers. Damage to cell membranes and intercellular cement substance can lead to intra-epithelial bullæ as in pemphigus. If the basement membrane is damaged subepithelial bullæ or blood blisters may result as in benign mucous membrane pemphigoid. Damage to submucosal vessels leads to thrombosis and a deep-penetrating ulcer as in *Behçet's disease*. Indirect immunofluorescent studies with serum from patients with pemphigus may demonstrate antibodies in the intercellular region in the epithelium of tagged monkey œsophagus. Direct immunofluorescence studies on biopsy material will demonstrate antibody on the basement membrane in benign mucous membrane pemphigoid.

If a biopsy is used in the diagnosis of these conditions a specimen should be taken which includes the adjacent mucous membrane. The fragment of tissue should be handled with particular care as the epithelium will separate readily from the underlying tissues, rendering it useless to the histopathologist.

Lichen planus, for which an auto-immune mechanism forms a less certain explanation, may also produce widespread oral erosions, submucosal bullæ and frank ulceration in addition to the more familiar lace-like hyperkeratotic lesions.

An immune reaction often in response to a drug such as sulphonamide or phenobarbitone underlies the severe bullous and ulcerative stomatitis which is seen in erythema multiforme and usually occurs in association with the characteristic target-like skin lesions.

5. *Certain chemicals* predispose to, or give rise to, a stomatitis. Inhalation of mercury vapour results in mercury poisoning, the ill effects of which include redness and swelling of the gums, and loosening of the teeth. Necrosis of the bone may follow. Chronic lead poisoning results in the appearance of a blue-black line at the gingival margin due to the deposition of lead sulphide in the tissues. The sulphur which reacts with the circulating lead to form the insoluble sulphide comes from bacterial activity in food debris at the gingival margin. Now bismuth is not used to treat syphilis, the similar black or purple bismuth line is no longer seen. In each of these conditions a secondary Vincent's gingivitis is prone to arise. The excessive ingestion of iodides leads to a sore mouth and excessive salivation.

Particular Types of Stomatitis

Aphthous Stomatitis.—This term is now applied to three specific entities: recurrent minor aphthous ulceration, recurrent major aphthous ulceration and herpetiform aphthous ulceration.

[1] The name means ' the red boy ' or ' the disease the child gets when the next baby is born ' in the Ga language of Ghana.

Hulusi Behçet, 1889–1948. Dermatologist, Istanbul, Turkey.

Minor aphthæ appear in crops of between one and many ulcers on a cyclical basis. The ulcers are up to 0·5 cm across, round or oval in shape, with a yellow base and a red erythematous margin (fig. 580). They are distinctly painful, occur in the unkeratinised mucosa of the cheek, lips, soft palate and floor of mouth and normally heal within 10–14 days. The frequency of the ulcerative episodes varies considerably. They occur more frequently in women than men and may then coincide with the second half of the menstrual cycle.

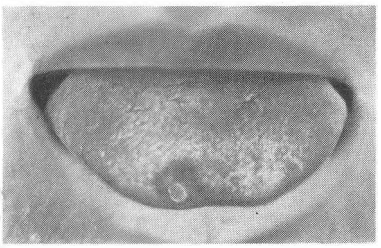

FIG. 580.—Aphthous ulcer of the tongue.

Major aphthæ are in many ways similar, but are usually larger and deeper. As a consequence they may enlarge to involve the keratinised mucosæ, take longer to heal and do so with scarring.

A chlorhexidine gluconate mouthwash will help many sufferers. Major apththæ can be coated, after the use of the mouthwash, with triamcinolone acetonide in Orabase; a pectin material which adheres to the moist surface of the ulcer and protects it (Adcortyl in Orabase). Choline salicylate gel can be applied to minor ulcers to relieve pain. The disappearance of pain from the ulcers heralds rapid healing.

If the patient has genital and conjunctival lesions as well as severe oral ulcers, the possibility of *Behçet's syndrome* should be considered. In *Reiter's syndrome* there is urethritis, arthritis, periarteritis nodosa, conjunctivitis and oral ulcers. The latter may either resemble minor aphthæ or the erosions seen in bullous lichen planus. They are noticeably less painful than aphthous ulcers and respond to tetracycline.

Herpetiform aphthous ulcers are quite small; only 1–2 mm in diameter. They occur in crops of many ulcers. They are probably of a different aetiology to minor and major aphthous ulcers. Despite their name they are *not* caused by the herpes simplex virus. 0·2% chlorhexidine or a tetracycline mouthwash will lead to rapid healing.

Herpes Simplex Infections

For most individuals the primary infection with the herpes simplex virus is subclinical. In others it results in a marked gingivostomatitis which usually occurs in infancy or childhood, but may also occur during adolescence or early adult life. Many small vesicles appear and rapidly break down to form small, yellow ulcers with bright red margins. They occur on the gingivæ, cheeks, lips and tongue. Lesions may also occur around the nostrils and even on the skin of the cheek. The patient is unwell, febrile and has markedly swollen submandibular lymph nodes. A soft diet, plenty to drink, an analgesic elixir and the gentle swabbing of the ulcers with 0·2% aqueous chlorhexidine is all that usefully can be done until the child recovers naturally in about 10–14 days.

Herpes labialis, which appears in response to cold winds, bright sunlight,

Hans Reiter, 1861–1969. President of the Health Service and Honorary Professor of Hygiene, Berlin.

or febrile illnesses is due to periodic reactivation of the virus. Parents who suffer from herpes labialis should avoid kissing their children when they have active lesions for obvious reasons. Doctors, dentists and nurses should be careful how they handle the lips and mouths of affected patients, lest they develop a herpetic whitlow or transfer the virus to their eyes to produce a herpetic keratitis.

Other Virus Infections.—Intraoral bullæ and painful ulceration can occur in herpes zoster. Small bullæ and ulcers also appear in the mouth, though mainly on the soft palate, in chicken pox. Ulceration of the soft palate and fauces occurs in both herpangina (due to coxsackie group A3 virus) and hand, foot and mouth disease (coxsackie group A16 virus) but the latter is not to be confused with the foot and mouth disease of cattle which only rarely affects humans. Small bullæ are also found on the palms of hands, soles of the feet and gingivæ in hand, foot and mouth disease which tends to occur in localised outbreaks affecting several individuals. A large sloughing ulcer on the faucial tonsil or posterior third of the tongue may be an early cause of complaint in glandular fever.

Vincent's Acute Ulcerative Gingivitis and Stomatitis (Syn: acute ulceromembranous stomatitis).—The *Borrelia vincentii* and the *Fusiformis fusiformis* are always to be found in large numbers in smears from slough over the lesions in these conditions. On this evidence they are likely to be the ætiological agents.

However, experiments in which material from the surface of active lesions has been packed into the interdental spaces of volunteers has failed to produce the disease. The *Borrelia vincentii* is a mobile spirochæte with three to four loose spirals while *Fusiformis fusiformis* is a large rod-shaped organism with pointed ends. They are both anærobic and gram negative. They may be demonstrated in smears of slough from the lesion stained with carbol fuchsin, but are very difficult to culture. Acute ulcerative gingivitis starts on the crests of the interdental papillæ and progresses to form a deep crater covered with a greenish grey slough which is composed largely of necrotic tissue. The organisms are found only in the slough and do not penetrate into the underlying tissue.

In some patients the infection starts around a partly erupted wisdom tooth and in others in the crypts of the tonsils. Tonsillar infection is called Vincent's angina. In severe cases it may spread over the adjacent tissues. The ulcers bleed readily and the patient may complain of spontaneous gingival hæmorrhage. In developed countries the disease is not seen in young children, but is not uncommon in adolescents and young adults. It does not affect the edentulous mouth. There is a characteristic musty fœtor oris.

In acute cases the patient is unwell, has a temperature, complains of a persistent, severe ache in the affected part and salivation. However, subacute infections may also occur with little constitutional disturbance. The incidence increases during the winter months and during war time, so that other intercurrent illnesses, or a mild degree of malnutrition may be important factors in the occurrence of the disease. The Vincent's organisms are sensitive to both penicillin and metronidazole, but treatment with antibiotics alone is insufficient to eradicate the gingival or pericoronal infection. Various

local factors also need to be treated by the patient's dental surgeon. Unless the disease is controlled, severe damage to the attachment of the teeth can result.

The organisms may also infect deep human bites where anærobic conditions exist in the depths of the wound.

Cancrum oris is a severe form of the disease affecting young, poorly nourished children and occurs as a complication of measles and other childhood illnesses. Malnutrition is a predisposing cause and the presence of erupted teeth appears necessary for the onset. Vincent's ulceration starts on the gingivæ and spreads over and into the bone of the jaw. From the gum it spreads on to the inside of the cheek and then through to the skin surface, producing a large area of full thickness tissue loss.

Unless treatment is prompt the child is likely to die. The treatment is systemic penicillin, local wound irrigation and the introduction of a high-protein, vitamin-rich diet with a nasogastric tube. Healing results but with gross scarring which prevents movement of the mandible and, of course, there is often a large hole through the cheek. All scarred tissue should be excised and the defect repaired with a lined, tubed pedicle flap which is inset with the mouth wide open.

Monilial Stomatitis.—Several clinical forms are seen of oral infection with candida albicans.

Acute pseudomembranous candidiasis or thrush occurs classically in debilitated infants. Small, thin, soft, moist, creamy-white plaques form on the mucous membrane, looking like adherent curds of milk. If the white patch is wiped off a bleeding erosion is revealed. Thrush may be seen also in the chronically ill, the elderly, or at any age during treatment with adrenocortical steroids or cytotoxic agents. 1% aqueous gentian violet applied direct to the lesions is the best treatment for the very young and amphotericin B lozenges to dissolve in the mouth four times a day for older patients.

Acute hypertrophic or hyperplastic candidiasis resembles the acute pseudomembranous form except that the tongue is usually involved and a thick, confluent, white mass of mycelia and keratin coats the dorsum.

Acute atrophic candidiasis is most often seen as a complication of treatment with broad spectrum antibiotics. Again it is the tongue which is most affected. The lateral part of the dorsum is smooth and red and the patient complains of a burning sensation.

Chronic atrophic candidiasis or denture sore mouth, produces a red œdematous mucosa over the area covered by an upper denture. Sometimes there is a papillary hyperplasia of the mucosa of the hard palate. Despite the name ' denture sore mouth ', it is usually painless. The patient's denture hygiene is usually poor and needs to be improved. Further, the patients are often wearing the dentures at night, and this they should not do. Amphotericin B lozenges are sucked to get rid of the candida in the palatal epithelium, or Nystatin cream may be applied to the inside of the dentures before they are worn.

Chronic Hypertrophic or Hyperplastic Candidiasis and Speckled Leucoplakia (see candida leucoplakia below).

Angular Cheilosis (Syn: Angular Stomatitis).—There are moist, infected and crusting cracks at the angles of the mouth. The cause is a leak of saliva at the corners of the mouth and the moist skin becomes infected by candida

and staphylococci. It may be seen in children who suck a finger, when it is called *perleche*, or in the middle-aged and elderly (fig. 581). In the older patient the face sags and wrinkles to produce a moist fold. A similar deepening of the crease at the angle of the mouth tends to occur in the edentulous person, particularly where atrophy of the ridges under the dentures permits overclosure. Loss of the canine eminence also permits the angles of the mouth to sag. The local infection can be treated with nystatin cream and fucidate ointment, or alternatively miconazole cream, which is effective against both candida and staphylococci. The denture wearer should go to the dentist for new dentures to correct the overclosure of the jaws. Any atrophic candidiasis under the upper denture should be treated at the same time.

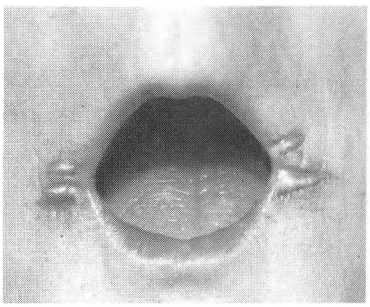

FIG. 581.—Angular stomatitis.
(*Professor Charles Wells, Liverpool.*)

Rhagades occur at the corners of the mouth in congenital syphilis which, when they heal, leave radiating scars and furrows. These usually extend further round the lips than the lesions of perleche for the latter condition rarely causes scarring.

Burns

Burns and scalds from hot food and drink may cause blistering, but rarely severe injury. Children occasionally put the spout of a kettle in the mouth and this causes more severe scalding. Hydrocortisone cream may help but little else is possible for the acute injury. Small electrical connections from a mains electric plug to portable radios and razors etc. may be placed in the mouth by a small child to produce severe electrical burns. The lips particularly are damaged, resulting in complete loss of skin and mucous membrane. Subsequent surgical correction of microstomia may be required, however a dentist is sometimes able to construct an appliance to keep the raw surface stretched until secondary epithelialisation occurs. Chemical burns are also seen occasionally. The commonest type is the aspirin burn. The patient places a tablet of aspirin in the sulcus against a painful tooth, where it produces a white, soggy, sloughing lesion.

The Single Oral Ulcer

The most frequent cause of a single ulcer in the oral cavity is acute or chronic trauma. Toothbrush abrasions produce linear ulcers in the sulcus. The flange of a denture which has been over extended, or which is too deep because the ridge has atrophied, will produce recurrent acute or chronic linear ulcerations also in the sulcus. Thinly epithelialised scars of healed ulcers may be seen. The tongue is an inquisitive organ and once it has discovered a sharp point or edge on a tooth, a cavity in a tooth, or a projection on a dental appliance it will constantly seek it out and explore it until an

ulcer results. Such ulcers may persist for weeks or months, in which case the possibility that a malignant neoplasm is the cause must not be overlooked.

Any obvious source of irritation should be dealt with, but a biopsy is necessary if rapid healing does not follow. In some benign ulcers biopsy provokes healings, perhaps because the surgical lesion is painful and deters the patient from rubbing his tongue.

Tuberculosis, syphilis and various fungi can produce chronic ulcers which require biopsy, or special microbiological examinations to find the cause. Wegner's granuloma, eosinophil granuloma and reticulum cell sarcoma may all produce chronic, slowly progressive ulcers. Because of the effects of secondary infection the diagnosis may not be readily apparent, even in a biopsy.

Salivary Mucus Extravasation and Retention Cysts

Mucus cysts of minor salivary glands produce pinkish, bluish, or yellowish, globular, soft swellings up to 1·5 cm in diameter on the inner aspect of lips or cheeks (fig. 582). A few are retention cysts behind minute calculi. The majority are extravasation cysts. Mucus escapes into the tissues following rupture of the duct. The cyst and the associated minor gland from which the saliva is leaking should be excised together.

A ranula.[1] Ranulas are extravasation cysts arising from a damaged sublingual gland. If the saliva distends the floor of the mouth a translucent bluish swelling is formed with prominent blood vessels running over its surface (fig. 583). If mucus is escaping from a posterior sublingual gland it

FIG. 582.—Retention cyst of a buccal mucus gland.

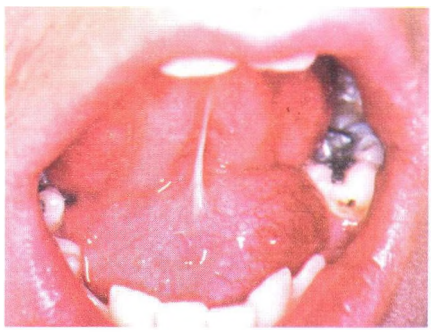

FIG. 583.—A simple ranula. (*R. N. Baird, F.R.C.S., Bristol.*)

may flow over the posterior margin of the mylohyoid and down into the neck to form a *plunging ranula*.

The wall of a ranula is composed of a delicate capsule of fibrous tissue and is lined by a layer of macrophages. Many ranulas rupture and discharge.

[1] So named because of the likeness of the swelling to the belly of a little frog (Latin, *ranula*, diminutive of *rana* = a frog).

Friedrich Rudolf Georg Wegner, 1843– . *German Pathologist.*

Some fail to refill, but many will recur. Gentle dissection to identify the sublingual gland from which the swelling is arising permits the offending gland mass to be removed. It is unnecessary to explore the neck for a plunging ranula. All that is required is to remove the entire mass of sublingual glands on the same side and to pass a drain from the floor of the mouth down into the neck cavity.

Lingual and Sublingual Dermoids

Dermoid cysts produce an opaque swelling. They may be lined by stratified squamous epithelium, with, or without, dermal appendages. Such cysts are filled with a doughy mass of keratin. Others are lined by ciliated, mucus secreting epithelium and filled with mucus. These are fluctuant. Dermoid cysts may be found in the midline of the tongue, or in the floor of the mouth, either in the midline or laterally in the sublingual region.

Median sublingual dermoids are seen more often than the other types (fig. 584). They are probably derived from epithelial rests left as the two contributions to the tongue from the back of the mandibular arch merge together, they enlarge backwards between the genial muscles, into the tongue and down towards the hyoid. They lie above the mylohyoid muscle, but bulge downwards towards the submental region as the mouth is closed and the oral swelling is compressed by the tongue. Their removal is best effected via an incision which extends vertically in the midline from the tip of the tongue to the attachment of the lingual frenum to the mandible.

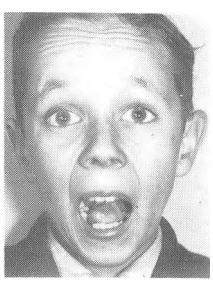

FIG. 584.—Large median sublingual dermoid.

Lateral sublingual dermoid cysts develop below the submandibular duct and lingual nerve, and anterior to the stylohyoid ligament; that is, from the region of the first branchial pouch. They displace the submandibular salivary gland backwards as they enlarge. Small ones are removed through the floor of the mouth and large ones through a submandibular incision.

TUMOURS OF THE CHEEK AND THE FLOOR OF THE MOUTH

A miscellaneous variety of abnormal lumps may be found within the substance of the cheek or floor of mouth or arising from their surface. Some are benign neoplasms, some are hamartomas and others are inflammatory hyperplasias. A few of the more common examples are:

Fibro-epithelial Polyp.—If the cheek is repeatedly traumatised at one place a thickened, submucous scar is formed which may be pulled out on to a stalk by the suction of deglutition (fig. 585). A lump of cheek may be sucked by a similar mechanism into a gap where a tooth is missing. The result is a soft, rounded, pedunculated, fibrous swelling.

Fibro-epithelial polyps are excised through the base of the pedicle.

Denture-induced Granuloma.—Fusiform, fibrous masses arising from a linear pedicle are formed in edentulous individuals in the buccal or lingual sulci; sometimes singly and sometimes several together parallel to one another. These granulomatous masses arise as a result of chronic irritation and ulceration from the flange of an ill-fitting denture.

Papilloma.—Papillomas may occur on the cheek, alveolar mucosa, palate or floor of mouth. They may be solitary or multiple. Some are a mass of tiny, close-set, finger-like processes, others have a knobbly surface like a cauliflower. They are covered with keratinised epithelium which is white in the moist environment of the mouth. Some patients have multiple papillomas in their mouth and may have viral warts elsewhere. In children, such other warts may be on the knuckles. In adults the lesions may be genital warts.

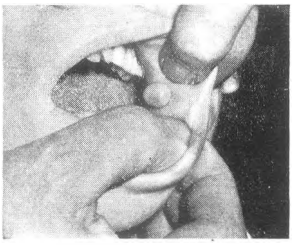

FIG. 585.—Fibro-epithelial polypus of the mucosa of the cheek.

Hæmangiomas.—May occur in the cheek and floor of the mouth. Small cavernous hæmangiomas up to 0·5 cm in diameter may be found on the inside of the cheek opposite the occlusal plane and arise as a result of a bite which damages a submucosal vessel. More massive hæmangiomas surround the buccal pad of fat in the buccal space.

Lipomas.—Produce yellowish submucosal swellings in the cheek and floor of mouth.

Neurofibromas.—Most often produce a diffuse thickening of the gums spreading out into cheek or floor of mouth. Occasionally a single, fusiform, soft, fibrous mass is found along the line of a nerve, such as the lingual nerve. Others may produce pedunculated swellings.

Lymphangiomas.—Tend to occur posteriorly in the cheek over the coronoid process and look like brownish frog spawn.

Salivary Tumours.—Pleomorphic adenomas, muco-epidermoid carcinomas, adenoidcystic carcinomas, and more rarely other salivary neoplasms, may arise in buccal, retromolar, labial and sublingual salivary glands (see Chap. 33).

Facial Lymph Node.—Some individuals have a lymph node which is an outlying member of the submandibular nodes; it is found lying over the body of the mandible along the path of the facial artery as it goes towards the angle of the mouth. When enlarged it may produce a palpable lump in the cheek or lower buccal sulcus (p. 551).

Carcinoma of Cheek

While adenocarcinomas may arise in a minor salivary gland, these are uncommon. The majority of carcinomas of the mucosal aspect of the cheek are squamous cell in nature. In members of western races they are more common among those who smoke heavily and drink spirits. Some arise in candida-infested speckled leucoplakia. Carcinoma of the cheek is specially common where betel nut is chewed and the pan stored in the cheek pouch. It is also a complication of submucous fibrosis. An exuberant papilliferous tumour may be produced by a verrucous carcinoma.

Treatment of Carcinoma of Cheek.—The best results from the point of view of function and appearance are achieved by radiotherapy. Interstitial radiation may be delivered by ^{137}Caesium needles or by ^{192}Iridium wire. The latter can be after-loaded into nylon tubes which may be placed accurately without risk to the operator and the position checked by radiography with non-active wires in position. Alternatively external beam treatment with megavoltage machines may be used.

Surgery is required for residual or recurrent tumour or where radiotherapy is not available. Post-irradiation surgery may also be advisable for large tumours involving the adjacent jaw. A small or superficial tumour can be excised and the resultant wound grafted with split skin applied on a polyurethane foam pad which is sutured in place for 10 days. Larger tumours require full thickness resection of the cheek and repair. A deltopectoral flap, previously delayed and lined with split skin, may be used, or a combination of a forehead flap on the inside and a deltopectoral flap on the outside of the cheek. Adequate size of flaps should be used to recreate buccal sulci so as to permit movement of the mandible.

Neck nodes are treated by pre-operative radiotherapy to 4,000 rad then block dissection of the neck 6–8 weeks later.

THE TONGUE

Developmental Anomalies

Tongue-tie.—The lingual frenum is short, often thicker than normal and fibrous. It holds the tip of the tongue close to incisal edges of the lower central incisors (fig. 586). Attempts to raise or protrude the tongue result in eversion of the lateral margin and a heaping up of the mid-portion of the dorsum. More often than not the deformity causes no disability and no action is required. However, it may result in lisping. The patient can also have difficulty in cleaning the backs of the lower front teeth. Where division is necessary a small quantity of local anæsthetic solution is injected with a fine needle. A traction suture is passed through the tip of the tongue and the frenum divided with scissors just below the undersurface of the tongue until adequate mobility of the tip is achieved. The wound takes on a linear shape in a vertical direction if the tip of the tongue is raised and may be closed with fine, plain catgut.

Congenital Fissured Tongue.—Fissures of varying depth run laterally from a median groove, but the surface between is covered with normal papillæ (fig. 587).

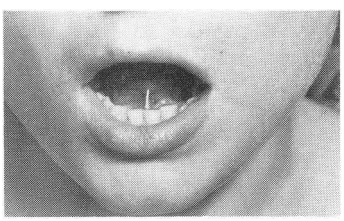

FIG. 586.—Short frænum linguæ.

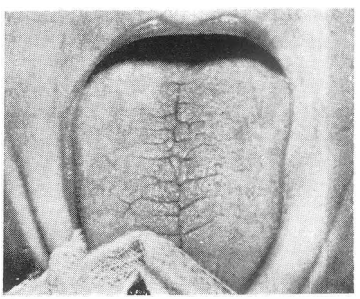

FIG. 587.—Congenital fissured tongue. Note the transverse direction of the fissures.

Sometimes the fissures become infected with candida albicans (the syphilitic tongue tends to be bald and with longitudinal fissures).

Lingual Thyroid.—Produces a reddish lobulated mass behind the foramen cæcum (p. 642).

Median Rhomboid Glossitis

A smooth, lobulated, oval, or triangular patch immediately anterior to the foramen cæcum. The mucosa may be of a rather deeper colour than the rest of the tongue and firm. Chronic infection of the fissures between the lobules with candida albicans is not uncommon. The condition may be mistaken for carcinoma which is rare in the midline of the tongue.

Geographic Tongue (Glossitis migrans)

Small red patches develop with a white, furred margin. The patches spread and recede in an irregular fashion and fresh patches appear (figs. 588–591). Keratinised epithelium and inflammatory cells accumulate on the filiform papillæ to form the white margin and are then shed. The condition is quite benign but the real ætiology is unknown. It is more common in patients with congenital heart defects. It is also seen in those with acute gastrointestinal problems.

Macroglossia

A variety of developmental conditions and hamartomas can result in a persistent, painless, enlargement of the tongue. The tongue may be of normal structure, but large in proportion to the oral cavity. The patient can usually retract it into the mouth, but is able to protrude it to a surprising degree. Neurofibromatosis may be a cause of macroglossia, but often one side only is affected. Cavernous hæmangiomas

also tend to produce unilateral enlargement while lymphangiomas tend to occur bilaterally and produce a thick and cumbersome tongue which prevents closure of lips and jaws. All developmentally enlarged tongues exert continuous pressure on

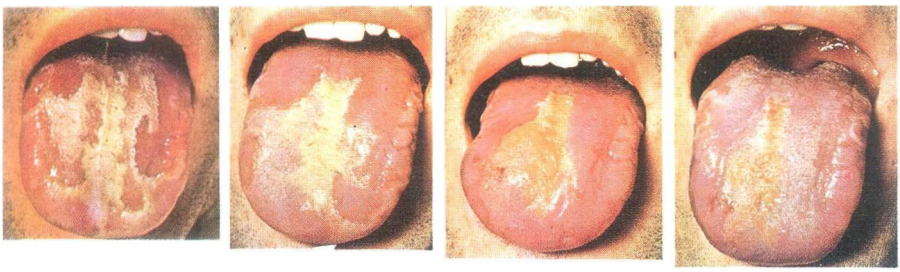

FIG. 588.—March 5th. FIG. 589.—March 7th. FIG. 590.—March 9th. FIG. 591.—March 12th, patient making satisfactory recovery.

FIGS. 588–591.—Glossitis migrans—this patient had a partial gastrectomy on March 1st, followed by paralytic ileus and pulmonary collapse which was treated by tetracycline.

the teeth and alveolar process resulting in spacing of the teeth and proclination of the incisors.

Elongation of the tongue may be corrected by wedge resection from the midline of the anterior third or more. Vertical thickening may be reduced by wedge-shaped slices removed from the lateral margins. Care should be taken not to damage the nerve supply or the major branches of the lingual artery. While the tongue is a very vascular organ, damage to both lingual arteries can lead to problems with wound healing.

Acquired Causes of Enlarged Tongue

Acromegaly.—The tongue enlarges in a similar fashion to the lips, nose, liver etc.

Amyloidosis.—Deposits of amyloid in the tongue in primary amyloidosis may cause macroglossia.

Cretinism.—In long-standing hypothyroidism the tongue is enlarged as a result of an accumulation of a mucoproteinous material in the tissues.

Laceration of the Tongue

Lacerations most commonly arise as a result of the patient biting his tongue. Epileptics may do this during the clonic convulsive phase of a grand mal attack unless a suitable pad, gag or rubber ring is placed between the posterior teeth. Fractured jaws sustained during road traffic accidents may also be associated with a laceration of the tongue if this organ lies between the teeth during the accident. A blow on the face or a fall while the patient is smoking a pipe can result in the stem breaking and lacerating the tongue. In an unconscious patient the brisk hæmorrhage which follows such an injury can endanger the airway.

Severe hæmorrhage as a result of damage to the lingual artery can be controlled by hooking the tongue forward with a finger and compressing the tongue against the mandible and between the fingers and a thumb in the submental region. Fortunately the tongue has such a good blood supply that quite extensive lacerations may be sutured with every hope of sound healing.

A closed injury of the tongue or a fracture of the mandible in an elderly patient can result in a hæmatoma which spreads in the tongue and floor of mouth, eventually producing respiratory embarrassment so that a tracheostomy may be required.

Inflammation of the Tongue

Pyogenic infections of the tongue are extremely rare. There may be some œdema of the tongue associated with a cellulitis of the sublingual space and in *Ludwig's angina* the infection spreads backwards between the hyoglossus and genial muscles, but not often into the tongue proper. The tongue of course is raised and protruded from the mouth in these conditions because of the swelling of the floor of the mouth.

Great swelling of the tongue can follow a *wasp sting*. Classically this occurs when a holiday-maker drinks from a beer bottle which has been left open. *Angioneurotic œdema*, or hereditary angio-œdema may affect the tongue to produce life-threatening swelling. The insertion of a nasopharyngeal airway is the quickest way to establish an airway behind a swollen tongue, but in some circumstances a tracheostomy may be required.

Tuberculosis can affect the tongue in two ways: to produce shallow, oval, indolent, painful ulcers with overhung margins, or to produce a circumscribed, interstitial tuberculoma. Both types of lesion only occur in advanced, untreated tuberculosis and are consequently now rarely seen where effective drug treatment is prescribed promptly.

Syphilis may produce lesions of the tongue at each stage of the disease.

Primary syphilis of the tongue. An extragenital chancre can occur on the tongue. The submaxillary and submental lymph nodes become greatly enlarged, as in the case of a similar lesion on the lip. There is a comparative lack of pain compared with other ulcers of the tongue.

Secondary Syphilis

1. Multiple shallow ('snail track') ulcers may be present on the sides and undersurface of the tongue.
2. Mucous patches occur on the tongue as well as on the fauces.
3. Hutchinson's wart, really a condyloma, is always found in the midline (fig. 592).

FIG. 592.—Hutchinson's wart. Wassermann reaction strongly positive.

Tertiary Syphilis

1. A gumma of the tongue is now uncommon in countries where primary syphilis is adequately treated. The lesion starts as a slowly enlarging, non-tender, midline swelling. Following necrosis of the overlying tissues the wash-leather slough separates to leave a deep, crater-like ulcer. Two features distinguish the gummatous ulcer from a carcinoma: firstly, carcinomas very rarely arise in the midline of the tongue and secondly there is no tethering of the tongue even with a large gumma.
2. Multiple small gummas heal with scarring to produce fissures which divide the central part of the dorsum into lobules.
3. Chronic superficial glossitis causes a loss of papillæ on a tongue which is also fissured due to the underlying interstitial glossitis. The result is a bald, lobulated tongue. The epithelium becomes hyperkeratotic and squamous cell carcinoma tends to arise in the resultant leucoplakia. There is a reduced blood supply to the syphilitic tongue due to endarteritis, and radiotherapy may be contraindicated as a treatment of carcinoma arising in a tongue affected by chronic superficial glossitis. The increased ischæmia which follows the radiotherapy may lead to necrosis.

Wilhelm von Ludwig, 1790–1865. Professor of Surgery and Midwifery, Tübingen, Germany.
Sir Jonathan Hutchinson, 1828–1913. Surgeon, The London Hospital.

Hyperkeratosis and Leucoplakia

In the mouth, a hyperkeratotic epithelium is kept moist by the saliva and appears white. The term ' keratosis ' is usually used where the ætiological factor is readily recognised and where withdrawal of the irritant tends, in time, to regression of the white patch. For example, a frictional keratosis, or a smoker's keratosis. Leucoplakia is also used as a descriptive term for a white, hyperkeratotic patch in the mouth, or on certain other mucous membranes. It is used in different ways by different clinicians, but in general is used where the ætiological agent is not recognised or known or where the lesion has become established to a point where it no longer regresses when the causative irritant is withdrawn. Some pathologists require that the epithelium should exhibit dyskeratosis on histological examination of a biopsy.

Tradition has it that **S**moking, **S**yphilis, **S**epsis, the **S**harp edge of a tooth (chronic frictional irritation), **S**pirits and **S**pices are the causes of leucoplakia (the Six **S**s). There is more than a grain of truth in this tradition and to this list should be added *candidiasis*.

The majority of ulcers caused by broken, carious teeth or dentures are innocent, but from time to time hyperkeratosis and a carcinoma is found just where the sharp edge rubs against the tongue. Syphilis, as was described above, causes chronic superficial glossitis in which leucoplakia develops, again with malignant change a likelihood.

Smoking produces a hyperkeratosis of the hard and soft palate in which the mucous gland ducts, plugged with keratin, stand up as prominent points. Hyperkeratosis also develops on the cheek behind the commissure of the lips, on the floor of the mouth, where chemical-laden saliva flows, and on the lips where cigarette or cigar is held. A white patch may also develop on the tongue opposite the end of the cigar or pipe stem. Leucoplakia and squamous cell carcinoma are most likely to develop if spirit drinking is added to smoking, but mainly of tongue or floor of mouth. Carcinoma rarely arises in the palatal smokers' keratosis.

The classical ' spice ' is betel nut. Areca (betel) nut mixed with tobacco, spices and slaked lime is wrapped in a betel leaf (from a different plant to the nut). Leucoplakia develops adjacent to where the ' pan ' is normally held in the mouth, usually on the cheek and the side of the tongue.

The relationship of candida albicans to leucoplakia is incompletely understood. Leucoplakia appears to arise following long-standing candida infection (*chronic hypertrophic candidiasis*). In other instances the candida seems to be a secondary invader of the hypertrophic epithelium. The clinical appearance may be speckled white on a pink background rather than a uniform white.

Speckled leucoplakia is particularly prone to develop a carcinoma. Initially in the development of leucoplakia there is a hyperkeratosis with a small increase in the prominence of the rete pegs. Lymphocytes and plasma cells accumulate in the dermal papillæ. At this stage there is a milky blush on the mucosal surface. Later there is a marked increase in the thickness

of the epithelium, the rete pegs elongate (acanthosis), and the round cell infiltration becomes quite dense (fig. 593). Mitosis increase in number among the basal cells. Clinically there is now a distinct, paint-like, smooth, white patch which is dry and rough to the touch. Finally mitoses appear in the cells above the basal layer and some cells become keratinised before reaching the surface (dyskeratosis). The leucoplakia is then only a step away from carcinoma *in situ* and the development of a frank squamous cell or verrucous carcinoma. At the dyskeratotic stage patchy loss of the keratinised layer tends to occur to give a patch-work appearance of red and white (fig. 594). A warty appearance of the surface heralds the development of a carcinoma.

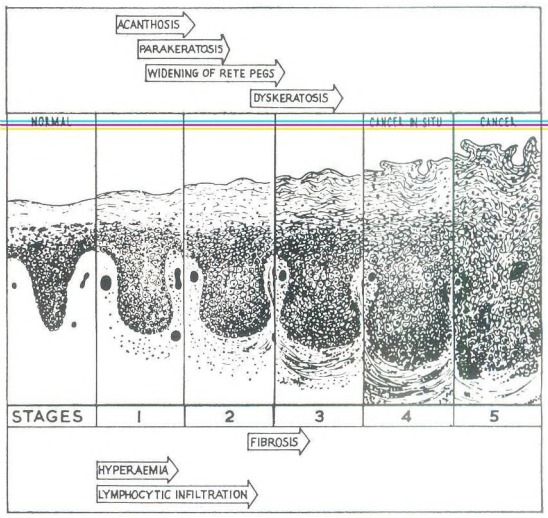

FIG. 593.—Diagram of the histological stages of leucoplakia leading to carcinoma.

Opinions vary as to the risk of carcinomatous change and what is advised depends partly on the extent of the lesion and partly on the judgement of an experienced clinician. The patient should be councilled to give up identified irritants, then early lesions may disappear after some months but will return promptly if the old habits are resumed. Small white patches may be excised and large ones biopsied. A prolonged treatment with local nystatin or amphotericin B should be tried if candida are present. Hyperkeratotic lesions without pre-malignant change should be watched and monitored by exfoliative cytological examinations from time to time.

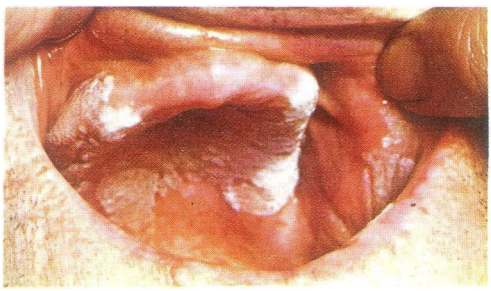

FIG. 594.—Leucoplakia on the palate and cheeks of an edentulous patient. This patient had had three carcinomas ' in situ ' removed from his tongue.

Dyskeratotic lesions and carcinoma *in situ* should be excised and the area grafted.

Where there is a likelihood of malignant change, this may not be confined to the obvious white patches however. In some mouths the whole of the mucosal surface is unstable and at risk. Atrophic red areas are as likely to undergo malignant change as the white patches.

Radiotherapy will get rid of the leucoplakia, though the hyperkeratosis tends to reappear after months or years. Unfortunately there is evidence that radiotherapy increases the likelihood of malignant change.

Other Hyperkeratotic Lesions.—Other conditions which produce white patches are: *lichen planus,* which characteristically produces a striated and lace-like appearance, but may form a plaque. White patches are sometimes seen in *discoid lupus erythematosis.* The *white sponge nævus* has a folded appearance and covers both cheeks and the floor of the mouth. It is inherited in some cases.

The symmetrical white patch of the underside of the tongue and sublingual region is also considered to be a developmental lesion, but can give rise to a squamous cell carcinoma.

Leucokeratosis mucosæ oris is another congenital abnormality in which all the oral mucous membranes have a filmy white appearance.

Submucous Fibrosis.—Submucous fibrosis produces a mottled or marbled pallor of the mucosa of cheeks, palate, tongue and gingivæ, but in this case the pallor is due to collagen deposited in the submucosa. The lips and cheeks become stiff and lose their elasticity. Bands of fibrous tissues develop beneath the mucosa and limit jaw movement so that opening of the mouth becomes greatly restricted. It is thought that the condition results from a hypersensitivity to chilli. Squamous cell carcinoma is a not infrequent complication.

Hairy Tongue.—The keratinised layer fails to desquamate normally from the filiform papillæ and becomes greatly elongated to resemble a coating of hair. The 'hair' may take on various colours due to the presence of chromogenic organisms to produce a black or brown hairy tongue. Sometimes, following the use of antibiotics, the normal filiform papillæ becomes slightly lengthened by keratin and coloured black by antibiotic-resistant organisms such as fungi. This form of black hairy tongue clears up soon after the use of the antibiotic ceases. True hairy tongue is more intractable, but can be kept under control by mechanical scraping. Both conditions must not be confused with the normal furring of the tongue in an ill patient. Dehydration and lack of mechanical cleaning in a patient who is not eating normally combine to let keratinised cells accumulate on the filiform papillæ. Bacteria and food debris accumulate among the papillæ. Heavy smoking increases the keratinisation of the papillæ and stains the fur black and brown to make it more obvious.

Ulcers of the Tongue

It is advisable at this stage to review the more common types of ulcers of the tongue, as their differential diagnosis is so important.

Dental Ulcers.—Mostly occur at the side of the tongue and an obvious cause will be seen. This may be a carious and broken tooth, or a broken denture. Usually the ulcer lies in relation to the sharp object at rest so the relationship is not apparent when the tongue is protruded for inspection. More extensive oval ulcers may occur to one side of the upper surface in relation to a sharp palatal cusp of an upper tooth, or even close to the midline and related to a rough or fractured surface of an upper denture. The dorsum of tongue is reasonably tough but once a rough surface is discovered the patient may explore it and habitually rub the tongue against it.

If the ulcer does not heal promptly when the apparent cause is removed, or if there are features suggestive of carcinoma, it *must* be biopsied. Often a biopsy will provoke an indolent, innocent ulcer to heal.

Post-pertussis ulcers occur on the upper part of the lingual frænum and the undersurface of the tip in patients who protrude the tongue over the lower incisors during bouts of coughing; typically with whooping cough.

Aphthous ulcers (p. 561).

Ulcers in lichen planus, pemphigus, erythema multiforme etc. (p. 561).

Syphilitic ulcers (p. 571) typically in the midline.

Tuberculous ulcers (p. 571) on the dorsum, towards the midline or near the tip and irregular with undermined margins. Pale, indolent, granulations in the floor, sometimes with a thin slough.

Malignant ulcer (p. 576) usually a squamous cell carcinoma, but may be a salivary adenocarcinoma or a lympho-epithelioma when the ulcer is on the posterior third.

Neurological Lesions of the Tongue

Common sites of surgical damage to the *lingual nerve* are at the upper pole of the submandibular salivary gland, beneath the mucoperiosteum lingual to the third molar and where the nerve crosses beneath Wharton's duct (p. 584) in the floor of the mouth. Return of sensation may be imperfect (paræsthesia), or accompanied by causalgic pain. Another cause of lingual neuralgia is as an aftermath of herpes zoster. Pain and anæsthesia, often with referred pain to the ear, result from invasion by a malignant neoplasm.

Hemiatrophy follows damage to the *hypoglossal nerve*, either during the extracapsular removal of the submandibular gland, or as the carotids are approached, high in the neck during a block dissection. In the former location the nerve is accompanied by easily damaged *veni comitantes* and the nerve can be included in hastily applied artery forceps. Paralysis of the musculature on the same side and deviation towards the affected side on protrusion are seen.

Glossodynia occurs typically in middle-aged and elderly women. Often no cause can be found. Sometimes there is an atrophic candidiasis. Sjögren's syndrome (p. 586) and diabetes are two possible underlying conditions which should be considered if this is so. Some middle-aged patients complain of an unpleasant change in taste. The abnormal sensation can be confirmed by appropriate taste tests. The cause is unknown.

BENIGN NEOPLASMS

Benign neoplasms are uncommon, compared with squamous cell carcinoma.

Papilloma (p. 568) must be distinguished from the rare Hutchinson's wart (p. 571) which resembles a pinkish, soft, cauliflower-shaped papilloma. Papillomas are readily excised, or if multiple can be treated with a small cryoprobe.

Fibro-epithelial polyps (p. 567), usually small and opposite a source of mild, chronic irritation such as a gap between the lower incisor teeth.

Pregnancy tumour (p. 543), usually found on the gum, but can occur on the tongue.

Hæmangioma and lymphangioma (p. 568).

Plexiform neuroma and neurofibroma (p. 568).

Lipoma is another uncommon entity recognised by its softness and yellow colour. Rarely gets large before advice is sought, but not always (fig. 595).

Osteoma of the tongue is a clinical curiosity. A hard swelling found beneath the foramen cæcum or in the posterior third of tongue.

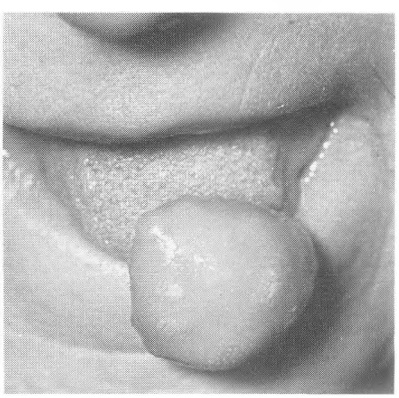

FIG. 595.—A large pedunculated lipoma of the tongue which the patient seemed to be able to accommodate within the mouth.

Sublingual varicosities should not be confused with a cavernous hæmangioma. The sublingual veins are greatly dilated and tortuous in this condition. Initially small veins enlarge to produce a caviare

appearance; then the larger ones. Not uncommon in the elderly. Reputed to indicate chronic heart failure, but this not so.

Granular cell myoblastoma produces a firm mass in a mobile tongue. The overlying mucous membrane is smooth and often hyperkeratotic. A biopsy of the epithelium alone reveals pseudo-epitheliomatous hyperplasia which may be mistaken for carcinoma. A deeper biopsy reveals the eosinophilic granular cells. A benign, but not encapsulated lesion which is cured by conservative local excision.

Juvenile fibroma produces a softer, central, slowly enlarging lump. At operation this benign tumour has no readily discerned margin from the adjacent muscle, consequently is not easy to remove, particularly as it tends to occur in the small tongues of young children.

CARCINOMA OF THE TONGUE

Since 1910 there has been a decreasing incidence of carcinoma of the tongue in *males*. Possible factors bringing this about are: more efficient treatment of syphilis so that while this disease is still common, the late effects are rare; the passing of the clay pipe, improved standards of oral hygiene and oral health, and possibly a decrease in the consumption of spirits. While the reduction of the incidence in males is gratifying, there has been an increase in females (Russell), possibly related to an increase in female smokers, so that the sex incidence is now equal.

Clinical Features

As in carcinoma in other situations, early stages of the disease are virtually symptomless. Observant and fastidious patients may seek advice for a comparatively small lesion. Others may mention it in passing to doctor or dentist. Because the mouth is so easily examined, doctors should inspect the

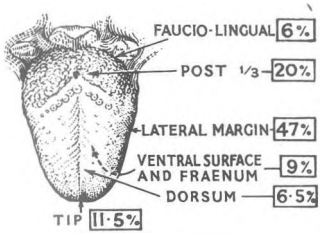

FIG. 596.—Relative frequency of the seat of carcinoma of the tongue. (*Birmingham United Hospitals' Statistics.*)

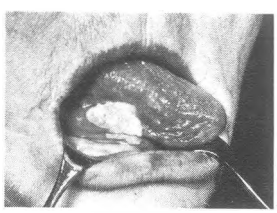

FIG. 597.—Carcinomatous ulcer of the tongue. (*Denis Bodenham, F.R.C.S., Bristol.*)

oral cavity and tongue whenever the fauces need to be examined. Dentists likewise should be on the look out for suspicious lesions when they examine a patient.

Carcinoma of Tongue may Present as:

1. An oval, raised, papillated plaque with white keratin flecks on the surface.
2. An ulcer with sloughing from an indurated base and with everted margins (fig. 597).
3. A deep, often infected, fissure with surrounding induration.

Marion H. Russell, 1907–1966. Medical Statistician, Christie Hospital, Manchester, England.

4. A lobulated, indurated mass, possibly with seemingly normal mucosa over it, sometimes with yellow patches of submucosal necrosis which appear like pointing abscesses.

Many patients seem to disregard the lesion in its early stages or are too frightened to seek help until a large and offensive tumour is present. These more advanced cases present with:

1. *Pain in the tongue*, which is initially due to infection and ulceration but later is due to involvement of the lingual nerve. Once the lingual nerve is involved there may be referred *pain in the ear*. Pain on swallowing or pain in the back of the tongue may mean a carcinoma of the posterior third. In such cases careful inspection of tongue and vallecula in the conscious patient with a laryngeal mirror, or direct examination with a laryngoscope with the patient under anæsthesia, is essential. The posterior third can be palpated, but induration due to neoplasm is not easily appreciated because of the lumpiness of the lingual tonsils.

2. *Salivation.*—Pain promotes salivation and a stiff, lumpy, partially fixed tongue makes swallowing difficult. The elderly man sitting in outpatients and spitting into a handkerchief may well have a carcinoma of tongue.

3. *Ankyloglossia.*—Inability to protrude the tongue or deviation to one side means it is fixed by extensive infiltration of the floor of the mouth.

4. *Dysphagia.*—Difficulty in swallowing may occur with any advanced lingual carcinoma, but is more pronounced when the growth is in the posterior third.

5. *Inability to articulate clearly.*

6. *Foetor.*—Once necrosis occurs the lesion becomes grossly infected and highly offensive.

7. *A lump in the neck* due to secondary deposits in the draining lymph nodes.

8. *Alteration in the voice* may be a first indication of a carcinoma of the posterior third. In this inaccessible situation a tumour may escape notice. Such tumours are more often anaplastic than those of the anterior third and a large tumour with nodes in the neck may be present by the time the diagnosis is made.

Spread of the Disease

Local Spread.—Carcinoma of the anterior two-thirds usually starts on the lateral margin and frequently reaches the floor of the mouth before it extends across the midline. At the junction of anterior two-thirds and posterior third it may invade the mandible. Carcinoma of the posterior third spreads laterally into the tonsil, side of the pharnyx and cervical spine and up into the soft palate, posteriorly into the epiglottis and downwards towards the larynx, and across to the other side of the tongue.

Lymphatic Spread.—Metastasis to the submandibular nodes, particularly if the floor of the mouth is involved, means that tumour is already present close to the periosteum of the mandible and resection of the relevant segment of the mandible may be necessary to ensure surgical clearance.

Once the anterior part of the floor of the mouth is involved bilateral spread to neck nodes is common.

Blood Stream Spread.—Death tends to occur as a result of uncontrollable primary tumour or lymphatic spread so that distant metastasis rarely manifests itself. Metastases by the blood stream is more likely from posterior third than anterior two-thirds tumours.

Terminal Events

Death from an uncontrolled primary tumour occurs as a result of:

(i) inhalation bronchopneumonia as a result of inhalation of infected material from necrotic neoplasm.

(ii) Hæmorrhage from erosion of the lingual artery, or in the case of a carcinoma of the posterior third, erosion of the internal carotid.

(iii) Combined cancerous cachexia and starvation due to the pain and difficulty of attempting to swallow where a large tumour replaces the tongue, or where there is compression of pharynx or œsophagus by metastatic deposits in lymph nodes.

(iv) Asphyxia, which is most often due to pressure in the air passages from enlarged and fixed carcinomatous lymph nodes. Sometimes obstruction is due to œdema of the glottis.

Treatment

Surgery or radiotherapy or a combination of the two. The most appropriate use of chemotherapy in relation to the main methods of treatment is not yet settled.

Establishment of the Diagnosis.—An adequate biopsy must precede treatment. But there is no place for the so-called excision biopsy. Only rarely will such an excision be generous enough to avoid incomplete removal and local recurrence. Indeed should the lesion prove not to be malignant, unnecessary mutilation will have been inflicted by such an excision.

Preliminary Preparation.—Teeth are scaled and polished and oral hygiene established. Where the surface of the lesion is necrotic or infected, frequent irrigation with eusol is required. Any teeth which will be in a field of substantial irradiation should be extracted.

Tests for syphilis should be carried out and treatment initiated if necessary. In some cases a diagnosis of syphilis will mean that radiotherapy is not advised.

Definitive Treatment

1. *Carcinoma in situ* may be excised with a 1 cm margin and to a depth of about 0·5 cm. Excision wounds on the lateral border may be closed by undermining and advancing mucosa from the floor of the mouth. Larger defects are grafted with split skin.

2. *Lesions less than 2 cm* are treated primarily with radiotherapy, because if treatment is successful there is virtually no functional disability. For lesions of the anterior two-thirds of the tongue of this size, and where there are no palpable lymph nodes, interstitial irradiation with ^{137}Cæsium needles or ^{192}Iridium wire is used. A close watch is kept upon the patient, with follow-up appointments at monthly inter-

vals. If regression is incomplete, or recurrence occurs, or neck nodes become suspiciously palpable, surgery is added.

3. *A small carcinoma confined to the lateral border* may be removed by local excision with a generous 1·5 cm of margin beyond visible and palpable disease. The configuration of the resection depends upon the site of the tumour, but approximates to a hemiglossectomy. The success of the operation depends upon the care with which the excision is carried laterally below the neoplasm for it is here that the operator may fail to maintain an adequate margin.

If there are palpable nodes a block dissection of neck is required. Advantage should be taken to tie the lingual artery before the primary lesion is resected as bleeding will be reduced and surgical precision increased. An incontinuity resection facilitates an adequate margin on the deep aspect of the primary tumour.

If a block dissection becomes necessary resection of the primary site should be considered even if it is apparently controlled by previous radiotherapy. Pre-operative irradiation of the neck to between 4,000 and 4,500 rad may be advisable.

4. *Primary lesions larger than 2 cm* are usually irradiated by external beam irradiation. The submandibular nodes may be included in the field of irradiation even if no nodes are palpable. Great care is required with follow up to avoid extensive spread occurring should the treatment fail to achieve healing. Large tumours require a submandibular dissection to enable adequate excision. Because this makes a subsequent block dissection difficult a prophylactic neck dissection should be considered if local control is not achieved, even if no nodes are palpable.

5. *If there is a large tumour with palpable nodes* both primary and the neck are irradiated to 4,500 rad then, 6–8 weeks later, an in-continuity resection is carried out. (Surgery should be undertaken within three months of the irradiation but after the reaction has subsided.) Where the tumour extends on to the floor of the mouth, or up to the anterior pillar of the fauces, the mandible should be included in the specimen.

6. *Tumours which involve the tip of the tongue* may metastasise to the submental nodes. Tumours which involve the anterior part of the floor of the mouth are likely to metastasise to both sides of the neck. Prophylactic irradiation of the second side should be considered, even if nodes are not palpable.

7. *Bilateral neck nodes* may be dealt with by various strategies without sacrifice of both internal jugulars. Resection of both internal jugulars is to be avoided because of the high mortality due to post-operative cerebral venous engorgement and œdema of the head and face should the patient survive.

(i) Bilateral block dissections may be carried out, but the internal jugular left on the least involved side. The operation should be completed first on the side in which the vein is to be retained.

(ii) Alternatively low-dose pre-operative irradiation should be given to the worst side followed by a block dissection and a therapeutic dose given to the other side. Contrary to previous opinion, squamous cell carcinoma metastases in lymph nodes will respond to megavoltage irradiation. The problem is that recurrence of tumour after irradiation of nodes is often not amenable to salvage surgery.

(iii) A full block on one side and a suprahyoid block on the other is another possible form of treatment.

Pre-operative irradiation of the neck reduces the incidence of diffuse, local recurrence of tumour which is difficult to differentiate from post-operative induration.

8. *Posterior extension of the carcinoma* requires division of the lower lip and section of the mandible for access. The incision should be vertical through the lip, curved around the chin pad, and then broken into a Z until the hyoid crease is reached. Posterior third primary tumours are treated almost entirely by external beam irradiation as they are rarely amenable to surgery.

Where the anterior part of the mandible is sacrificed with a carcinoma involving the floor of the mouth, a Bowerman-Conroy implant is inserted and the hyoid and tongue suspended from it (Wilson).

Repair.—The tip of the tongue may be repaired by suture. Small lateral defects can be closed by advancing floor of mouth mucosa (fig. 598).

There are two ways in which mobile raw surfaces in the mouth, like those resulting from section of the tongue, can be covered with a Thiersch graft (p. 132); by quilting

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John Ernest Bowerman, Contemporary. Oral Surgeon, Westminster Hospital and Queen Mary's Hospital, Roehampton, London.
B. Conroy, Contemporary. Senior Chief Maxillo-Facial Technician, Queen Mary's Hospital, Roehampton, London.

on the graft (McGregor) and by use of a foam plastic pad (Seward). In McGregor's method small, plain, interrupted, catgut sutures are placed at the corners of squares across the graft after suture of the periphery. Tiny stab incisions are made in the centres of the square to let out any exudate or hæmatoma. Alternatively (Seward), a pad of 1 cm thick polyurethane foam is cut slightly larger than the defect, and the Thiersch graft is draped over it, wrapped round, and sutured to the outer surface. A

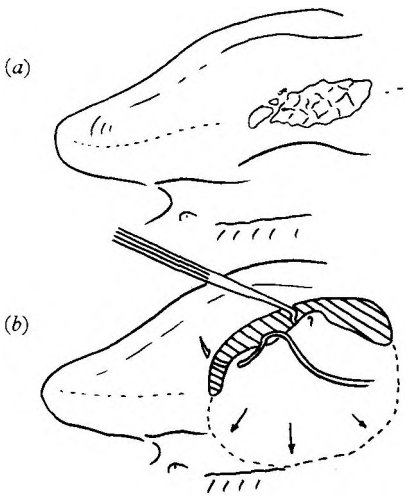


FIG. 598.—(*a*) and (*b*) An intra-epithelial carcinoma (carcinoma *in situ*) on the lateral border of the tongue may be excised and the adjacent mucosa of the floor of the mouth undermined and advanced to cover the defect. The undermining must not involve the sublingual plica.

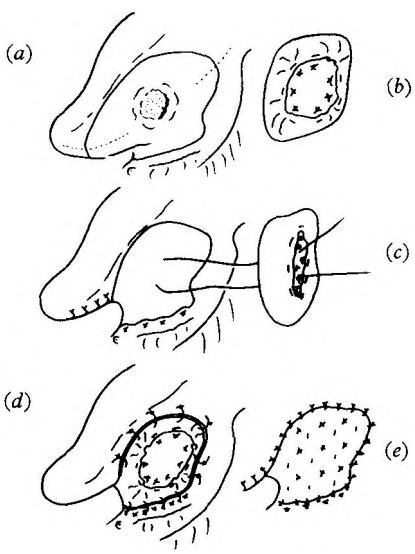


FIG. 599.—(*a*) A diagram of a small squamous cell carcinoma on the lateral margin of the tongue. The outline of the tissue to be excised is shown. Excision with a margin of about 1·5 cm around the palpable extent of the neoplasm with particular care to achieve a proper margin on the deep aspect. (*b*) A pad of soft, polyurethane foam has been cut and a Thiersch graft wrapped round and sewn on to it. (*c*) A transfixing suture has been placed. The tip of the tongue is repaired with sutures and (*d*) the pad and skin graft sewn to the lateral defect. Inset (*e*) shows how a Thiersch graft can be applied by McGregor's quilting technique.

suture transfixes the centre of the pad, picks up the centre of the defect and passed back through the pad and is tied. The margins of the pad are then sewn to the margins of the defect (fig. 599).

Where there is a sizeable loss of tongue a forehead flap makes an excellent repair. The forehead flap is brought into the mouth deep to the zygomatic arch. Adequate space to bring it through may be created by resecting the coronoid process, by sectioning the zygomatic bone or by resection of the anterior part of the temporalis muscle (fig. 600).

Ian Alexander McGregor, Contemporary. Consultant Plastic Surgeon, Canniesburn Hospital, Glasgow.
Gordon Robert Seward, Contemporary. Professor of Oral Surgery, The London Hospital, London.

For very large defects of tongue and floor of mouth a delto-pectoral flap must be brought in via the submandibular wound. It can be de-epithelialised over a strip to enable the oral wound to be sealed by a suture line.

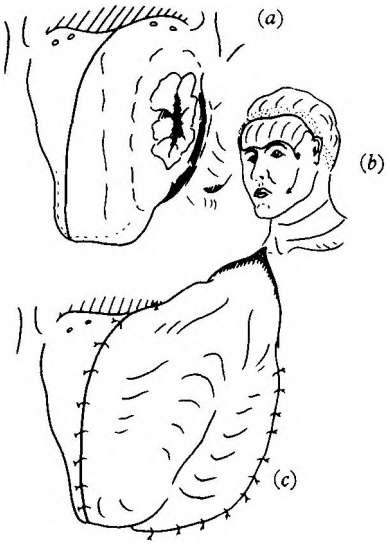


FIG. 600.—(*a*) A diagram of a large squamous cell carcinoma of lateral border of tongue. Excision with a 2 cm margin requires removal of a substantial part of the tongue, floor of mouth and mandible on that side. (*b*) A forehead flap is raised, based on the superficial temporal artery of the same side. A radical neck dissection will be required and the primary tumour should be excised in continuity. (*c*) The flap is brought into the mouth beneath the zygomatic arch, sectioning the zygoma if necessary. The flap is sewn on to the wound margins, folding it to reconstitute the shape of the tongue and lingual sulcus.

Palliation.—Where there is a large, unresectable primary tumour or where there are already fixed nodes in the neck a full dose of therapeutic irradiation should be delivered to the tongue. Where irradiation has been used already and either primary or the nodes are unresectable, resection of the primary alone may make the patient more comfortable. Necrotic lingual carcinoma is difficult to tolerate for the patients, their relatives or the attendants. Irrigation of the mouth with eusol and hydrogen peroxide can reduce the local infection and the foul smell.

In some patients chemotherapy with the Price-Hill regimen may be helpful and indeed, in some patients, of substantial benefit. This treatment involves the administration of a carefully selected list of cytotoxic drugs to a precise schedule. In 1975 Price and others reported a trial involving the use of the following drugs in the treatment of oral squamous cell carcinoma, including cases of carcinoma of the tongue: Vincristine, Adriamycin, Bleomycin, Methotrexate, Fluorouracil, Hydroxyurea and Mercaptopurine.

Rapid advances are being made in this field, and clinicians intending to use this form of treatment would be advised to make themselves familiar with the most recent papers and with the pharmacology and toxic effects of these highly potent drugs. Strict adherence to the chosen advocated regimen is necessary if unexpected problems are to be avoided.

Difficulty with swallowing may require the patient to be fed via a nasogastric tube and respiratory obstruction may require a tracheostomy. Adequate analgesia and sedation with morphine, or the 'Brompton cocktail', should be used to control pain and apprehension (Chap. 62).

Prognosis.—Despite advances in the treatment of cancer and significant improvement in the functional results achieved for patients with carcinoma

Leonard Anthony Price, Contemporary. The Institute of Cancer Research and the Royal Marsden Hospital, and Bridget T. Hill, Contemporary. Imperial Cancer Research Fund.

of tongue, the overall mortality at 5 years has remained surprisingly constant. The prognosis for women is better than men and approaches 50% survival at 5 years while no more than 25% of men are alive at the end of 5 years.

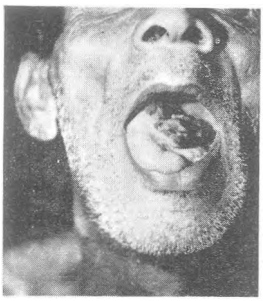


FIG. 601. — Malignant melanoma of the tongue. There were secondaries in the cervical lymph nodes. (*Mohan Kumar, F.R.C.S., Calicut, South India.*)

Sarcoma of the tongue is rare and usually fatal. **Malignant melanoma** of the tongue is also rare and has a bad prognosis (fig. 601), although vigorous local treatment may eradicate the primary tumour and permit death from metastasis.

CHAPTER 33

THE SALIVARY GLANDS

Surgical Anatomy of the Parotid Salivary Gland

Facial Nerve.—The trunk of the facial nerve emerges from the stylomastoid foramen and lies deeply beneath the surface in the angle between the bony external auditory meatus and the mastoid process. It passes forwards around the neck of the condyle of the mandible and rapidly becomes superficial. As it does so, it divides; first into an upper *temporofacial division* and a lower *cervicofacial division* and then subsequently into a varying number of branches, some of which may be interconnected (Pes Anserinus[1]) (figs. 602 and 603).

The Parts of the Gland.—Because the branches of the facial nerve lie, broadly speaking, in the same plane, the gland is described as being composed of a *superficial part* or lobe and a *deep part* or lobe. The superficial part overlies the posterior border of

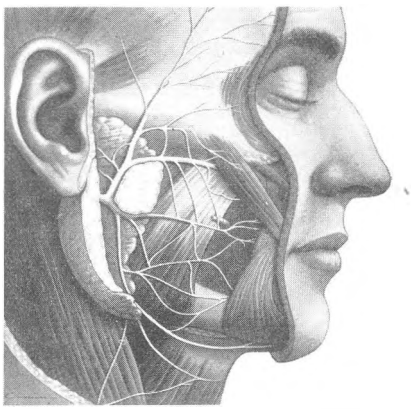


FIG. 602.—Showing the disposition of the facial (seventh) nerve in a case where the superficial lobe has been removed.

FIG. 603.—A photograph of the left facial nerve after a superficial parotidectomy.

the ramus of the mandible and the masseter muscle. A prolongation immediately above the parotid duct is called the *accessory parotid* (or socia parotidis), but only rarely is the latter separated from the rest of the gland. The size of the deep part is less obvious as it is tightly wedged into the space behind the mandible and medial pterygoid muscle. It embraces the lateral surface of the styloid process and the muscles attached thereto and insinuates itself into every adjacent crevice.

The Duct.—The parotid (Stensen's) duct is 2–3 mm in diameter. Beginning deep to and behind the angle of the mandible it curves upwards and forwards through the gland receiving interlobular ducts as it goes. The last three interlobular ducts join it from above, the final one being that from the accessory parotid. At its termination it turns inwards between the fibres of the buccinator muscle and then forwards submucosally to open into the mouth via a small papilla on the inside of the cheek, opposite the second upper molar tooth.

[1] Pes Anserinus = Like the foot of a goose.

Niels Stensen, 1638–1686. Professor of Anatomy, Copenhagen. He abandoned medicine or the church on being appointed Bishop of Titiopolis. He is also regarded as 'The Father of Geology.'

Surgical Anatomy of the Submandibular Gland

The gland is composed of a superficial part and a deep part.

The superficial part lies in the submandibular triangle, above and between the two bellies of the digastric muscle. It is superficial to the sloping undersurface of the mylohyoid muscle and superficial to the hyoglossus. One-third to half its bulk lies below the lower border of the mandible and one half to two-thirds above. The upper part lies in the concavity of the submandibular fossa on the medial aspect of the mandible.

The deep part varies in volume with the size of the posterior sublingual glands and lies below and lateral to the line of the submandibular duct, in the floor of the mouth, and above and deep to the mylohyoid (fig. 604).

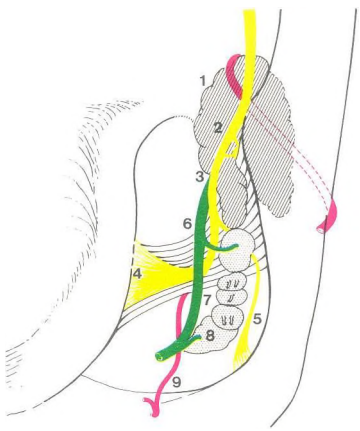


FIG. 604.—Diagram of the relations of the submandibular gland and lingual nerve from above (i.e. through the floor of the mouth, between the tongue and the mandible).

1. The path of the facial artery as it passes upwards in a deep groove in the gland, or through the gland, to reach its lateral surface.
2. The lingual nerve is enclosed in the fascial sheath of the gland at its upper pole. The submandibular ganglion is attached to the nerve at this point and is also within the sheath of the gland.
3. The submandibular duct lies beneath the lingual nerve as it emerges from the upper pole of the gland.
4. The lingual nerve lies first between the duct and the deep part of the gland, then crosses medially beneath the duct to ascend on the hyoglossus to supply the tongue.
5. The lingual nerve gives off a sublingual branch which lies deep in the interval between the sublingual glands and the mandible. It supplies the lingual alveolar mucosa and lateral part of the floor of the mouth.
6. In some subjects a posterior sublingual gland drains into the submandibular duct.
7. Minor sublingual glands drain individually into the plica sublingualis.
8. Bartholin's major sublingual gland may drain via a single duct into the submandibular duct or by a separate opening close to the submandibular papilla.
9. The sublingual artery emerges from deep to the anterior border of the hyoglossus and enters with its fellow of the other side into a small foramen in the mandible, in the midline above the genial tubercles. The artery and sublingual veins lie medial to the submandibular duct.

The duct (Wharton's duct), about 5 cm long, runs forward from the deep part of the gland to enter the floor of the mouth on a papilla beside the frenum of the tongue. The sublingual veins lie lateral and the sublingual artery medial to the duct, more anteriorly.

The lingual nerve is attached to the upper pole of the gland (superficial part) by the fascial sheath of the gland. The sublingual (submandibular) ganglion may be seen attached by two roots to the lower side of the nerve at this point. The nerve passes forwards and downwards between the duct and the deep part of the gland before pass-

Thomas Wharton, 1616–1675. Physician, St. Thomas's Hospital, London. Said to have remained on duty during the Great Plague.

ing medially under the duct opposite the first lower molar. While still lateral to the duct it gives off its sublingual branch, which runs close to the mandible, lateral to the sublingual glands.

The facial artery emerges from under the stylohyoid muscle and either enters the gland substance towards the posterior aspect of its deep surface or deeply grooves the gland. It passes upwards to gain the lateral surface of the gland deep to the lower border of the mandible, around which it curls to enter the face. It lies behind the duct and is at risk from incisions into the upper pole of the gland. Small vessels also enter the gland from the posterior border of the mylohyoid muscle which is firmly attached to the corresponding groove in the gland between the superficial and deep parts.

The venous drainage is into both the anterior facial vein and into the venæ comitantes of the lingual artery.

Sialography

A suitable radio-opaque liquid such as Hypaque® (sodium diatrizoate) or Lipiodol® is introduced into the duct system of the parotid or submandibular gland as appropriate and a radiograph taken (fig. 607). One-half to two ml of solution, depending on the gland, is introduced either through a fine polythene catheter, or a blunt metal cannula which is inserted into the orifice of the duct. The radiograph records the outline of the lumen of the duct system. By this means radiolucent obstructions, and dilatation and narrowing of the duct may be shown. The position and size of a salivary neoplasm, or even an extraglandular mass may be determined because the related ducts will be displaced by the expanding lesion. Fistulæ and abscess cavities can be displayed.

INFLAMMATIONS

Acute Parotitis

May be due to a virus, a non-specific bacterial infection or more rarely actinomycosis or tuberculosis. Mumps is the usual virus infection but other viruses such as the Coxsackie A virus may produce a similar illness. A tuberculous infection of a salivary gland is usually as a result of spread from a tuberculous lymph node.

Bacterial infections mostly reach the gland by ascending the duct from the oral cavity. There are two possible predisposing factors—either a reduction in salivary flow or partial obstruction to the duct with retention of secretions. Salivary flow may be reduced:

Following Major Surgical Operations.—Acute parotitis as a complication of major surgery is less common now that the fluid and electrolyte balance is better controlled. Care should be taken to maintain a high standard of oral hygiene during the postoperative period, and patients with neglected mouths should have dental treatment pre-operatively. Serious acute infections in the dehydrated patient are usually due to *Staphylococcus aureus*, as are the rare metastatic abscesses of the parotid gland. Some infections secondary to obstruction of the duct also may be due to staphylococci but more often are due to *streptococcus viridans* or pneumococci.

During debilitating illness, especially those like typhoid fever and cholera, where the patient may become dehydrated, even while under medical care.

Following radiotherapy where one or more of the major salivary glands are within the field of irradiation. Usually the suppression of salivation following radiotherapy is temporary, but may be permanent.

In Sjögren's Syndrome where the gland substance is destroyed by auto-antibodies (p. 596).

Clinical Features.—There is a brawny swelling of the side of the face (fig. 605). In the case of a virus parotitis or early suppurative parotitis the swelling corresponds to the shape of the gland and raises the lobe of the ear. As suppurative parotitis progresses, widespread cellulitis is seen and the overlying skin becomes dusky red. Pus can be expressed from the parotid duct, and a swab should be taken for culture, identification of the organism, and antibiotic sensitivity tests. The temperature is usually well over 37·8 C. Fluctuation will be elicited only after pus has penetrated the dense fascia of the parotid sheath.

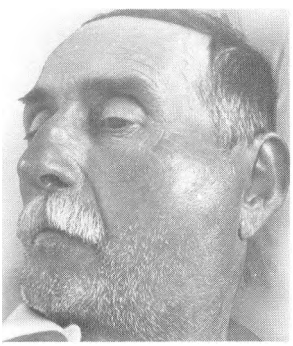


FIG. 605.—Acute parotitis.

Treatment.—Everything should be done to improve the general state of the patient, and antibiotics are administered. Meticulous oral hygiene should be practised with toothbrush and paste where teeth are present and with sodium bicarbonate mouthwashes for the edentulous patient. Dentures should be left out except at meal times. A soft diet is prescribed as chewing is difficult. The gland can be massaged gently at regular intervals to express pus. If pus ceases to drain through the duct, and improvement in the general and local condition is not seen within 48 hours of the start of treatment, drainage of the gland should be considered. Drainage is essential if the dusky redness becomes localised to the lower pole because an abscess here might drain spontaneously into the external auditory meatus.

Drainage of Suppurating Parotitis.—The line of the incision runs vertically in the crease in front of the tragus, curves under the lobe of the ear and curves forward again over the mastoid process and around the posterior aspect of the lower pole of the gland. The transverse incisions in the capsule traditionally used to decompress the gland are rarely necessary. Mosquito artery forceps can be thrust through the fascia and opened within the gland at several levels to effect drainage. Both the extent of the incision and the drainage depend on the severity of the case. A corrugated drain is inserted. When the swelling reduces and drainage ceases the wound in front of the ear may be sutured under local anæsthesia.

Acute Suppurative Sialoadenitis of the Submandibular Gland

Is usually secondary to an obstruction to Wharton's duct. The organism is frequently sensitive to penicillin and incision and drainage rarely required, unless suppuration of the adjacent lymph nodes supervenes.

Recurrent Subacute and Chronic Sialoadenitis

These inflammations are always secondary to a pre-existing condition such as an obstruction of the duct or autoimmune disease (fig. 606). The recurrent attacks of pain and swelling are accompanied by the discharge of

Henrik Samuel Conrad Sjögren, Contemporary. Professor in Ophthalmology, Jönköping, Sweden.

flecks of pus in the saliva. The infection should be controlled by antibiotics and then a sialogram performed. If the sialographic changes are confined to the side affected by infection then an obstructive cause is most likely (fig. 607). Where changes are present on both sides Sjögren's syndrome is the more likely predisposing cause even if the attacks of pain and swelling are only unilateral. However, calculi can be a complication of the reduced salivary flow of Sjögren's syndrome and this possibility should be borne in mind (p. 596).

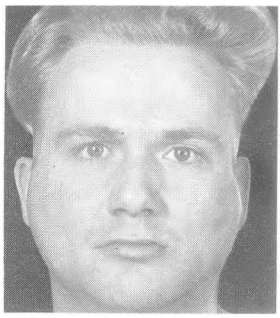


FIG. 606.—Sjögren's syndrome with superimposed bilateral chronic bacterial parotitis. Present for nine months.

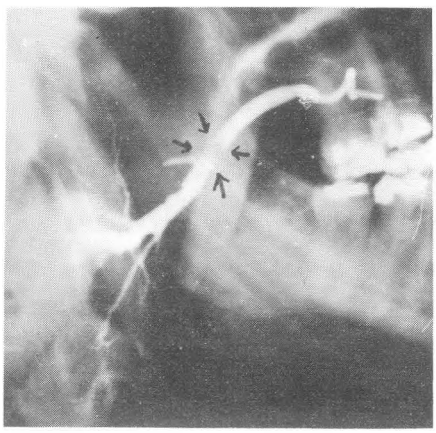


FIG. 607.—A parotid sialogram showing an oval filling defect due to a calculus.

Recurrent Subacute Parotitis of Childhood

The patient may present at any age between 3 months and 10 years with swelling of one of the parotid glands. Both pain and constitutional symptoms are mild. There may have been one or more previous episodes each lasting 10–14 days and affecting either gland. The first attack may have been mistaken for mumps. A sialogram reveals an appearance of multiple, small, proximal dilatations—like a snowstorm. The appearance is called punctate sialectasis. While characteristic of this particular condition, punctate sialectasis may also be seen in Sjögren's syndrome, or after irradiation of the gland.

Katzen has found that the condition remits spontaneously by age 15. His biopsy material shows an appearance suggestive of an autoimmune cause and simliar to that seen in Sjögren's, but there is no evidence that the childhood condition precedes, or progresses to, adult Sjögren's. More than one child in the family and the children of more than one generation may be affected, suggesting a congenital predisposition to the condition. Antibiotics will control the acute episodes, but a sialogram will often cut short the attacks.

Children who combine recurrent parotid swelling with the upper respiratory allergic condition may benefit from regular doses of an anti-histamine mixture.

Obstruction to the Duct of a Major Salivary Giand

The characteristic symptom is recurrent painful swelling of the affected gland at meal times (fig. 608), but the first indication may be an acute or subacute infection as described above.

Michael Katzen, Contemporary. Surgeon, Transvaal Memorial Hospital for Children and Department of Pediatric Surgery, University of Witwatersrand, Johannesburg, South Africa.

Proximal to a site of chronic obstruction the duct will be dilated with retention of secretions and chronic infection. Causes of obstruction include salivary calculi, strictures of the duct wall, œdema or fibrosis of the papilla, pressure on the duct due to an adjacent mass, or invasion of the duct by a malignant neoplasm.

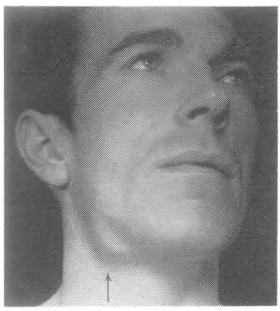
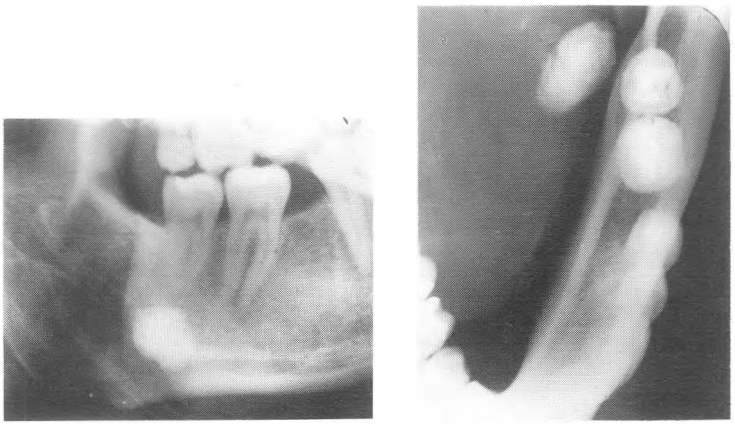


FIG. 608. — Enlargement of the submandibular salivary gland due to a calculus in Wharton's duct.

Papillary Stenosis.—Ulceration of the papilla of either the submandibular or parotid duct may follow trauma from a denture. Ulceration of the parotid papilla may also follow irritation from a sharp tooth, or a bite of the cheek. The obstruction and recurrent swelling will subside as the ulcer heals. Repeated trauma results in fibrosis which is only relieved by papillotomy with suture of the duct lining to the oral mucosa.

Salivary Calculi.—*Submandibular calculi* are by far the most common and are relatively easy to demonstrate by plain radiography (fig. 609). *Calculi within the duct* may be removed via the floor of the mouth, although those near to the gland require special experience. For calculi anteriorly in the floor of the mouth a stitch is passed under the duct proximal to the stone



(*a*) (*b*)

FIG. 609.—(*a*) An oblique lateral radiograph demonstrating a submandibular calculus, but the image of the stone is superimposed on the mandible. (*b*) A posterior oblique occlusal view which reveals the stone, medial to the mandible and without bony superimposition. A small calculus at this site in the upper pole of the submandibular gland is best shown by a posterior oblique occlusal.

to stop the stone slipping backwards. An incision is made in the mucosa over the duct and the latter mobilised. A stay suture is passed under the duct to bring it up into the top of the wound and to control it while the wall is incised to release the stone. *Where the calculus is within the intraglandular* part of the duct (fig. 609), or the gland severely damaged by chronic infection, then excision of the gland is indicated.

Excision of the submandibular gland proceeds as follows:—The gland is removed through an incision in a skin crease over the lower third of the gland and about 5 cm long. The tissues are divided down to the platysma muscle and the subcutaneous tissues separated from the surface of the muscle by pressure with a swab (gauze sponge) to facilitate closure of the wound in layers. The muscle is divided to expose the deep fascia which is opened with care to avoid damage to the facial nerve. The marginal mandibular branch of the facial nerve is sought beneath the deep fascia and retracted upwards. The anterior facial vein is found within the fat superficial to the gland. If the anterior facial vein is divided over the gland and below the marginal mandibular branch it may be retracted upwards, drawing the nerve upwards and out of the operative field, because the nerve passes superficial to the vein. The lower pole of the gland is freed keeping close to the surface of the gland, grasped with Allis' tissue forceps and turned upwards and forwards. The posterior belly of the digastric and the stylohyoid muscles are identified and retracted backwards with a Langenbeck's retractor. The facial artery with its veni comitantes will be seen emerging from deep to the muscles and entering the deep surface of the gland. It is ligated three times using an aneurysm needle and divided between the distal two ligatures. Double ligation of the proximal end is advisable as the artery retracts beneath the muscles once it is cut and a slipped ligature results in troublesome hæmorrhage. The facial artery is divided again at the lower border of the mandible and the gland drawn down and separated from the lower border of the jaw dissecting close to the gland substance. The submandibular branch of the facial artery will be divided as this is done. If the lower pole is now retracted backwards the posterior border of the mylohyoid muscle may be separated with scissors from the groove between the superficial and deep parts of the gland. Small arteries entering the gland from the muscle should be sealed with diathermy. A finger is passed around the gland separating loose connective tissue around the capsule and the gland drawn down to bring the lingual nerve into view. The nerve is within the fascial sheath of the gland at the upper pole and will be drawn into a V shape by traction on the gland. The nerve is separated under direct vision after which the duct may be drawn down through the loop of the nerve. The duct should be clamped, divided and ligated well forward to leave only a short stump. If the dissection keeps close to the under surface of the gland the hypoglossal nerve should remain covered with loose connective tissue and should not be at risk. Sometimes veins from the gland drain into the veni comitantes of the hypoglossal nerve or lingual artery and will be divided as the gland is separated. They must be clamped and ligated with care or the nerve will be damaged. The wound is closed in layers with vacuum drainage.

Parotid calculi are not infrequent, but only the larger ones can be seen against the soft tissue image of the cheek in plain radiographs. Sialograms are necessary to identify and locate them but even these are not always easy to interpret.

Parotid calculi anterior to the accessory parotid can be reached via the oral cavity. A ≺ shaped incision is made around the parotid papilla and a flap raised which includes the submucosal part of the duct. The duct is mobilised for a distance out into the cheek and drawn as a loop into the mouth so that the calculus can be reached. Calculi in the intraglandular part of the duct are approached through a pre-auricular incision as for parotidectomy. The duct is identified at the anterior border of the gland and traced backwards until the calculus is uncovered. Care is taken to identify and conserve related branches of the facial nerve.

Strictures and Fistulæ

If a duct ulcerates around a calculus a characteristic short stricture will form after the calculus has been removed. There will then be a recurrence of mealtime swelling. Removal of the stricture or excision of the gland will be required. Fortunately the submandibular duct is the one most often affected. A stricture anterior to the second molar region may be circumvented by transecting the duct behind the obstruction, slitting the end to increase the size of the opening and implanting it in the floor of the mouth.

Oscar Huntingdon Allis, 1836–1921. American Surgeon.
Bernard Rudolph Konrad von Langenbeck, 1810–1887. German Surgeon.

Some submandibular calculi ulcerate directly into the mouth and an internal fistula is formed. Unless there is also a stricture present this is of no consequence.

Bilateral strictures affecting sizeable lengths of the parotid ducts are seen. The ducts proximal to the stricture dilate and both recurrent obstructive swellings and recurrent infective swellings are experienced by the patient. Mostly these patients do not have positive tests for autoimmune antibodies, nor do they suffer from rheumatoid arthritis, and they lack the characteristic histological changes as seen in labial salivary gland biopsies in Sjögren's syndrome. Dilatation of the stricture with gum elastic filiform bougies lubricated with lignocaine gel will often relieve symptoms temporarily.

Partial excision or a laceration of the parotid gland may be followed by an accumulation of saliva in the tissues. Aspiration of the pool of saliva through an oblique needle track, followed by the application of a firm pressure dressing, and repeated as necessary will cure this, but may take some weeks or months. Incisional drainage of such a collection of saliva, or of pus in a parotid abscess can result in an external parotid fistula as can a laceration involving Wharton's duct.

Management of Fistulas.—Fistulas from divided gland substance usually granulate and heal. Duct fistulas need to be explored because quantities of saliva pour out on to the face each meal time, and even the amount which leaks out between meals is sufficient to keep the skin and a dressing moist. Following a pre-operative sialogram a flexible bougie is passed into the duct from the mouth, to mark the distal segment. A pre-auricular flap is raised and the proximal end is sought. If repair without tension is possible the ends of the divided duct are slit longitudinally so that they may be joined by an oblique suture line which will not contract to a stricture.

If the distal part has been destroyed, or if a stricture obliterates part of the distal 2 cm of duct, continuity can be restored using a rectangular pedicle flap of mucosa from the inner aspect of the cheek which is rolled into a tube and anastomosed to the duct end (fig. 610). If no reconstructive procedure is possible the gland is excised.

Cysts

As a rare entity a cyst lined with epithelium may be found in the parotid gland and behind the angle of the mandible. The origin of such cysts is uncertain, they may be related to the developmental preauricular sinuses occasionally seen in front of the ear, or they may be related to the branchial cleft cysts which more typically are found beneath the upper third of the sternomastoid muscle. A lateral dermoid cyst of the floor of the mouth which develops from the first branchial cleft can enlarge backwards until the submandibular salivary gland is spread out over its surface. It will then present as a fluctuant swelling both in the floor of the mouth and in the submandibular triangle. The treatment of these cysts is enucleation.

NEOPLASMS

The International Classification of Salivary Gland Tumours is recommended and reproduced below.

I. Epithelial Tumours

A. Adenomas
   1. Pleomorphic[1] adenoma (mixed tumour)
   2. Monomorphic adenomas
      (a) Adenolymphoma
      (b) Oxyphilic adenoma
      (c) Other types

B. Mucoepidermoid tumours

C. Acinic cell tumour

D. Carcinoma
   1. Adenoid cystic carcinoma
   2. Adenocarcinoma
   3. Epidermoid carcinoma
   4. Undifferentiated carcinoma
   5. Carcinoma in pleomorphic adenoma

II. Non-Epithelial Tumours

[1] Pleomorphic = many forms.

Many salivary neoplasms are distinctly rare and of interest only to the specialist surgeon and pathologist. Only those which are common enough to be of general interest will be described.

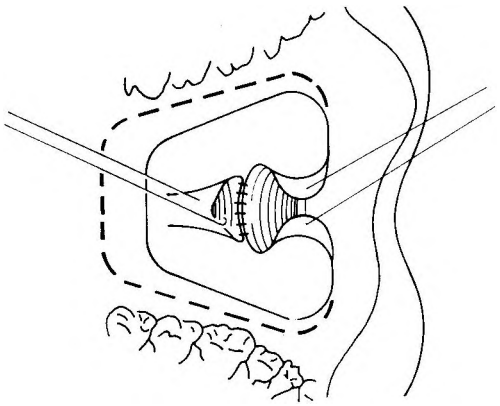


FIG. 610.—The technique of using a flap of cheek mucosa to repair a damaged parotid duct. The mucosa is tubed and sutured to the proximal segment of duct. In the case of a stricture the obstructed segment is excised prior to the anastomosis.

Incidence.—Approximately 75% of all salivary neoplasms arise in the parotid gland. Of these some 80% are benign and 80% of the benign tumours are pleomorphic adenomas.

Approximately 15% of salivary neoplasms arise in the submandibular salivary gland. Of these 60% are benign and 95% of the benign tumours are pleomorphic adenomas.

Approximately 10% of salivary neoplasms arise in the minor salivary glands of palate, lip and cheek and in the sublingual glands; with the site incidence decreasing in that order. Only 40% of these are benign but virtually all the benign tumours are pleomorphic adenomas.

Pleomorphic Adenomas.—Epithelial cells proliferate in strands and some take on a duct like arrangement. Other cells, probably of myoepithelial origin, proliferate in sheets. In parts of the tumour a mucoid material is produced which separates the cells producing a myxomatous appearance and then an appearance resembling cartilage in histological sections. On occasions sufficient mucoid material may accumulate to produce a cystic part to the swelling. In general the tumour is slow growing and forms a firm or elastic, lobulated mass (figs. 611 and 612). The pleomorphic adenoma is classed as benign, but strands of tumour cells tend to penetrate the thin capsule of compressed gland substance and connective tissue which surrounds it. Further, lobules of tumour attached only by a narrow neck of tissue may extend beyond the main limits of the mass. For these reasons, simple enucleation will leave residual neoplasm behind and result in multicentric recurrence. After perhaps 10, 20 or 30 years a few pleomorphic adenomas will exhibit rapid growth and clinical signs of malignancy. This is described as a carcinoma arising in pleomorphic adenoma.

Adenolymphoma or Warthin's Tumour.—A benign neoplasm composed of a double-layered epithelium which lines spaces which are frequently cystic. The epithelium is markedly eosinophilic and the inner cells are

Aldred Scott Warthin, 1866–1931. Professor of Pathology, University of Michigan, Ann Arbor, U.S.A.

columnar. It tends to be folded inwards into the cavities to produce a papillary appearance in section. Characteristically the stroma contains lymphoid tissue, including lymph follicles.

Clinically it presents as a slowly enlarging soft, sometimes fluctuant swelling, usually towards the lower pole of the parotid gland in a middle age

FIG. 611.—Pleomorphic adenoma of parotid gland, typical location.

FIG. 612.—Unusually large pleomorphic adenoma of the submandibular salivary gland. It weighed 1·2 kg. (*M. K. Rana, M.S., Mandvi-Kutch, India.*)

or elderly male. More than one tumour may be found at times, either on one side, or one bilaterally. Adenolymphomas form 10% of parotid tumours, are rare in the submandibular gland, and found only exceptionally in the minor glands. Unlike all other neoplasms which form a ' cold ' spot, the adenolymphoma produces a ' hot ' spot in a $^{99}Tc^{m}$ Pertechnetate ' scan ' so that a firm pre-operative diagnosis is possible without biopsy. Other types of monomorphic adenoma are rare. Again, they are found in older people and may be bilateral.

Muco-epidermoid Tumour.—Is composed of sheets and masses of epidermoid cells and clefts and cystic spaces lined by mucous secreting cells. The cartilage-like appearance and myxoid appearance characteristic of the pleomorphic adenoma is not seen.

These tumours are of varying degrees of differentiation and speed of growth. Mostly they are slow growing and invade local tissues to a limited degree and only occasionally metastasise to lymph nodes, lungs or skin. Clinically they are usually hard; noticeably harder than a typical pleomorphic adenoma, yet become fixed only when large. Mostly they do not cause facial paralysis when they occur in the parotid gland.

Acinic Cell Tumour.—Almost all acinic cell tumours occur in the parotid gland. They are composed of cells resembling those of serous acini. They occur in women more often than in men. This comparatively rare and usually slow growing neoplasm is important in that like the muco-epidermoid carcinoma it is invasive, though, if slow growing, to a limited extent. Again, like the muco-epidermoid carcinoma, those with a relatively benign

appearance may metastasise unexpectedly. Acinic cell tumours tend to be soft and occasionally cystic.

Adenoid cystic carcinoma consists of myoepithelial cells and duct epithelium cells. The former form sheets of cells within which a basophilic material accumulates in blobs to give them a cribriform, or lace-like appearance. Duct epithelium cells form strands and cords, but tend also to form duct-like structures and microcysts in which an eosinophilic material accumulates. These also add to the cribriform appearance.

Many adenoid cystic carcinomas are comparatively slow growing and difficult to differentiate clinically from the previously described tumours. However, sooner or later pain, areas of anæsthesia of the skin and paralysis of muscles appear due to involvement of related nerves. Unlike the previously described neoplasms, however, the chance of eradicating an adenoid cystic carcinoma is poor because it infiltrates for long distances in the perineural tissues of adjacent nerves and may invade medullary bone for many centimetres before inducing significant bone resorption. Thus, the tumour is always more extensive than the physical signs or radiographic appearances suggest. The percentage incidence increases from parotid to submandibular to accessory salivary glands. The adenoid cystic carcinoma tends to be both hard and fixed.

Adenocarcinomas, Epidermoid Carcinomas and Undifferentiated Carcinomas.—Some salivary carcinoma cells and cell arrangements resemble the various glandular elements seen in salivary glands. These are the adenocarcinomas. Some specialist pathologists subdivide these according to the predominant cell type, but their subdivisions are not of clinical importance. Epidermoid and undifferentiated carcinomas resemble those seen at other sites.

All these neoplasms tend, at an early stage, to produce obvious clinical signs of malignancy. These are fixation, resorption of adjacent bone, pain, anæsthesia of skin or mucous membrane and paralysis of muscles. In the case of the parotid gland, facial nerve irritability occurs and muscle spasm can be produced if the tissues over the nerve are tapped. Later this gives way to facial paralysis. Limitation of mandibular movement results from invasion of the jaw and masticatory muscles.

Treatment of Salivary Tumours

Treatment of Slow Growing Salivary Neoplasms.—Slow growing *parotid* neoplasms are assessed clinically and are *not biopsied* (p. 595). Warthin's tumours and pleomorphic adenomas are usually recognisable with reasonable certainty. Both are excised by partial parotidectomy with conservation of the facial nerve (fig. 613). The main trunk is identified proximal to the gland and each branch is traced out in turn (fig. 614). A margin of normal gland and if necessary masseter muscle, is taken with a pleomorphic adenoma. The connective tissue sheath of the nerve is adequate margin at this site and only the occasional branches actually penetrating the tumour need be sacrificed and replaced by a graft. Unusually hard and fixed, slow

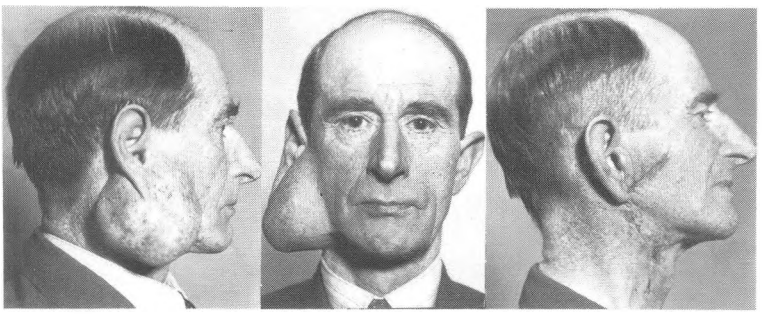


FIG. 613.—Mixed parotid tumour of over twenty years' duration, latterly increasing rapidly in size. Complete parotidectomy with preliminary ligation of the external carotid artery. The facial nerve was preserved. The patient was free from recurrence twelve years later.

growing, neoplasms are treated similarly, but a larger margin is taken with a greater readiness to sacrifice nerve branches. Further treatment depends on the histological report and adequacy of the excision. Most muco-epidermoid carcinomas will be effectively managed this way.

Slow-growing *submandibular* gland neoplasms not fixed to the mandible are treated by excising the whole gland with the adjacent connective tissue, but conserving lingual and hypoglossal nerves. To prevent rupture and

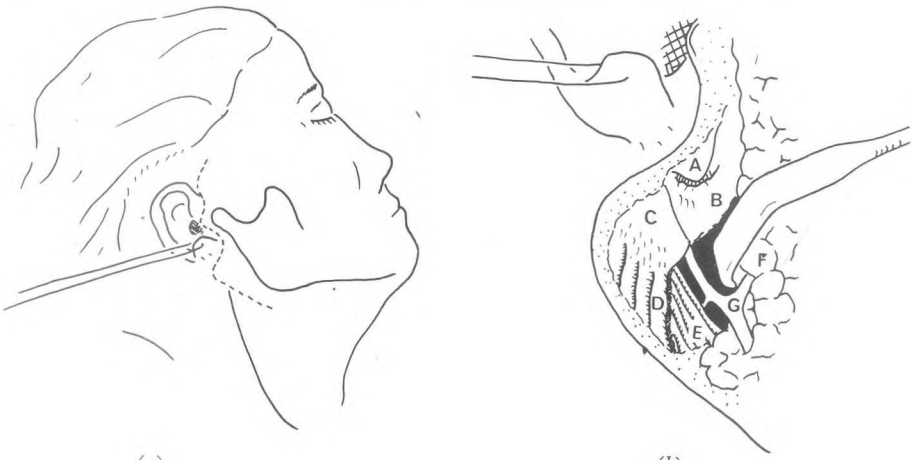


FIG. 614.—**Parotidectomy.** (*a*) The dotted line outlines the pre-auricular incision through which the parotid gland is explored. It starts within the hair line, uses the edge of the tragus, turns underneath the lobe of the ear and then turns again over the mastoid process to follow a skin crease in the neck. The whole or part of this line is used for an incision as indicated.

(*b*) The main trunk of the facial nerve (G) and the two main divisions as exposed at the beginning of a superficial parotidectomy. Each division and branch is traced out in turn to permit excision of the gland. The important landmarks for finding the nerve are:
(A) the pointed lower end of the tragal cartilage,
(B) the bony external auditory meatus,
(C) the mastoid process,
(D) the sternomastoid muscle,
(E) the posterior belly of the digastric.
(F) the retracted lower pole of the parotid gland.

seeding of the neoplasm the gland must not be grasped with tissue forceps or other instruments, nor biopsied.

After a pre-operative biopsy, *palatal* pleomorphic adenomas are circumscribed with an incision well wide of the obvious swelling as the tumour here is flattened and dish-shaped rather than spherical. The periosteum is raised from the bone, the greater palatine vessels mobilised and drawn down until they can be clamped, divided, and diathermised beyond the tumour. The wound is packed and heals by granulation. Palatal bone need be sacrificed only if obviously invaded clinically or radiographically.

Biopsy?—Salivary neoplasms shed cells if they are incised and will ' seed ' readily giving rise to recurrence. Biopsy of the parotid also carries the risk of damage to the facial nerve. Pre-operative biopsy is largely confined to tumours of the sublingual and accessory glands, as these carry the greatest likelihood of being malignant.

Dumbell Parotid Tumours.—Some parotid neoplasms arising in the deep part of the gland produce virtually no visible or palpable swelling in the pre-auricular region. The tumour enlarges medially, passing between the styloid process and the mandible to present as a swelling of the soft palate, lateral wall of pharynx and posterior pillar of the fauces. Computerised axial tomography will demonstrate the size and anatomical relations of the mass.

If, on examination under anæsthesia, a lump with a history suggestive of slow growth is palpably lobulated and the lateral pharyngeal wall moves freely over it, the assumption may be made that it is a pleomorphic adenoma and no biopsy performed. Other tumours should be biopsied through a limited incision in the posterior pillar of the fauces and the biopsy site removed with the tumour.

The surgical approach is through a parotidectomy incision with a submandibular extension. After mobilising the facial nerve and its branches from the parotid gland the mandible is divided anterior to the mental foramen and the angle retracted upwards. Division of the styloid process between the origin of the stylopharyngeus and the styloglossus and stylohyoid muscles completes access to the tumour for dissection under direct vision. The parotid gland and the tumour, with a covering of connective tissue, are removed together. The mandible and styloid process are repaired and the wound closed with vacuum drainage.

Treatment of Neoplasms which present with Clinical Signs of Malignancy

Where the neoplasm presents the clinical signs of malignancy, a radical excision is carried out. In the case of the parotid gland the facial nerve is sacrificed, but may be grafted using the great auricular nerve. Such other structures are sacrificed as are both surgically reasonable and necessary. A specimen can be taken for frozen section during the operation and before any irrevocable or mutilating step is carried out. Depending on the pathologist's report on the type of neoplasm and the adequacy of the surgical excision, postoperative radiotherapy is arranged. Unfortunately most salivary neoplasms are relatively radio-resistant.

Radical excision of a parotid neoplasm is limited by the internal carotid artery on its deep aspect and the cranial cavity posteriorly. Excision of the parotid including the mandible is straightforward, but inclusion of the temporal bone requires special surgical expertise.

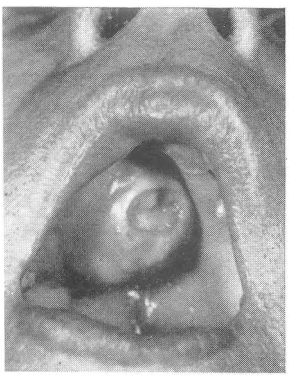


FIG. 615.—Ulcerating salivary gland neoplasm of palatal mucous salivary glands. A neoplasm presenting like this is likely to be a muco-epidermoid carcinoma or an adenocystic carcinoma.

A similar management is followed for the submandibular gland with fewer anatomical restraints. Radical excision of the submandibular gland with, if necessary, the adjacent mandible and tongue and in continuity with a neck node dissection presents no special problems.

In the case of the minor salivary glands a biopsy is made pre-operatively (fig. 615). Excision with a wide margin is carried out. In the case of palatal neoplasms this means a maxillectomy. The cavity in the upper jaw is lined with a split skin graft held in place by a gutta percha bung on a dental plate which is wired to the remaining facial skeleton. Most adenoid cystic carcinomas require postoperative radiotherapy irrespective of their site of origin.

Postoperative Persistent Facial Paralysis

Where nerve grafting is not possible or proves unsuccessful following radical surgery, consideration can be given to transposing the hypoglossal nerve and anastomosing it to the peripheral branches. Fascial slings may also be used to support facial tissues and mask the deformity (fig. 616).

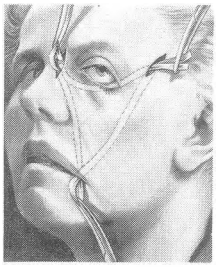


FIG. 616.—A strip of fascia lata inserted subcutaneously as shown helps to overcome the deformity of facial palsy. (*After W. O. Lodge.*)

Non-epithelial Tumours

Hæmangiomas, lymphangiomas and neurofibromas may involve the parotid salivary gland. Hæmangiomas most often present during childhood and account for almost half of the hamartomatous and neoplastic parotid lesions seen in this age group. Excision requires special care, not only because of the problems of hæmorrhage, but because the facial nerve is not so deeply placed in the child.

THE AURICULO-TEMPORAL SYNDROME (FREY'S SYNDROME)

In this condition there is flushing and sweating of the skin innervated by the auriculotemporal nerve whenever salivation is stimulated. The condition follows surgery in the region of the parotid gland or temporomandibular joint, but may follow accidental injury of the parotid gland or joint. Some cases are congenital and possibly due to birth trauma. It is thought that following injury to the auriculotemporal nerve, postganglionic parasympathetic fibres from the otic ganglion become united to sympathetic nerves from the superior cervical ganglion destined to supply the vessels and sweat glands of the skin. Mostly the condition is an inconvenience rather than a real disability. Only in severe cases is division of the lesser superficial petrosal nerve within the skull considered as a means of interrupting the secretomotor fibres.

SJÖGREN'S SYNDROME

Sjögren's syndrome is the clinical triad of dry eyes (keratoconjunctivitis sicca), dry mouth (xerostomia) and rheumatoid arthritis. More recently other connective tissue diseases such as systemic lupus erythematosis or scleroderma have been recognised as a possible third component. If only the dry eye and dry mouth aspects of the condition are present the patient is said to have the sicca syndrome. In these patients the salivary and lacrimal glands are infiltrated with lymphocytes and the acini progressively destroyed. The epithelium of the ducts becomes hyperplastic, forming casts within the lumen and blocking smaller ducts. Mucous gland metaplasia of the duct epithelium leads to the formation of a gelatinous saliva in some patients. Strictures, duct dilatations and ascending infection complicate the picture. The histological changes can be seen in biopsies of the labial mucous salivary glands. Hypergamma-globulinæmia may be detected in an electrophoretic strip and autoantibodies

Luiji Frey, 1889–1944. Physician, Neurological Clinic, Warsaw, Poland.
Henrik Samuel Conrad Sjögren, Contemporary. Professor of Ophthalmology, Gothenburg, Sweden, described this syndrome in 1933.

such as rheumatoid factor, antinuclear factor and salivary duct antibody may be demonstrated in the serum. The lack of lacrimal secretion can be shown by the Schirmer test and the keratitis by Rose Bengal and fluorescein staining. Salivary secretion studies are conducted by collecting saliva under standard conditions using small cups applied by a vacuum ring over the parotid papillæ. Gland function can also be quantified by $^{99}Tc^{m}$ Pertechnetate scans.

The effect of the disease in the salivary glands is a progressively more severe dryness of the mouth. Rampant, uncontrollable caries of the teeth follows. After the loss of the natural teeth, dentures are almost unwearable and lips, tongue and palate stick together. The tongue becomes cracked and attacks of monilia stomatitis occur.

Intense infiltration of the glands with lymphocytes can result in diffuse enlargement or the formation of localised nodules, which must be distinguished from neoplasms.

Mikulicz Disease.—Mikulicz described his triad which constituted (1) Symmetrical enlargement of all the salivary glands (2) Narrowing of the palpebral fissures due to enlargement of the lachrymal glands and (3) Parchment-like dryness of the mouth (fig. 617). Subsequently Mikulicz disease and Mikulicz syndrome were recognised, both producing the same clinical picture. Mikulicz syndrome is enlargement of the salivary and lacrimal glands due to leukæmia or some other generalised disease resulting in infiltration of the glands with round cells. Mikulicz disease is due to an autoimmune process in the glands themselves and is generally looked upon as a clinical variant of Sjögren's syndrome.

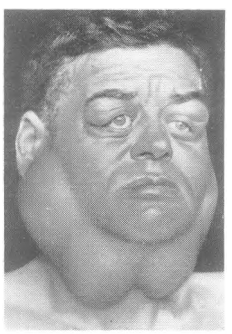


FIG. 617. — Mikulicz disease. (*C. Fischer.*)

Treatment.—The dry eyes can be treated by diathermy obliteration of the lacrimal punctum and the instillation of artificial tears composed of methyl cellulose drops. Meticulous oral hygiene with toothbrush and paste, dental floss and a special 1% chlorhexidine gluconate preparation will help control the caries and periodontal disease and delay tooth loss. Methyl cellulose mouth washes help to keep the mouth moist, but are less successful than artificial tears.

Steroids and immunosuppressive agents will generally alter the course of the disease, but their use is rarely warranted in view of their side effects. In addition, such treatment increases the risk of ascending infection. Radiotherapy will reduce the enlargement of Mikulicz disease, but increases both the dryness of the mouth and any tendency to attacks of infection.

Patients with Sjögren's syndrome are at greater risk than the rest of the population from the development of reticulum cell sarcoma, either in the glands, or in the related lymph nodes.

Other Causes of Salivary Gland Enlargement

A variety of drugs will cause enlargement of the salivary glands in a percentage of patients, notably those used in the treatment of thyrotoxicosis. Many other drugs reduce the salivary flow, cause dryness of the mouth, and predispose to ascending infection of the salivary glands.

Sialosis is enlargement of the salivary glands for metabolic reasons. It is seen in certain hormonal disturbances such as diabetes and acromegaly, in patients who stick to bizarre diets, and in patients who are overweight. In the case of the latter the glands may be infiltrated with fat. The condition is important in drawing attention to an underlying cause or in the differential diagnosis from other causes of salivary gland enlargement.

Johann von Mikulicz-Radecki, 1850–1905. Polish Surgeon. Described this disease in 1892.

CHAPTER 34

THE NECK

THE BRANCHIAL APPARATUS AND ITS ABNORMALITIES

In a fœtus approximately thirty-five days old, four grooves can be seen on each side of the neck. These are the branchial clefts, which resemble the gills of a fish; the intervening bars are the branchial arches. Each arch contains a central cartilage. The clefts in human embryos are composed of grooves on the outside and pouches on the inside (pharynx). The first cleft persists as the external auditory meatus; the second, third, and fourth clefts normally disappear. The whole, or a portion, of one of the clefts that normally disappear may persist. Alternatively, a portion can become sequestrated.

Branchial Cyst.—From the vestigial remnants of the second branchial cleft the cyst is usually lined by squamous epithelium, and its contents are either clear fluid or like toothpaste. It appears in young adults, sometimes in later life. It protrudes from beneath the anterior border of the upper third of the sternomastoid as a fluctuant swelling which may transluminate. If infection has occurred, it may be difficult to differentiate from a tuberculous abscess. If the aspirated fluid contains cholesterol crystals, the diagnosis is made.

A rare variety of branchial cyst is found lying closely related to the pharynx. It is lined by columnar epithelium, and filled with mucus. Occasionally small symptomless cysts of this type are discovered at necropsy.

Treatment.—A branchial cyst should be excised. Through an incision parallel to the skin creases the anterior wall of the cyst is exposed and some of the content is aspirated. This procedure permits the wall of the cyst to be grasped in suitable forceps, and assists materially in a dissection which, in some instances, entails following a track that passes through the fork of the common carotid artery as far as the pharyngeal wall. It passes superficial to the hypoglossal and glossopharyngeal nerves but deep to the posterior belly of the digastric. The hypoglossal and the spinal accessory nerve may be in danger and should be recognised and protected. If the specimen is examined microscopically, the wall of the cyst is often found to be surrounded by a layer of lymphadenoid tissue. This suggests that the cyst arose as a result of branchial epithelium becoming entrapped within a lymph node during development. It also explains why branchial cysts become inflamed: the lymphadenoid shell participates in regional lymphadenitis.

Fig. 618.—Bilateral branchial fistulæ; left side inflamed.

Branchial fistula may be unilateral or bilateral (fig. 618) and it is highly probable that the fistula represents a persistent second branchial cleft, the occluding membrane of which has broken down. Nearly always the external orifice of the fistula is situated in the lower third of the neck near the anterior border of the sternomastoid muscle.

The internal orifice is located on the anterior aspect of the posterior pillar of the fauces, just behind the tonsil (Wilson), but more often than not the track ends

Charles Paul Wilson, 1900–1970. Surgeon, Ear, Nose and Throat Department, Middlesex Hospital, London.

blindly on the lateral pharyngeal wall, the condition then being a sinus rather than a fistula. The track is clothed with muscle and lined by ciliated columnar epithelium until destroyed by recurrent attacks of inflammation. The discharge is mucus or muco-pus. The condition may also be secondary to an incision in an infected branchial cyst.

Treatment.—When causing troublesome symptoms, e.g. by a discharge of mucus, branchial fistulæ should be excised.

Operation.—A ureteric catheter or a probe is passed up the track via the orifice, so that by means of an incision higher in the neck the track may easily be identified and dissected as it passes through the fork of the common carotid artery towards the pharyngeal wall. When the upper dissection is completed the orifice below is freed by a small elliptical incision and the whole of the lower part of the track can be eased out of the upper wound (Small) (fig. 619).

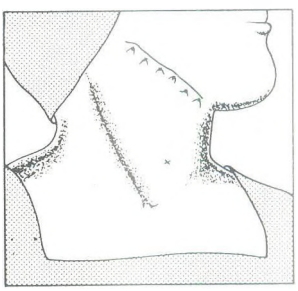


FIG. 619.—The catheter method of removing a branchial fistula, showing the lower elliptical incision marked by x.

Branchial Cartilage.—A small piece of cartilage, connected to the deep surface of a cutaneous dimple in the position of an external orifice of a branchial fistula, is occasionally encountered.

Cervical Auricle.—So named because of its morphological significance, this cutaneous projection is found almost invariably in the position of the external orifice of a branchial fistula. Cervical auricles were recognised in the days of the Roman Empire, and are represented in some of the statuary of that period.

Pharyngeal Pouch (p. 692)
Laryngocele (p. 708)
} are possibly derived from the branchial apparatus.

Branchiogenic carcinoma is very rare; such a diagnosis is unjustifiable until every possible source of a primary growth in the mouth, pharynx, and external auditory meatus has been excluded.

Cystic Hygroma (Cavernous Lymphangioma)

About the sixth week of embryonic life the primitive lymph sacs develop in mesoblast, the principal pair being situated in the neck between the jugular and subclavian veins; these, which correspond to the lymph hearts of lower animals, are known as the jugular lymph sacs. Sequestration of a portion of a jugular lymph sac from the lymphatic system accounts for the appearance of these swellings.

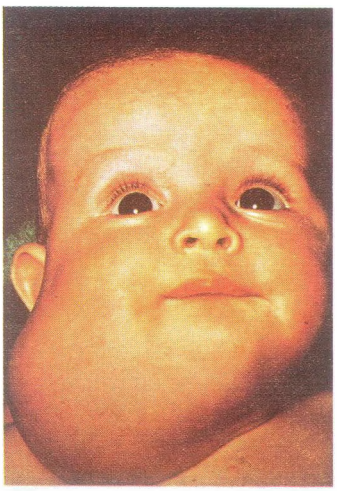


FIG. 620.—A cystic hygroma.
(*Harold William Rodgers, F.R.C.S.*)

Of all the swellings of the neck, cystic hygroma rivals, and at times surpasses, sternomastoid 'tumour' as the earliest to appear: usually it manifests itself during early infancy, occasionally it is present at birth, and exceptionally it is so large as to obstruct labour. Typically, the swelling occupies the lower third of the neck, and as it enlarges it passes upwards towards the ear (fig. 620); often it is the posterior triangle of the neck that is mainly involved. Due to intercommunication of its many compartments, the swelling is softly cystic and is partially compressible; it visibly

Hugh Alan Dugleby Small, Contemporary. Formerly Surgeon, The Royal Northern Hospital, London.

increases in size when the child coughs or cries, but *the* characteristic that distinguishes it from all other cervical swellings is that it is brilliantly translucent.

The cheek and the axilla are other, though less frequent, sites for a cystic hygroma. Another infrequent, though striking dual lesion is that of a cystic hygroma and a lymphangiogenetic macroglossia. Exceptionally a cystic hygroma occurs in the groin or in the mediastinum. When situated wholly within the thorax (Chap. 40), it cannot be differentiated, prior to operation, from other benign neoplasms.

Clinical Course.—The behaviour of cystic hygromas during infancy is so uncertain that it is impossible at that age to prognosticate as to what will happen. Sometimes growth is extremely rapid and occasionally respiratory difficulty ensues, a contingency that demands immediate aspiration of much of the contents of the cysts and possible tracheostomy. At other times, as a result of nasopharyngeal infection, the swelling becomes inflamed and spontaneous regression of the cysts may then occur.

Pathology.—The swelling consists of an aggregation of cysts like a mass of soap bubbles. The larger cysts are near the surface, viz.:——————————→ while the smaller ones lie deeply and tend to infiltrate muscle planes. Each cyst is filled with clear lymph and is lined by a single layer of endothelium having the appearance of mosaic.

Treatment.—Excision of all the cysts at an early age is the treatment of election. It is often helpful to give preliminary injections, at weekly intervals, of sclerosing solutions or even boiling water into the cysts. The swelling will then slowly reduce in size and the cyst walls become more fibrous. Dissection is thus facilitated.

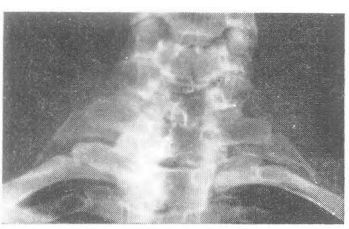


FIG. 621.—Cervical ribs that recurred after (subperiosteal) removal. Male aged forty-three years.

CERVICAL RIB AND THE SCALENE SYNDROME

By mass radiography it has been ascertained that a rib arising from the seventh cervical vertebra occurs in 0·46 per cent. of persons. In a little more than half of these the cervical rib is unilateral, and somewhat more frequent on the right side. It is paradoxical that a cervical rib or ribs found in the course of routine X-ray examination hardly ever gives rise to symptoms, whereas more often than not, when a radiograph of the cervical region is requested on account of nerve-pressure symptoms, no such rib is demonstrable.

Usually extra ribs spring from the seventh cervical vertebra (figs. 621 and 625) and maybe associated with spinal anomalies elsewhere.

Four main varieties of cervical rib are recognised:

(*a*) A complete rib, often containing a false joint in its length, articulates anteriorly with the manubrium or the first rib.——→

(*b*) The free end of rib expands into a large bony mass.——→

(*c*) A rib ending in a tapering point, which is connected by a fibrous band to the scalene tubercle of the first rib.

(*d*) A fibrous band closely applied to, or incorporated in, the scalenus medius alone is present. This not infrequent variety, of course, cannot be demonstrated radiologically.

At their exit from the neck, the branchial plexus and the subclavian artery pass through a narrow triangle (fig. 622). It is to the *base* of the triangle that attention must be focused.

Pathology.—Should the base of the triangle be raised the height of one vertebra by the interposition of a cervical rib, the subclavian artery and the first dorsal nerve are bound to be angulated, if not compressed, as they pass over the new floor, i.e. that formed by the cervical rib, instead of the first thoracic rib.

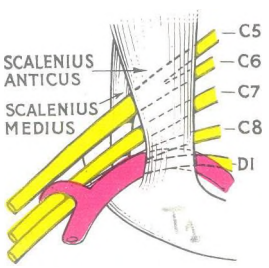


FIG. 622.—The anatomy of the parts concerned. (*After the late Professor Lambert Rogers, F.R.C.S., Cardiff.*)

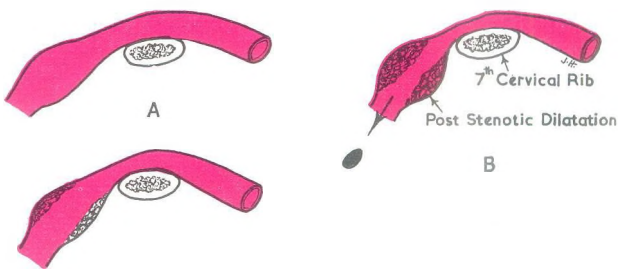


FIG. 623.—A, showing the narrowing and post-stenotic dilatation of the vessel, with early thrombus formation; B, an embolus has been detached.

Pathology of the Vascular Symptoms.—Owing to the angulation over the cervical rib, the lumen of the artery at this site becomes constricted. *Pari passu* with the formation of the constriction, fusiform dilatation of the first 2 to 4 cm. of the artery occurs distal to the constriction (fig. 623A). Within the post-stenotic dilatation (or possibly at the site of the constriction) clotting occurs on the intima. Portions of this mural thrombus may become detached, and give rise to an embolus (fig. 623B) or emboli. Also proximal extension of the thrombus can occur so that the vertebral artery may be involved and cerebrovascular embolic episodes occur.[1] (Shucksmith).

Three clinical types are encountered:

1. **Cervical Rib with Local Symptoms.**—From time to time a patient presents on account of a lump in the lower part of the neck which may be visible, or, more commonly, because of tenderness in the supraclavicular fossa. On palpation the lump is found to be bony hard and totally fixed. Type (b), (p. 600), is most likely to give rise to these signs.

2. **Cervical Rib with Vascular Symptoms.**—*Vascular symptoms* occur only when a cervical rib is complete.

Pain is the prevailing symptom. It is located in the forearm, but in some instances it radiates to the upper arm. What is so characteristic is that the pain is brought on by use of the arm: should the arm be in a raised position

[1] Described by Sir Charles Symonds in 1927.

Henry Samuel Shucksmith, Contemporary. Formerly Surgeon, General Infirmary, Leeds, England.

at the time of exercising it, the onset of the pain is accelerated. The pain is relieved by rest. Without doubt, this pain is ischæmic muscle pain, comparable to intermittent claudication in the leg.

Temperature and Colour Changes.—The hand on the affected side tends to (*a*) be colder than its fellow, (*b*) become unduly pale when held aloft and (*c*) become unduly blue when it is dependent for any length of time.

The Radial Pulse.—Sometimes it is as full as that of the other side; sometimes it is absent, and at others it is feeble, depending upon whether the collateral circulation is good, bad or indifferent. The distal part of the subclavian artery should be auscultated; a systolic bruit is significant.

FIG. 624.—Cervical rib with vasomotor symptoms, culminating in gangrene of the index finger. (*Telford and Stopford, Manchester.*)

Numbness of the fingers may be complained of, and ulceration or, more rarely, gangrene (fig. 624) may occur.

Treatment is timely extraperiosteal excision of the cervical rib, together with any bony prominence of the first rib. At the same time it is advisable to perform sympathetic denervation of the upper limb.

3. **Cervical Rib with Nerve-Pressure Symptoms.**—Nerve-pressure symptoms, due to angulation of the first dorsal nerve is of doubtful occurrence and most, if not all, cases previously described were due to cervical spondylosis (p. 496) or the carpal tunnel syndrome (p. 310). Only if these and other localised nerve lesions are excluded, should the scalene syndrome be considered as the cause of pain and tingling in the hand and forearm, whether wasting of the thenar and hypothenar muscles is present or not.

Differential Diagnosis.—Many of the nerve-pressure symptoms formerly attributed to a cervical rib can be, and are, produced by pressure on the cervical roots in the region of the intervertebral foramina by lateral protrusion of intervertebral discs. Secondly, paræsthesia and wasting of the thenar eminence are often due to carpal tunnel syndrome (p. 310). Thirdly, hypothenar wasting can also arise from angulation of the ulnar nerve behind the elbow (Griffiths). Other conditions to be considered include motor neurone disease and syringomyelia.

Treatment.—In mild cases the use of a sling and exercises aimed at strengthening the muscles of the shoulder girdle may alleviate the symptoms, at least temporarily. In about 70 per cent of cases even if a cervical rib cannot be recognised, the symptoms are relieved by dividing the scalenus anterior (scalenotomy) (fig. 625). Other surgeons remove the cervical rib or the corresponding band in addition, and in this way reduce the number of un-

David Lloyd Griffiths, Contemporary. Director, University Department of Orthopædic Surgery, Royal Infirmary, Manchester, England.

satisfactory results. When a cervical rib is excised, it is essential to remove it with its periosteum or it will probably regenerate (fig. 621). Particular care must be exercised to avoid damage to the brachial plexus and phrenic nerves.

INJURIES

Cut Throat.—In more than half the cases of cut throat that reach surgical aid the wound does not involve any vital structure—only the skin, platysma, and perhaps the sternomastoid or other muscles are severed.

Wounds above the Hyoid Bone.—The cavity of the mouth may have been entered. The epiglottis is often partially divided near its base. This should be repaired with catgut sutures. The mucosa of the pharynx is trimmed and united and the skin wound is closed.

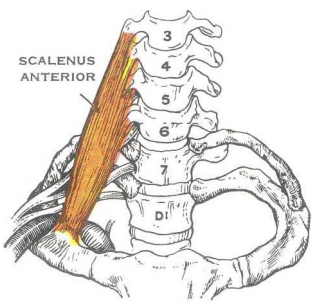


FIG. 625.—Showing the rational of scalenotomy.

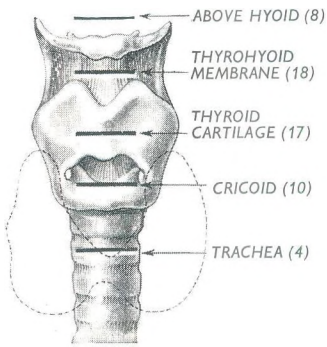


FIG. 626.—Position of the wound into the air passages of fifty-seven cases of suicidal cut throat with a deep wound.

Wounds of the Thyrohyoid Membrane.—Again the epiglottis is often damaged. The severed thyrohyoid membrane can usually be sutured. If there is respiratory distress, it is advisable to perform tracheostomy.

Wounds of the Thyroid or Cricoid Cartilage.—A tracheostomy well below the larynx is usually indicated. The laryngeal skeleton is then fully exposed and the damaged cartilages repositioned and sutured. An indwelling stent or ' keel ' is moulded to fit within the lumen and also shaped with a narrow waist to lie between the vocal cords. This stent is held by a wire or nylon retaining suture through the neck. In 2–3 weeks the stent is removed endoscopically after cutting the retaining suture. The tracheostomy is then closed.

Division of the Trachea.—Wounds of the trachea are comparatively rare. In order to obtain adequate exposure it is usually necessary to divide the thyroid isthmus between hæmostats. In most instances it is advisable to perform tracheostomy below the wound, and then to proceed to repair the latter with sutures.

Injury to Nerves.—It is remarkable how rarely important nerves are injured in self-inflicted wounds. In stab wounds any nerve may be involved (fig. 146). In one of our patients, a sailor, the most inaccessible nerve in the neck, the cervical sympathetic, was divided in this way, the assailant's weapon being a small penknife.

Complications of Cut Throat.—Hæmorrhagic shock can be severe and blood transfusion is necessary. Venous air embolism is a likely cause of death. Infection of the wound is common and, if cellulitis supervenes, it may spread downwards to cause mediastinitis. Pneumonia also is common. Rare complications include surgical emphysema due to the omission of a tracheostomy, and aerial fistula between the air passages and the exterior, which can be prevented or treated by the stent or ' keel ' operation described above. Oesophageal or pharyngeal fistula usually heals by itself. Aphonia or dysphonia may follow injury to the vocal cords or division of a

recurrent laryngeal nerve, while stenosis of the trachea or larynx due to scarring may necessitate permanent tracheostomy.

WOUNDS OF THE CERVICAL PORTION OF THE THORACIC DUCT

Wounds of the thoracic duct are rare, and usually occur during dissection of lymph nodes in the left supraclavicular fossa. When the accident is not recognised at the time, chyle pours from the wound—as much as 1 to 1·5 litre in twenty-four hours—and, as a result, the patient wastes rapidly.

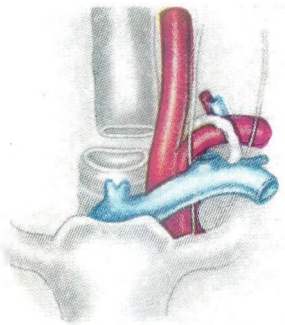


FIG. 627.—The termination of the thoracic duct.

Treatment.—Should the accident be recognised during an operation, the proximal end of the duct must be ligated with fine silk. Ligation of the duct is not harmful, for there are a number of anastomotic channels between the lymphatic and the venous systems in the neighbourhood. Usually the first intimation of a severed thoracic duct is a copious chylous discharge from the wound on the day following the operation. That the fluid *is* chyle is substantiated if it has a specific gravity of over 1·012 and if fat can be extracted from it with ether. Firm pressure by a pad and bandage should be applied, but this simple expedient may not be successful. More often the wound must be reopened. If the patient is given cream to drink an hour before the operation, more especially if the cream is coloured with confectioners' green dye (D-C 6), there is seldom any difficulty in locating a cut thoracic duct, which is about the size of a straw and an immediate external relation of the last 3·75 cm. of the left internal jugular vein (fig. 627). If the duct is found, it should be ligated, but in any case the wound should be packed firmly and allowed to heal by granulation. Thanks to subsidiary anastomotic channels, these measures are regularly satisfactory.

INFLAMMATORY CONDITIONS

Acute cellulitis is either superficial or deep to the deep cervical fascia.

Superficial cellulitis is common, and methods of treating it follow that of cellulitis elsewhere. When, however, it occurs above the level of the hyoid bone it is especially dangerous, because sudden asphyxia from œdema of the glottis is an ever-present possibility.

Deep cellulitis in the lower third of the neck, on the other hand, is free from this danger. Consequently, it can be treated by antibiotic therapy with every confidence, and should an abscess develop, it is opened.

Ludwig's Angina.—Ludwig described a clinical entity characterised by a brawny swelling of the submandibular region combined with inflammatory œdema of the mouth. It is the *combined* cervical and intrabuccal signs that constitute the characteristic feature of the lesion (fig. 628). The cause is a virulent (usually streptococcal) infection of the cellular tissues surrounding the submandibular salivary gland. It may be a complication of advanced carcinoma of the floor of the mouth.

Clinical Course.—Unless the infection is controlled, these cases may rapidly assume a grave aspect. The swollen tongue is pushed towards the palate and forwards through the open mouth, while the cellulitis extends down the neck in that most dangerous plane—deep to the deep fascia.

FIG. 628.—Ludwig's angina. The brawny swelling beneath the mandible and the œdema of the floor of the mouth are characteristic.

Wilhelm von Ludwig, 1790–1865. Professor of Surgery and Midwifery, Tübingen, Germany.

Ludwig's angina is an infection of a closed fascial space, and, untreated, the inflammatory exudate often passes via the tunnel occupied by the stylohyoid to the submucosa of the glottis, in which event the patient is in imminent danger of death from œdema of the glottis (p. 703).

Treatment.—When the condition is diagnosed early, the results of antibiotic therapy are sometimes dramatic. In cases where the swelling, both cervical and intrabuccal, does not subside rapidly with such treatment, a curved incision beneath the jaw, as is shown in fig. 629, is made. The incision is deepened, and after displacing the superficial lobe of the submandibular salivary gland, the mylohyoid muscles are divided. This decompresses the closed fascial space referred to. The wound is lightly sutured and drained. The operation can be conducted with the greatest safety under local anæsthesia. Thiopentone anæsthesia usually precipitates laryngeal spasm and asphyxia.

Infection of the Pharyngo-maxillary Space (*Parapharyngeal Abscess*). — The pharyngo-maxillary space is a potential cone-shaped space, base uppermost. The base is formed by the base of the skull; the apex abuts the great cornu of the hyoid bone; the medial wall consists of the superior constrictor muscle; the lateral wall, from above downwards, is composed of the fascia covering the internal pterygoid muscle, the mandible about its angle, and the submandibular salivary gland, below which the apex of the space becomes relatively superficial. Usually infection of this space originates in the tonsil, and may occur after tonsillectomy, especially when the operation has been performed under local anæsthesia.

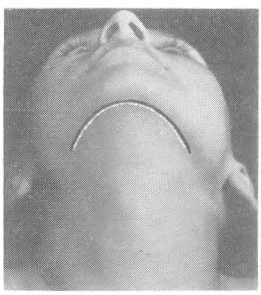


FIG. 629.—Incision for decompressing thoroughly the space beneath the mylohyoid muscle.

Clinical Features.—Every posterior, peritonsillar abscess is a potential pharyngomaxillary space infection, the general reaction of which is greater than that accompanying peritonsillar abscess. There is often *slight* trismus, and swelling over the lower part of the parotid gland; this is never present in quinsy. Tenderness, and later swelling below the angle of the mandible, appears when the apex of the space is involved. As the carotid sheath runs through the space, the dreaded complications of thrombophlebitis of the internal jugular vein and/or erosion of an artery, usually the internal carotid, may occur if the space is not adequately drained. Sometimes the abscess bursts spontaneously between the cartilaginous plates of the external auditory canal, but obviously such an eventuality is a fortunate escape from death, for which the patient's medical advisers can take no credit.

Treatment.—As soon as the diagnosis is strongly suspected an incision should be made below and behind the angle of the mandible, on a line towards the hyoid bone. A finger is passed upwards, medial to the mandible and the distended space is entered by rupturing its wall. The space is drained with a large soft wick drain.

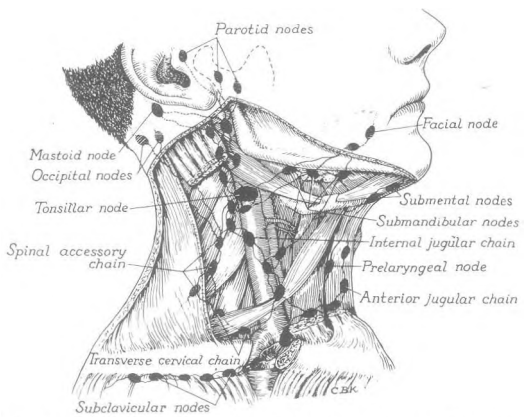


FIG. 630.—The lymphatics of the facio-cervical region. (*After Murry M. Copeland.*)

CERVICAL LYMPHADENITIS

There are approximately 800 lymph nodes in the body; no fewer than 300 of them lie in the neck (fig. 630). Inflammation of the lymph nodes of the neck is exceedingly common. Infection occurs from the oral and nasal cavities, the ear, the scalp, and face. The source of infection must be sought for systematically.

Acute Lymphadenitis.—The affected lymph nodes are enlarged and tender, and there is a varying degree of pyrexia. The treatment, in the first instance, is directed to the general condition and to the focus of infection, the neck itself being simply protected by a bandage over wool. If, in spite of antibiotic therapy, pain continues or certain lymph nodes appear to be getting larger, fomentations are applied locally. Abscess formation calls for adequate drainage.

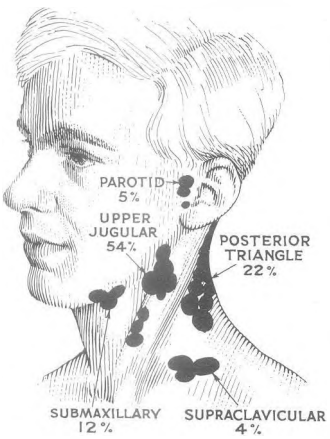


FIG. 631.—Groups of cervical lymph nodes infected by tuberculosis, founded on 372 consecutive cases. The 3 per cent. not labelled are divided between the mid-jugular group (shown) and the submental group (not shown).

Chronic Lymphadenitis.—In the early stages it is extremely difficult to distinguish tuberculous adenitis from chronic non-tuberculous adenitis, but clinical experience shows that chronically inflamed lymph nodes which do not resolve in the space of three or four weeks are nearly always tuberculous.

Tuberculous adenitis (p. 189).

The majority of patients affected are children or young adults, but the condition can occur for the first time at any age. Usually one group of cervical nodes is first infected (fig. 631), most frequently those of the upper jugular chain. More rarely there is widespread cervical lymphadenitis, and in these cases especially, periadenitis or matting of the lymph nodes is evident.

Source of Infection.—In the majority of instances tubercle bacilli gain entrance through the tonsil of the corresponding side. The nodes of the posterior triangle are infected in 22% of cases, probably stemming from adenoidal infection.

Contrary to what is believed, it is the human,[1] and not the bovine, bacillus that is responsible for tuberculous cervical adenitis in about 70 per cent. of cases. In fully 80 per cent. of cases the tuberculous process is virtually limited to the clinically affected group of lymph nodes; nevertheless, a primary focus in the lungs must be suspected. Renal tuberculosis may co-exist and the urine should be examined for this organism.

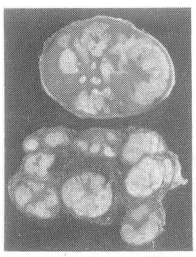


FIG. 632.—Caseating tuberculous lymph nodes.

In the event of the patient developing a natural resistance to the infection or (more often) as a result of appropriate general treatment, fibrosis or calcification may occur. In other circumstances the caseating material (fig. 632) liquefies, breaks through the capsules of the lymph nodes, and a 'cold abscess' forms. The pus is at first confined by the *deep* cervical fascia. In a few weeks this dense sheet

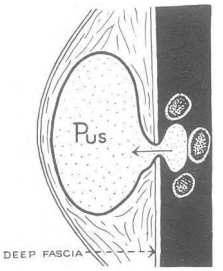


FIG. 633.—Showing the abscess and the source of the pus beneath the deep fascia.

[1] The human bacillus is responsible for 100 per cent. of cases among the Bantu-speaking urban population of South Africa (Keen).

Paul Keen, Contemporary. Formerly Senior Surgeon, Non-European Hospital, Johannesburg, South Africa.

becomes eroded at one point, and the pus flows through the small opening into that more commodious space beneath the *superficial* fascia. The process has now reached the well-known stage of collar-stud abscess (fig. 633 and p. 189). The superficial abscess enlarges steadily, and unless suitable treatment is adopted, the skin will soon become reddened over the centre of the fluctuating swelling, and before long a discharging sinus, with its attendant evils, is at hand.[1]

DIFFERENTIAL DIAGNOSIS

When the swelling is solid, from
- Chronic non-tuberculous lymphadenitis
- Hodgkin's disease (p. 191).
- Reticulo-sarcoma.
- Secondary malignant disease.

When the swelling is cystic, from
- Branchial cyst.
- Extension of an abscess connected with a tuberculous cervical vertebra.

When a sinus or sinuses have formed, from
- Actinomycosis.
- An acquired branchial fistula.

TREATMENT OF TUBERCULOUS LYMPHADENITIS

The patient should be treated by general measures and appropriate chemotherapy (p. 37). If abscess is present it should be aspirated and an attempt should be made to grow the tubercle bacillus and find out its sensitivity to the anti-tuberculous drugs (p. 37). Repeated aspiration of a collar-stud abscess cannot be recommended because this predisposes to sinus formation and secondary infection.

Operation for Collar-stud Abscess.—After an incision in line with the skin creases, the pus in the superficial compartment is mopped away, and the hole in the deep fascia is enlarged to admit a small curette, so that the caseating lymph node can be scraped out and the cavity packed with iodoform gauze. This is brought out through the edge of the wound which is closed by careful primary suture. It is removed after 24 hours, but the sutures are left for 10 days.

Excision of Lymph Nodes.—This becomes necessary when there is no local response to chemotherapy and if a sinus persists. When there is active tuberculosis of another system, e.g. pulmonary tuberculosis, removal of tuberculous lymph nodes in the neck is, of course, illogical.

An oblique incision (fig. 619) conforming with the skin creases, gives satisfactory access. If the nodes are related to the internal jugular vein this structure should be identified first and then the nodes can be dissected away from it.

To minimise unnecessary injury to large veins, no tissue should be divided when stretched taut. Should the internal jugular vein prove to be involved to such an extent that freeing it is difficult or impossible, this great vessel can be ligated, or a portion of it can be resected, without any untoward effect at any age (cf. block dissection of the neck, p. 610).

There is no contraindication to dividing the sternomastoid muscle if, as is frequently the case, such a step facilitates access to the diseased nodes and enables the operator to visualise the spinal accessory nerve more easily. The divided muscle is subsequently reunited.

[1] For 600 years the King's touch was believed to cure this prevalent disease. Charles II touched on an average 10,000 sufferers a year. In addition, he presented each with half a sovereign. The condition was known as the King's Evil.

During the dissection of cervical lymph nodes every effort must be made to preserve: the spinal accessory nerve; the mandibular branch of the facial nerve; the hypoglossal nerve; which are the nerves most likely to be injured.

Actinomycosis of the Neck (See p. 39).

PRIMARY MALIGNANT TUMOURS OF THE NECK

Carotid Body Tumour ('Potato' Tumour).—The carotid body, which is situated at the bifurcation of the carotid artery, is the most important moiety of the chemoreceptor system (fig. 634). The cells of this system are sensitive to changes in the pH and temperature of the blood. Hence a tumour is called a chemodectoma, though it is classified histologically as a non-chromaffin paraganglioma. It will remain localised for years but regional metastases will occur in about 20% of cases and distant metastases less frequently.

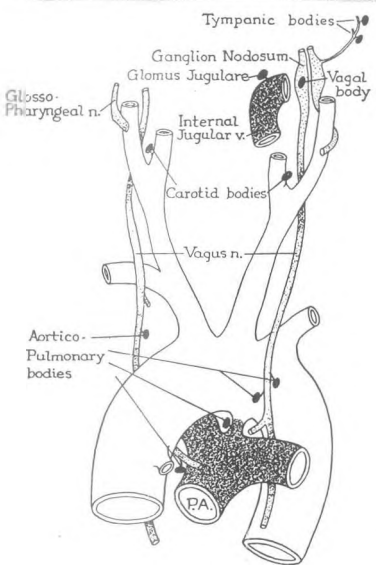


FIG. 634.—Sites where chemodectomas are known to occur. In addition, they have been found in the orbit, along the course of the inferior alveolar artery, and in proximity to the femoral artery. (*After S. O. Burman.*)

Clinical Features.—Usually unilateral, it becomes apparent in middle life, but occasionally earlier. The diagnosis is suggested by a long history and a lump at the carotid bifurcation which moves from side to side but not vertically, and usually a pulsating vessel overlies its outer surface (Westbury).

Arteriography is valuable and shows the carotid fork to be splayed and a 'blush' outlining the abnormal tumour vessels.

The Special Danger of Excision of a Carotid Body Tumour is due to its vascularity and the way it is blended with the carotid bifurcation. Therefore:—

1. There is considerable danger of torrential hæmorrhage occurring if a biopsy is attempted in the erroneous belief that the lump is an infected or neoplastic lymph node. Control of this bleeding by artery forceps inevitably results in occlusion of the carotid artery with hemiplegia or death occurring in at least 33% of cases.

2. If extirpation is to be attempted it is essential to have ready a length of silicone bypass tubing in case carotid occlusion becomes necessary, otherwise a similar tragedy is likely.

In some cases it is possible to dissect the tumour away from the fork of the carotid (fig. 635). But when the tumour is large and inseparable from the vessels resection will be necessary (fig. 636) and a bypass is essential while a vein autograft is being inserted to restore arterial continuity.

Recurrence after complete removal is unusual.

Gerald Westbury, Contemporary. Surgeon, Westminster Hospital, London.

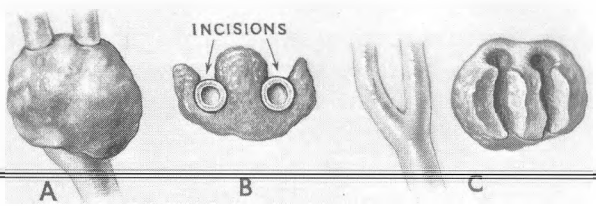


FIG. 635.—The tumour is split in the manner shown, so that it can be dissected from the arteries it envelops.
(After T. Farrar et al.)

In the old and enfeebled it may be decided that it is best to let Nature take its course. Hardly any of the tumours are radiosensitive.

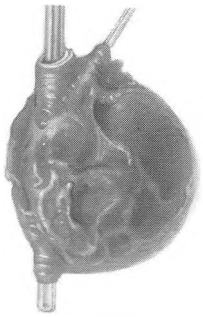


FIG. 636.—Carotid body tumour removed successfully.

Hodgkin's Disease (syn. Lymphadenoma in neck), see p. 191.

Reticulosarcoma (including Lymphosarcoma in neck) see p. 190.

SECONDARY CARCINOMA OF THE NECK

Secondary carcinomatous infiltration of the cervical lymph nodes is only too common. When a patient presents with enlargement of cervical lymph nodes that are suspiciously indurated, search for a primary growth is imperative and must be made *before* a node is removed for biopsy. Often the primary growth lies within the buccal cavity; when this is not the case, the search must continue. Among the sites that are prone to be overlooked are the nasopharynx, pharynx, larynx and cervical œsophagus, the paranasal sinuses, external auditory meatus, the bronchi, the breast, the stomach, and the testes.

Management.—In so far as cervical lymph nodes are concerned, patients with oral, laryngeal, or facial carcinoma can be categorised as follows:

Stage 1.—*Those without clinical evidence of metastases.* These patients are kept under careful (bi-monthly) observation, dissection of the neck being withheld unless the nodes become palpable. Exceptions to this rule are (1) melanoma, (2) papilliferous carcinoma of the thyroid, and (3) possibly cases of carcinoma of the tongue (see p. 578) and floor of mouth.

Stage 2.—*Those with palpable cervical lymph nodes where the primary growth is operable,* or can be destroyed with radium. It should be noted carefully that lymph node enlargement, especially when the primary growth is foul, is not necessarily carcinomatous. After removal of the primary neoplasm a period of non-intervention and antibiotic therapy will determine whether or not the enlargement subsides. In the latter event block dissection of the neck is indicated, provided, of course, there is no clinical or X-ray evidence of distant metastases, and the involved cervical nodes have not progressed to a stage where it seems unlikely that they can be encompassed. Adherence to the mandible, or to the larynx, or to the skin, should not be regarded as constituting inoperability, for each of these structures can be sacrificed, provided

the patient's general condition is such as to be able to withstand either of these procedures, which are extremely formidable.

Stage 3.—*Those in whom the primary growth is inoperable* or otherwise incurable, and/or the cervical metastases are fixed to deeper structures, and/or distant metastases are present. Palliative deep X-ray treatment is sometimes indicated, or the malignant swelling can be treated by insertion of radioactive gold seeds. The latter course gives minimum discomfort to the patient and, although only palliative, can effectively reduce the size of the tumour and relieve pain. There is always the risk that high dosage of irradiation will cause necrosis of the mandible or the laryngeal cartilages.

Block Dissections of the Neck

1. **Crile's block dissection** is conducted through the wide display afforded by specially placed skin incisions (fig. 637).

The skin flaps having been dissected up, the sternomastoid is divided about 2·5 cm above the clavicle. The muscle is freed and retracted upwards. Next, the internal jugular vein is divided between ligatures low down in the neck. The dissection proceeds upwards methodically and the muscle, fascia, fat, lymph nodes, the internal jugular vein, together with the submaxillary salivary gland, are dissected and removed *en bloc*. Attention must be directed to clearing the space between the parotid and the great vessels, and also the submental triangle between the hyoglossi,

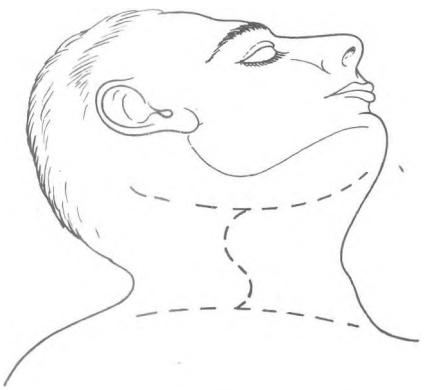


FIG. 637.—An incision for block dissection of the neck.

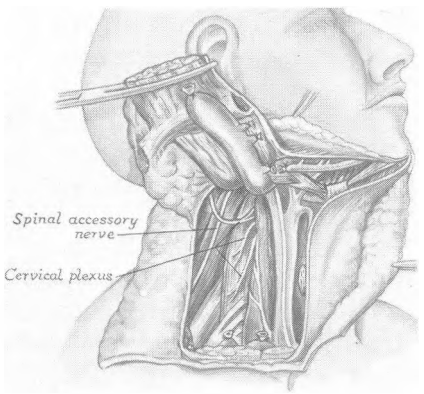


FIG. 638.—Crile's block dissection of the neck nearing completion. (*After W. W. Carroll.*)

for it is in these areas that a lymph node can easily be overlooked. Bleeding vessels are ligated as they occur; finally the upper end of the internal jugular vein is ligated by transfixion, and divided. When the dissection has been completed, the carotid artery is laid bare, and lying with it is the vagus nerve, which has been carefully preserved. The operation aims at removing the whole of the lymphatic-bearing tissues on the affected side of the neck (fig. 638). The skin flaps are approximated and the wound drained. Surprisingly little deformity follows this extensive dissection, but the neck is stiff and there is drooping of the corner of the mouth. (The cervical branch of the facial nerve is severed.) When *bilateral* block dissection is required it must be undertaken, not simultaneously, but consecutively with an interval of about three weeks. Removal of both internal jugular veins can be attended by obstructed cerebral circulation (p. 579).

2. **Suprahyoid block dissection** of the neck is indicated in cases of carcinoma of the lower lip, early cases of carcinoma of the tip of the tongue, and carcinoma of the floor of the mouth. Its advantage is that both sides of the neck can be attended to at

George Washington Crile, 1864–1943. Professor of Surgery, Western Reserve University, and one of the Founders of the Cleveland Clinic, Cleveland, Ohio, U.S.A.

one operation. Sometimes a unilateral suprahyoid block dissection is indicated combined with a Crile's block dissection of the opposite side.

During these operations it is advisable to transfuse an amount of blood proportional to the estimated loss. Usually tracheostomy is required only when a monobloc (commando, p. 579) operation is to be performed; in other circumstances tracheostomy should be avoided when possible, because it heightens the incidence of wound infection. Should it be necessary to keep the bronchial tree free from accumulating secretions during the post-operative period, this contraindication must be over-ridden.

Post-operative Treatment.—Suction-drainage of the wound for two or three days reduces to a minimum the possibility of accumulated blood and blood-clot pressing on the larynx and trachea. Blood transfusion is continued for as long as necessary, and antibiotics are given routinely.

Mortality and Prognosis.—The mortality of cervical-block dissection is, with modern anæsthesia which has done so much to facilitate it, about 3 per cent. The over-all five-year survival rate is 35 per cent.

CHAPTER 35

THE THYROID GLAND AND THE THYROGLOSSAL TRACT

SURGICAL ANATOMY AND PHYSIOLOGY

Embryology.—The thyroid gland is developed from the median bud of the pharynx (the thyroglossal duct) which passes from the foramen cæcum at the base of the tongue to the isthmus of the thyroid. The ultimo-branchial body which arises from a diverticulum of the fourth pharyngeal pouch of each side amalgamates with the corresponding lateral lobe, and parafollicular cells (C-cells) are derived from this bud. It is doubtful whether the branchial apparatus contributes to the thyroid follicular cells.

Surgical Anatomy.—The normal gland weighs 20–25 g. The functioning unit is the lobule supplied by a single arteriole and consisting of 20–40 follicles which are lined by cubical epithelium. The resting follicle contains colloid in which thyroglobulin is

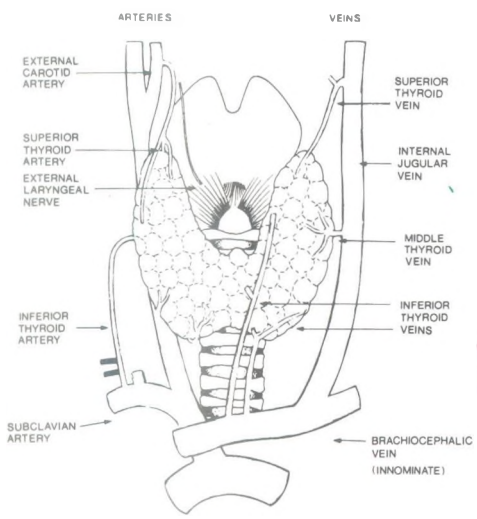


FIG. 639.—The thyroid gland from the front.

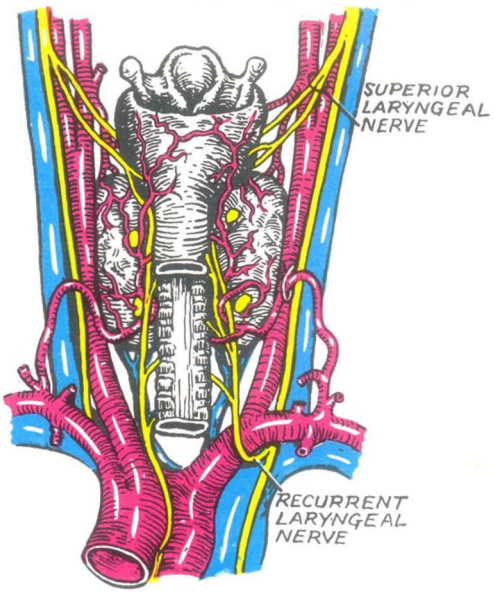


FIG. 640.—The thyroid gland from behind, showing the laryngeal nerves and the four parathyroids. *(After Martin Norland.)*

stored. The arterial supply is rich, and extensive anastomoses occur between the main thyroid arteries and branches of tracheal and œsophageal arteries (figs. 639 and 640). There is an extensive lymphatic network within the gland. Although some lymph channels pass directly to the deep cervical nodes, the subcapsular plexus drains principally to the juxtathyroid nodes—i.e. pretracheal (Delphic)[1] and paratracheal

[1] Pythia, the snake-woman oracle of Delphi, sat on her tripod, clutching the ribbons of the monolithic ' omphalos ' of the world. She inhaled sulphurous fumes and laurel, and uttered a meaningless jargon which was interpreted equivocally by the attendant priests for those who came to consult her. Formerly, the meaning of these lymph nodes was uncertain and they were therefore called ' Delphic '.

nodes, and nodes on the superior and inferior thyroid veins, and thence to the deep cervical and mediastinal group of nodes.

Physiology.—The hormones tri-iodo-thyronine (T^3) and thyroxine (T^4)[1, 2] are bound to thyroglobulin within the colloid. Synthesis within the thyroglobulin complex is controlled by several enzymes, in distinct steps: (1) Trapping of inorganic iodide from the blood. (2) Oxidation of iodide to iodine, and binding of iodine with tyrosine to form iodotyrosines. (3) Coupling of mono-iodotyrosines and di-iodotyrosines to form T^3 and T^4. (4) When hormones are required thyroglobulin is broken down; T^3 and T^4 are liberated and enter the blood where they are bound to serum proteins. A small amount of hormone remains free in the serum in equilibrium with the protein bound hormone and is biologically active. T^3 is quick acting (within a few hours) whilst T^4 acts more slowly (4–14 days). T^3 is the more important physiological hormone.

Thyrocalcitonin (see calcitonin, p. 646).

The Pituitary Thyroid Axis (fig. 641)

Synthesis and liberation of thyroid hormones from the thyroid is controlled by thyroid stimulating hormone (T.S.H.) from the anterior pituitary. Secretion of T.S.H. depends upon the level of circulating thyroid hormones. In hyperthyroidism, where hormone levels in the blood are high, T.S.H. production is suppressed, whilst in hypothyroidism it is stimulated.

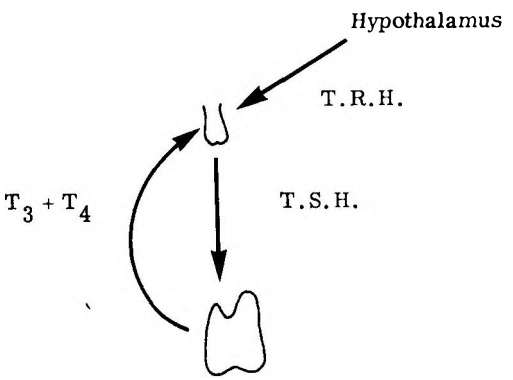


FIG. 641.—The pituitary-thyroid feedback mechanism. Thyrotrophin releasing hormone (T.R.H.) from the hypothalamus, stimulates secretion of T.S.H. by the anterior pituitary, but its precise role in the feedback mechanism is, as yet, uncertain.

Long-acting Thyroid Stimulator (L.A.T.S.)

This is an IgG immunoglobulin which acts as an antibody to certain particulate thyroid antigens. Its action on thyroid cells is similar to T.S.H. but the duration of the stimulus is more prolonged and it is estimated to produce its maximum effect after 16–24 hours as compared with 1·5 to 3 hours for T.S.H. It is measured by a mouse bioassay system and can be detected in the sera of many hyperthyroid patients (figures vary from 14% to 76%). At least one other abnormal thyroid stimulator has been identified which is specific for human thyroid tissue and hence not detected by the previously used mouse bioassay. This is L.A.T.S.—protector—, also called Human Specific Thyroid Stimulator. Other abnormal stimulators probably exist.

These Thyroid Stimulating Immunoglobulins—as they have been called (T.S.I.)—are now thought to be responsible for all cases of hyperthyroidism not due to autonomous toxic nodules.

TESTS OF THYROID FUNCTION

The principal value of these tests is in assessing the thyroid status when the clinical picture is equivocal. However, even if the clinical diagnosis appears quite certain, it is valuable to have confirmatory evidence to look back upon should doubt as to the diagnosis arise at a later date. Several parameters[3] should be obtained because no single test is conclusive.

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[1] Extracted by E. C. Kendall in 1916.

[2] Therapeutic Notes—L-thyroxine (T^4) is the official name; trade name Eltroxin; tablet size 0·1 mg. and 0·05 mg. Tri-iodothyronine (T^3), official name lio-thyronine, trade names, Cynomel, Tertroxin. Tablet size 20 μg.

[3] A parameter is any yard-stick or independent variable.

Edward Calvin Kendall, 1886–1972. Professor of Physiological Chemistry, Mayo Clinic, Rochester, Minnesota, U.S.A.

1. Measurement of Thyroid Hormones in the Serum

(*a*) Total Serum Thyroxin (T^4).
(*b*) Total Tri-iodo-thyronine (T^3).
(*c*) Free Serum Thyroxine (T^4).

The iodine containing hormones T^3 and T^4 are transported in the plasma mainly by specific binding proteins, Thyroxine binding globulin (T.B.G.), and Thyroxine binding prealbumin. As only a small amount of T^3 and T^4 are free in the blood, estimation of the serum bound protein iodine (P.B.I.) was used for many years as representing total circulating thyroid hormones. However, false low estimations of P.B.I. are due to lowering of T.B.G. by hereditary factors, androgens or the nephrotic syndrome, and false high results are due to iodine contamination, e.g. X-ray contrast media and expectorants containing potassium iodide, and also when thyroid binding proteins in the serum are increased in pregnancy and when taking oral contraceptives.

The Total Serum T^4 (Euthyroid[1] range 55–150 nmol/l) is not subject to these inaccuracies and, now that the test is freely available, it has replaced the serum P.B.I. Estimations of Total Serum T^3 (Euthyroid range 1·3–3·5 nmol/l) is also freely available and is essential if T^3 toxicosis is suspected. The Free Serum Thyroxin—i.e. the T^4 that is free in the serum and not protein bound—is in theory more representative of the level of hormones available to the individual body cells, but estimation is difficult and the test is not generally available.

2. Measurement of Free (unoccupied) binding sites for thyroid hormones in the blood.

In the T^3 uptake test radio-active T^3 is incubated with the patient's serum so that it becomes fixed to any thyroid binding protein not already carrying T^3 or T^4. The amount so fixed can be measured and from this can be estimated the number of binding sites in the serum which are unoccupied. In hyperthyroidism the number of free binding sites is low because few are not already carrying hormone. In hypothyroidism and myxœdema, however, the number of free sites is high. Using the Thyopac method and taking 100 per cent as the mean normal value for free binding sites, a figure of 85 per cent or less suggests hyperthyroidism and a figure of 120 per cent or more suggests hypothyroidism. This is not a very accurate test in itself, but in conjunction with Total Serum T^4 the Free Thyroxin Index (F.T.^{4}I.) can be calculated from the formula F.T.^{4}I. = Serum T^4 × 100/T^3 uptake per cent. Similarly, the Free Tri-iodo-thyronine Index (F.T.^{3}I.) can be calculated. The euthyroid range of the F.T.^{4}I.) is 55–145: and that of the F.T.^{3}I. is 1·4–3·7. These indices give close approximations to Free Serum T^4 and Free Serum T^3 and are the best parameters of thyroid hormone levels generally available at present.

3. Measurement of Serum T.S.H.

The estimation of the level of Serum T.S.H. is now generally available: the normal range is up to 5 m units/l. Levels over 40 m units/l are present in gross thyroid insufficiency. The test is invaluable in the early detection of the mild (but disabling) degrees of hypothyroidism so frequently seen after the treatment of thyrotoxicosis by surgery or by radio-iodine.

4. The T.R.H. test.

Thyrotrophic Releasing Hormone (T.R.H.) which arises probably from the hypothalamus stimulates T.S.H. secretion by the Anterior Pituitary. When Thyroid Hormone levels are high—as in Hyperthyroidism—T.S.H. secretion is suppressed and the intravenous injection of T.R.H. does not result in a rise in serum T.S.H. When Thyroid hormone levels are normal or low, injection of T.R.H. does increase the Serum T.S.H.

Serum T.S.H. is estimated at the beginning of the test and again 20 minutes and 60 minutes after the injection of 200 μg of T.R.H. In thyrotoxicosis and when a full suppressive dose of exogenous T^3 or T^4 is being given, Serum T.S.H. levels remain at below 2·5 m units/l throughout the test[2]. In the euthyroid and the hypothyroid patient the 20 minute and 60 minute T.S.H. levels rise above the basal level.

[1] Euthyroid—Euthyroidism = the state of normal thyroid function.
[2] Estimations of T.S.H. levels below 2·5 m units/l are inaccurate. A zero level is, therefore, recorded as 2·5 m units/l.

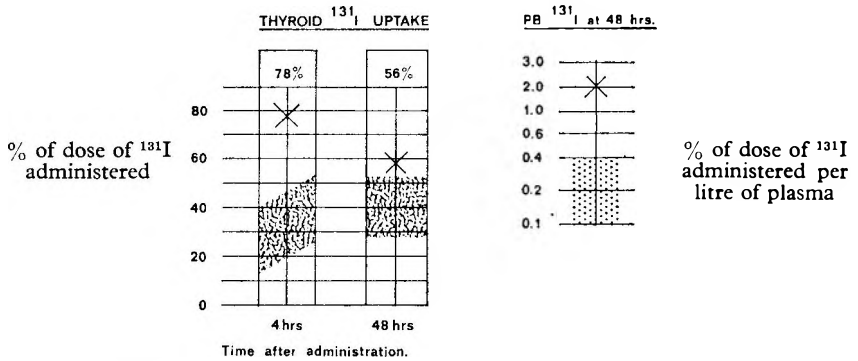


FIG. 642.—Radio-iodine uptake test. Thyroid uptake at 4 hours and 48 hours after administration of ^{131}I and serum protein bound ^{131}I at 48 hours in a typical case of diffuse toxic goitre. Stippled areas represent the euthyroid range.

This test is generally available because T.S.H. estimations are now freely available, but the test is time consuming and only occasionally gives information not obtained from routine tests of thyroid function.

5. **In vivo tests of uptake and discharge of ^{131}I by the thyroid gland.**

(*a*) *Radioactive iodine uptake test* (fig. 642). The thyroid treats radio-active isotopes of iodine exactly as it treats inorganic iodine (^{127}I). When a tracer dose of ^{131}I is given (5 microcuries[1] is the usual dose) it is rapidly absorbed from the small bowel into the blood, and the thyroid and kidneys compete for it. In hyperthyroidism the thyroid uptake is rapid and little is excreted in the urine. Ultimately the isotope—now incorporated into the T^3 and T^4 molecules—passes back into the serum and can be measured as protein bound ^{131}I (P.B.^{131}I.). A rapid uptake of ^{131}I (high counting rate over the thyroid at four hours) and a rapid turnover (high serum P.B.^{131}I. at 24 or 48 hours) are typical of hyperthyroidism. This can, however, occur when the mass of functioning thyroid tissue has been reduced so that radio-iodine studies after surgery or radio-iodine therapy must be interpreted with caution. ^{132}I may also be used as a diagnostic tracer but only for thyrotoxicosis as it has a short life (213 hours as opposed to the 8 days of ^{131}I). Because the radio-active dose is low it can be used on children, for serial tests and in pregnancy.

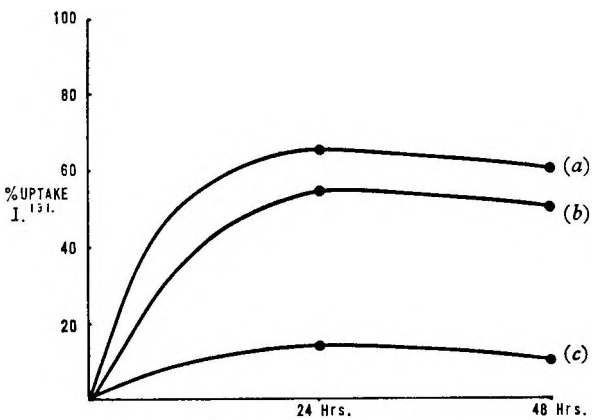


FIG. 643.—T^3 suppression test

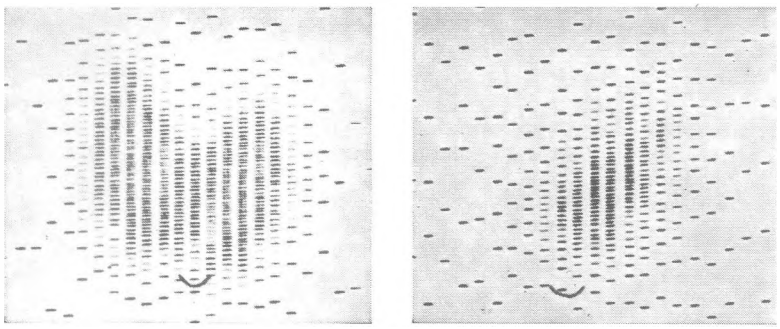
(*a*) hyperthyroid or normal—represents a doubtful hyperthyroid uptake curve.

(*b*) non-suppressed—would confirm hyperthyroidism.

(*c*) suppressed—would confirm a euthyroid state.

[1] Microcurie—a millionth part of a Curie (abbreviation μCi). Millicurie—a thousandth part of a Curie (abbreviation mCi).

Pierre Curie, 1859–1906, and Marie Sklodowska Curie, 1867–1934. Co-discoverers of radium. Madame Curie succeeded her husband as Professor of Physics at the Sorbonne, Paris.



(*a*) (*b*)

FIG. 644.—(*a*) Thyroid scan of a normal gland. The position of the sternal notch is indicated. (*b*) Thyroid scan showing autonomous 'hot' thyrotoxic nodule in the left lobe. (*Dr. A. W. G. Goolden, London.*)

(*b*) *T^3 Suppression Test (Werner)* (fig. 643). An iodine avid goitre, e.g. due to iodine deficiency in endemic areas, has a rapid ^{131}I uptake and this may simulate a hyperthyroid uptake. However, a simple goitre, as opposed to a toxic goitre, is under T.S.H. control, so that the uptake of ^{131}I can be diminished by suppressing T.S.H. production. This is done by giving 40 μg of tri-iodo-thyronine (T^3) 8 hourly for 7 days. ^{131}I uptake curves are compared before and after T^3 suppression, when a toxic goitre may show up to 10–20 per cent reduction in uptake, whilst a simple goitre will show 50–80 per cent reduction.[1] This test tells the clinician whether or not the thyroid tissue is under T.S.H. control. It is used, (1) In the diagnosis of thyrotoxicosis in difficult cases, (2) In the assessment of ophthalmic Graves' disease—where the patient may be euthyroid but the thyroid tissue is under T.S.I. control, (3) In checking the response to the treatment of thyrotoxicosis by antithyroid drugs.

(*c*) *Thyroid Scanning* (fig. 644). Scanning of the thyroid after a tracer dose of ^{131}I shows which parts of the gland are functioning or functionless (hot or cold).[2] Whilst scanning is sometimes helpful in cases of thyroid carcinoma, its principle value is in the diagnosis of an autonomous toxic nodule—either as a solitary toxic nodule or as part of a toxic multinodular goitre.

Whole body scanning can also be used to demonstrate metastases but the patient must have no normal functioning thyroid tissue when the isotope is given because thyroid cancer can only very rarely compete with normal thyroid tissue in the uptake of iodine.

6. Miscellaneous Tests

These comprise the B.M.R., E.C.G., serum cholesterol and measurement of tendon reflexes. Some clinicians use the ankle reflex as a measurement of hypothyroidism but otherwise these tests are of little value today.

Routine Tests of Thyroid Function

These will vary in individual laboratories. The following tests are suggested.

1. Thyroid Profile. This comprises estimations of Total Serum T^4 and Total Serum T^3: and the Thyopac test for unoccupied binding sites. From these tests the F.T.^{4}I. and F.T.^{3}I. are calculated.

2. ^{131}I uptake test with uptake at 4 hours and 48 hours together with P.B.I.^{131} at 48 hours.

3. Serum T.S.H. estimation is automatically done if the serum T^4 is below 60 nmol/l.

4. T.R.H.[3] test if the thyroid status is still doubtful.

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[1] Failure to suppress is strong but not conclusive evidence of hyperthyroidism. Suppression rules out hyperthyroidism absolutely.

[2] Technitium ($^{99}Tc^m$), taken up exactly like ^{131}I is also used for scanning.

[3] T.R.H. = Thyrotrophic releasing hormone secreted by the Hypothalamus.

Sidney Charles Werner, Contemporary. Professor of Clinical Medicine, Columbia University College of Physicians and Surgeons, New York, U.S.A.

HYPOTHYROIDISM

CLASSIFICATION OF HYPOTHYROIDISM

1. **Failure of Thyroid Development**—(*a*) Complete (Sporadic Cretinism), (*b*) Partial.
2. **Endemic Cretinism** (often goitrous).
3. **Iatrogenic**—(*a*) After thyroidectomy, (*b*) After radio-iodine therapy, (*c*) After pituitary ablation, (*d*) Drug induced.[1]
4. **Auto-immune thyroiditis**—(*a*) Non-goitrous (Primary myxœdema), (*b*) Goitrous (Hashimoto goitre).
5. **Dyshormonogenesis** (p. 618).
6. **Goitrogens** (p. 618).
7. **Vascular damage to anterior pituitary.**

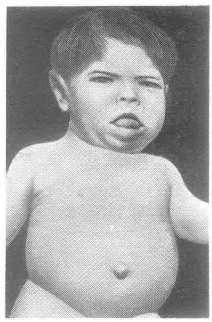


FIG. 645.—An infant cretin. (*The late Professor de Quervain, Berne.*)

Cretinism (Fœtal or Infantile Hypothyroidism). Sporadic cretinism is due to complete or near complete failure of thyroid development (partial failure causes juvenile myxœdema): the parents and other children may be perfectly normal. In endemic areas goitrous cretinism is common, and is due to maternal and fœtal iodine deficiency. Fig. 645 depicts the infant cretin, potbellied with umbilical hernia, protruding tongue and pale puffy face. In adolescence and adult life (fig. 646) the cretin is mentally and physically retarded with dry wrinkled skin and supraclavicular pads of fat. Immediate diagnosis and treatment with thyroxine within a few days of birth are essential if physical and mental development are to be normal, or if further deterioration is to be prevented when damage has already occurred *in utero*.

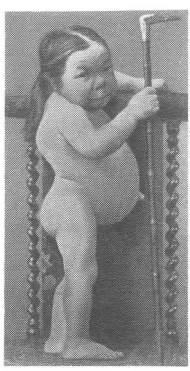


FIG. 646.—A cretin girl aged twenty-two.

Adult Hypothyroidism

Myxœdema is a very advanced form of adult hypothyroidism and this term should never be applied to the milder degree seen much more frequently after thyroidectomy and ^{131}I therapy, and with Autoimmune Thyroiditis. Significant early symptoms are tiredness, mental lethargy, cold intolerance, increase in weight, constipation, menstrual disturbance and a carpal tunnel syndrome due to increased tissue fluid deep to the flexor retinaculum (Hubble). Significant physical signs are a slow pulse rate, dry skin and hair, cold extremities, periorbital puffiness, hoarse voice, slow movements and slow relaxation of the ankle jerks. Comparison of the facial appearance with a previous photograph is helpful.

Tests of Thyroid Function.—The serum T^4 is below 55 nmol/l (3·0 μg/100 ml). Radio-iodine studies show reduced thyroid uptake[2] and increased renal excretion. In an E.C.G. the voltage is reduced and the T wave flattened or even inverted. Serum T.S.H. values are raised and in doubtful cases a T.R.H. test will be helpful. A therapeutic trial of thyroxine is also helpful.

Treatment.—Dried thyroid extract is unstable, difficult to assay, and should not be used. It has been replaced by l-thyroxine—which is no more expensive. A full replacement dose is 0·3–0·4 mg a day and, because of its slow action, it can be given in a single dose. In the elderly or those with myocardial insufficiency, the initial dose must be as low as 0·05 mg a day and

[1] Antithyroid drugs, P.A.S., and iodides in excess.
[2] Thyroid ^{131}I uptake of less than 12% at 24 hours is diagnostic.

Sir Douglas Vernon Hubble, Contemporary. Dean, Faculty of Medicine, Haile Sellassie I University, Addis Ababa, Ethiopia.
Sir William Withey Gull, 1816–1890. Physician Guy's Hospital, London. Wrote the first account of Myxœdema in 1873.

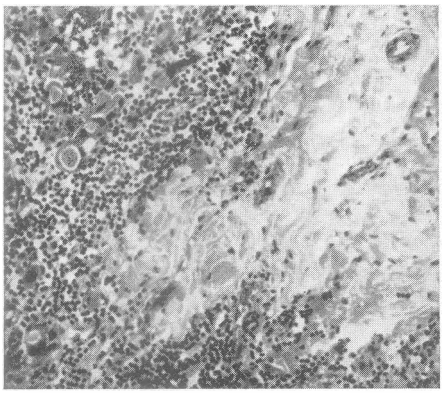


FIG. 647.—Myxœdema. Histology × 100. Gross fibrosis, lymphocytic infiltration and atrophy of acini. Compare with normal histology fig. 653. *(Dr. Bernard Fox, London.)*

FIG. 648.—Myxœdema : note the bloated look, the pouting lips, and the dull expression. *(Dr. V. K. Summers, Liverpool.)*

cautiously increased. If a rapid or short-lived response is essential tri-iodothyronine is used.

Myxœdema.—The signs and symptoms of hypothyroidism are accentuated. The facial appearance (fig. 648) is typical, and there is often supraclavicular puffiness, a malar flush, and a yellow tinge to the skin. Myxœdema coma occurs in neglected cases and carries a high mortality: the body temperature is low and the patient must be warmed slowly: 1 g of intravenous hydrocortisone should be given daily, and intravenous tri-iodo-thyronine in slowly increasing doses.

Auto-immune Thyroiditis.—The so-called primary or atrophic myxœdema is now considered an autoimmune disease similar to Hashimoto thyroiditis (p. 640) but without goitre formation from T.S.H. stimulation. Because of the delay in diagnosis the hypothyroidism is usually much more severe than in goitrous auto-immune thyroiditis.

Dyshormogenesis and Goitrogens.—Genetically determined deficiencies in the enzymes controlling the synthesis of thyroid hormones, if severe, are responsible for goitre formation with hypothyroidism. If of moderate degree, a simple (euthyroid) goitre results.[1, 2] Similarly goitrogens may produce a goitre with, or without, hypothyroidism.

GOITRES

A goitre[3] is an enlarged thyroid gland.

[1] A number of non-endemic goitrous cretins have been born to a group of itinerant tinkers living in Scotland who intermarry (Hutchison). This was due to a deficiency of the enzyme dehalogenase.

Another classical example is Pendred's syndrome, where goitre is associated with congenital deafness. This is due to deficiency of peroxidase, the enzyme responsible for organification of trapped iodides (p. 613).

[2] When thyroglobulin is broken down, uncoupled iodotyrosines are liberated as well as T^3 and T^4. They are broken down by the enzyme dehalogenase and the iodine retained within the thyroid.

If dehalogenase is deficient, iodotyrosines pass into the blood, are excreted in the urine, and this may result in iodine deficiency and goitre formation.

[3] Goitre—Latin, *guttur* = the throat.

James Holmes Hutchison, Contemporary. Professor of Child Health, Glasgow, Scotland.
Vaughan Pendred, 1869–1946. General Practitioner, East Sheen, Surrey, England. Described this Syndrome in 1896.

CLASSIFICATION OF GOITRES

Simple Goitre (Endemic[1] or sporadic[2]) (Euthyroid)	Diffuse hyperplastic goitre. Nodular goitre.
Toxic Goitre	Diffuse toxic goitre (Graves' disease). Toxic nodular goitre. Toxic nodule.
Neoplastic Goitre	Benign. Malignant.
Thyroiditis	Granulomatous thyroiditis. Auto-immune thyroiditis. Riedel's thyroiditis.
Other Rare Goitres	Acute bacterial thyroiditis. Chronic bacterial thyroiditis (Tuberculosis and Syphilis). Amyloid Goitre.

SIMPLE GOITRE

Simple goitre is due to stimulation of the thyroid gland by the anterior pituitary, i.e. by increased levels of circulating T.S.H. T.S.H. secretion is increased by low levels of circulating thyroid hormones. Any factor, therefore, that maintains a persistently low level of circulating thyroid hormones can be responsible for a simple goitre. The most important factor is iodine deficiency, but defects in hormone synthesis may be responsible.

1. **Iodine Deficiency.** The daily requirement of iodine is about 100–125 μg. In nearly all districts where simple goitre is endemic there is a very low iodide content in the water and food. Endemic areas are in the mountainous ranges, such as the Rocky Mountains, the Alps, the Andes and the Himalayas. In Great Britain endemic goitre is found in the Mendips, Chilterns, Cotswolds and the Pennine Chain of Derbyshire and Yorkshire. Endemic goitre is also found in lowland areas where the soil lacks iodide or the water supply comes from far away mountain ranges, e.g. the Great Lakes of North America, the Plains of Lombardy, the Struma Valley,[3] the Nile Valley and the Congo. Calcium is also goitrogenic and goitre is common in low iodine areas on chalk or limestone, e.g. Derbyshire and Southern Ireland. Although iodides in food and water may be adequate, failure of intestinal absorption may produce iodine deficiency (McCarrison).

2. **Defects in Synthesis of Thyroid Hormones.** (*a*) Enzyme deficiency within the thyroid gland (p. 613). (*b*) Goitrogens.

It is probable that enzyme deficiencies of varying degree of severity are responsible for many sporadic goitres, (i.e. in non-endemic areas), and there is often a family history of goitre in these cases suggesting a genetic defect. If the iodine intake is very high an enzyme deficiency may be overcome, e.g. in Iceland where goitre is

[1] Endemic = prevalent in a people or a district.
[2] Sporadic = haphazard occurrence.
[3] Struma. In the mountains of Bulgaria arises the River Struma that flows into the Aegean Sea. Along its banks, and those of its tributaries, dwell persons of several nationalities among whom endemic goitre has long been prevalent. Struma is a continental term for a goitre.

Sir Robert McCarrison, 1878–1960. Indian Medical Service. Director of the Nutrition Research Laboratories, Coonoor, Madras, India.

practically unknown. Enzyme deficiency is often associated with a low iodine intake, e.g. a dislike of sea-fish.

Well known goitrogens are vegetables of the brassica family (cabbage, kale and rape) which contain thiocyanate, drugs such as P.A.S. and, of course, the antithyroid drugs.[1]

Surprisingly enough, iodides in large quantities are goitrogenic as they inhibit the organic binding of iodine and give an iodide goitre.[2]

The Natural History of Simple Goitre. This is investigated by autoradiographs of slices of goitre from patients to whom a pre-operative tracer dose of I^{131} has been given. Blackening of the X-ray film takes place in areas where colloid contains iodine, and the autoradiographs are compared with histological sections of the same slice. In any single lobule all follicles behave uniformly. Active follicles are small, lined by tall cells and their colloid contains iodine. Inactive follicles are three times larger, lined by flattened cells and contain colloid without iodine (Taylor).

Stages in goitre formation are:

1. Persistent T.S.H. stimulation causes diffuse hyperplasia; all lobules are composed of active follicles and iodine uptake is uniform. This is a diffuse hyperplastic goitre which may persist for a long time but is reversible if T.S.H. stimulation ceases.
2. Later, as a result of fluctuating T.S.H. levels, a mixed pattern develops with areas of active lobules and areas of inactive lobules.
3. Active lobules become more vascular and hyperplastic until hæmorrhage occurs causing central necrosis and leaving only a surrounding rind of active follicles.
4. Necrotic lobules coalesce to form nodules filled either with iodine-free colloid or a mass of new but inactive follicles.
5. Continual repetition of this process results in a nodular goitre. Most nodules are inactive and active follicles are present only in the internodular tissue.

CLINICAL TYPES OF SIMPLE GOITRE

1. Diffuse Hyperplastic Goitre

Diffuse hyperplasia corresponds to the first stages of the natural history. The goitre appears in childhood in endemic areas but in sporadic cases it usually occurs at puberty when metabolic demands are high—Puberty Goitre (fig. 649). If T.S.H. stimulation ceases the goitre may regress, but tends to recur later at times of stress such as pregnancy. The goitre is soft, diffuse and may become large enough to cause discomfort. A colloid goitre is a late stage of diffuse hyperplasia when T.S.H. stimulation has fallen off and when many follicles are inactive and full of colloid (fig. 650).

FIG. 649.—Physiological hyperplasia of the thyroid gland (goitre of puberty).

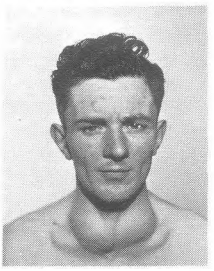


FIG. 650.—Colloid goitre.

[1] Antithyroid drugs—Thiocyanates and perchlorates interfere with iodide trapping, Carbimazole, Thiouracil Compounds and Resorcinol interfere with the oxidation of iodide and the binding of iodine to tyrosin (p. 613).

[2] Iodide goitre is usually seen in asthmatics who have taken proprietary preparations containing iodides (e.g. Felsol) over a prolonged period.

Selwyn Francis Taylor, Contemporary. Formerly Surgeon, Royal Postgraduate Medical School, London.

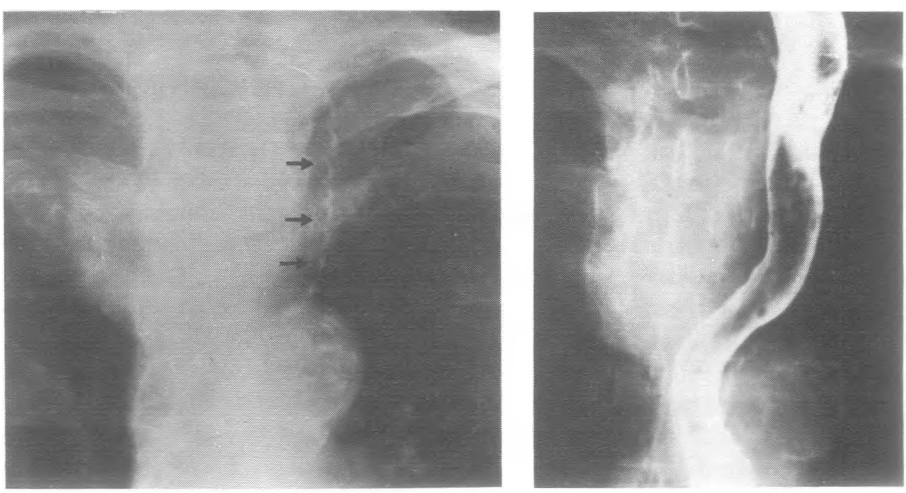
2. Nodular Goitre

Persistent fluctuating T.S.H. stimulation results inevitably in progressive nodule formation. Nodules are usually multiple, forming a multinodular goitre. Occasionally, only one macroscopic nodule is found, but microscopic changes will be present throughout the gland: this is one form of a clinically solitary nodule (p. 622). Nodules may be colloid or cellular, and cystic degeneration and hæmorrhage are common, as is subsequent calcification. Nodules appear early in endemic goitre and later (between 20 and 30) in sporadic goitre, although the patient herself may be unaware of the goitre until the late forties or fifties. All types of simple goitre are far more common in the female than in the male.

Diagnosis is usually straightforward. The patient is euthyroid; the nodules are palpable and often visible; they are smooth, usually firm and not hard, and the goitre is painless and moves freely on swallowing. Hardness and irregularity, due to calcification, may simulate carcinoma. A painful nodule, sudden appearance, or rapid enlargement of a nodule, raises suspicion of carcinoma, but is usually due to hæmorrhage into a simple nodule. Differential diagnosis from a Hashimoto goitre (p. 640) in a euthyroid patient is often difficult.

Investigations.—Tests of thyroid function may be necessary to exclude mild hyperthyroidism, and the estimation of titres of thyroid antibodies to differentiate from Hashimoto goitre. Plain X-rays may show calcification and tracheal deviation or compression (fig. 651).

Complications.—(i). *Tracheal obstruction*—due to gross lateral displacement, or compression in a lateral or antero-posterior plane. Acute respiratory obstruction may follow hæmorrhage into a nodule impacted in the thoracic inlet.



(*a*) (*b*)

FIG. 651.—Retrosternal goitre causing (*a*) deviation of the trachea and (*b*) œsophagus, by barium swallow.

(ii). *Secondary Thyrotoxicosis*—many patients with nodular goitres develop hyperthyroidism sooner or later. The incidence is difficult to estimate, but figures as high as 30% have been suggested.

(iii). *Carcinoma*—which is usually of follicular pattern. It is uncommon—an incidence of 5–8% has been suggested—but an increased incidence has been reported from endemic areas.

Prevention and Treatment of Simple Goitre.—All table salt should be iodised (potassium iodide 1 part in 10,000). In endemic areas (e.g. Switzerland, parts of the U.S.A. and Argentine) the incidence of goitre has been strikingly reduced by this prophylactic measure.

In the early stages a hyperplastic goitre is reversible if l-thyroxine is given in maximum doses—0·3 mg. a day for several months—and then very slowly tailed off to 0·1 mg. a day which should be continued for many years. If regression does not occur, thyroidectomy may be indicated for cosmetic reasons or pressure symptoms.

The nodular stage of simple goitre is irreversible. A multinodular goitre is often uncomfortable and unsightly and, in view of the possible complications, subtotal thyroidectomy is advisable unless the expectation of life is short. Resection aims at removing nodules and leaving up to 8 g. of relatively normal tissue in each remnant. The technique is essentially the same as described for toxic goitre (p. 632) as are the post-operative complications. In many cases the causative factors persist and recurrence is likely, particularly in the younger patient, unless further T.S.H. stimulation is prevented. 0·1 mg. of l-thyroxine a day should be given post-operatively to all patients until after the menopause. Some clinicians give 0·2 mg. a day and consider that it should be given indefinitely. If one thyroid lobe appears normal in size and consistency at operation, it is justifiable not to resect it, but post-operative thyroxine is more than ever essential.

3. The Problem of the Clinically Solitary Nodule

The clinically solitary nodule may be defined as 'A goitre which, on clinical examination, appears to be a single nodule in an otherwise normal gland'.

Clinically solitary nodules fall into two categories. In the first are those in which there is a certainty or grave suspicion of malignancy; in these exploration is essential. In the second and far larger category, there is a smooth, firm, mobile nodule which is probably benign but carries a small but significant risk of being a carcinoma. In this category about 50% prove to be simple multinodular goitres when explored.

The thyroid status of these patients must be established by clinical examination and by laboratory tests, and the nodule should be scanned. This should divide the patients into three categories:

(i) Hyperthyroid with a hot (overactive) nodule.[1]

[1] A hot nodule is one that takes up isotope, whilst the surrounding thyroid tissue does not. Here the surrounding thyroid tissue is inactive because the nodule is producing such high levels of thyroid hormones that T.S.H. secretion is suppressed.

(ii) Euthyroid with a warm (active) nodule.[1]

(iii) Euthyroid with a cold (inactive) nodule.[2]

The solitary toxic nodule is never malignant, it is a toxic adenoma: it is treated by excision or radio-iodine.

The euthyroid warm nodule is a functioning adenoma or a simple nodule with some active thyroid tissue in it. Very rarely, a well differentiated carcinoma will take up isotope. For this reason, and because a functioning adenoma may develop into an overactive adenoma, it should be excised.

The euthyroid cold nodule is suspect, because a carcinoma so rarely takes up isotope if normal thyroid tissue is present. It should be excised.

Resection entails taking a wide margin of healthy thyroid tissue—preferably a lobectomy. Incisional biopsy is absolutely contraindicated. It could result in seeding of malignant cells.

Other investigations which may be undertaken are:

(i) *Needle biopsy.*—This is of little value in the diagnosis of a well differentiated thyroid carcinoma. It should differentiate a solid from a cystic nodule.

(ii) *Ultrasound.*—This should differentiate between solid and cystic nodules and will often detect other impalpable nodules, i.e. demonstrate that the goitre is multinodular.

(iii) *Selenium Scan.*—A highly cellular tumour, synthesising protein, will show up well on a radioactive selenium scan. It should, therefore, distinguish between a benign cold nodule and a malignant cold nodule. Unfortunately, many well differentiated carcinomas are of such low cellular activity that they do not take up selenium.

Note.—The clinically solitary nodule presents a diagnostic problem when the nodule is smooth, firm and mobile in a euthyroid patients. Scanning, needle biopsy and ultrasound may increase or decrease the suspicion of malignancy, but there is only one certain diagnostic procedure and that is excisional biopsy.

4. Retrosternal Goitre

A very few retrosternal goitres arise from ectopic thyroid tissue, but most arise from the lower pole of a nodular goitre. If the neck is short and the pretracheal muscles are strong, as in men, the negative intrathoracic pressure tends to draw these nodules into the superior mediastinum. The degree of descent varies accounting for—(*a*) *Substernal type*—when the nodule is palpable. (*b*) *Plunging type*—when an intrathoracic goitre is occasionally forced into the neck by increased intrathoracic pressure. (*c*) *Intrathoracic goitre.*

Clinical Features.—A history of a previous cervical goitre which has disappeared is not uncommon. A retrosternal goitre may be symptomless or produce severe obstructive symptoms: (*a*) Dyspnœa, particularly at night, cough and stridor.[3] (*b*) Engorgement of neck veins and, in severe cases, of

[1] A warm nodule takes up isotope and so does normal thyroid tissue about it.
[2] A cold nodule takes up no isotope.
[3] Stridor = harsh sound on inspiration.

superficial veins on the chest wall. (*c*) Dysphagia is rare, as is recurrent nerve paralysis. It may also be malignant or toxic.

X-rays show a soft tissue shadow in the superior mediastinum—sometimes with calcification—and often causing deviation and compression of the trachea (fig. 651). An ^{131}I scan may help to distinguish a retrosternal goitre from a mediastinal tumour.

Treatment.—If obstructive symptoms are present it is unwise to treat a retrosternal goitre with antithyroid drugs or radio-iodine, as these may enlarge the goitre. Resection can almost always be carried out from the neck and a sternal split is hardly ever necessary. The cervical part of the goitre should first be mobilised by ligation and division of the superior thyroid vessels, and by ligature and division of the middle thyroid veins and of the inferior thyroid artery. The retrosternal goitre can then be delivered by traction and finger mobilisation. If carcinoma can definitely be excluded, an impacted goitre can be broken up and removed piecemeal. Hæmorrhage is rarely a problem because the goitre takes its blood supply with it from the neck. The recurrent nerve should be identified if possible before delivering the restrosternal goitre, as it is particularly vulnerable to injury from traction or tearing.

TOXIC GOITRE

Thyrotoxicosis (Hyperthyroidism)

The term thyrotoxicosis is retained because hyperthyroidism, i.e. symptoms due to a raised level of circulating thyroid hormones, is not responsible for all manifestations of the disease.

Clinical Types. 1. Diffuse toxic goitre. (Graves' disease).[1] 2. Toxic nodular goitre. 3. Toxic nodule. 4. Hyperthyroidism due to rarer causes.

FIG. 652.—Primary toxic goitre.
(*Professor E. J. Wayne, Glasgow.*)

1. **Diffuse Toxic Goitre.**—Graves' disease—a diffuse vascular goitre appearing at the same time as the hyperthyroidism, usually in the younger woman and frequently associated with eye signs. The syndrome is that of primary thyrotoxicosis (fig. 652). The whole of the functioning thyroid tissue is involved, and the hypertrophy and hyperplasia are due to abnormal thyroid stimulators such as L.A.T.S.

2. **Toxic Nodular Goitre.**—A simple nodular goitre is present for a long time before the hyperthyroidism, usually in the middle aged or elderly and very infrequently associated with severe eye signs. The syndrome is that of secondary thyrotoxicosis.

In many cases of toxic nodular goitre, the nodules are inactive, and it is the

[1] First noted in 1786 by Caleb Hillier Parry, 1755–1822, Physician, General Hospital, Bath, England. His account was published posthumously in 1825.

Robert James Graves, 1796–1853. Physician, Meath Hospital, Dublin. Published his account of exophthalmic goitre in 1835.

internodular thyroid tissue that is overactive. Here the hyperthyroidism is due to abnormal thyroid stimulators such as L.A.T.S. However, in some toxic nodular goitres one or more nodules are overactive and here the hyperthyroidism is due to autonomous thyroid tissue as in the toxic nodule.

3. **Toxic Nodule.**—A solitary overactive nodule. It is autonomous and its hypertrophy and hyperplasia are not due to L.A.T.S. Because T.S.H. secretion is suppressed by the high level of circulating thyroid hormones, the normal thyroid tissue surrounding the nodule is itself suppressed and inactive.

Histology. The normal thyroid gland (fig. 653) consists of acini lined by flattened cuboidal epithelium and filled with homogenous colloid. In hyperthyroidism (fig. 654) there is hyperplasia of acini which are lined by high columnar epithelium. Many of them are empty, and others contain vacuolated colloid.

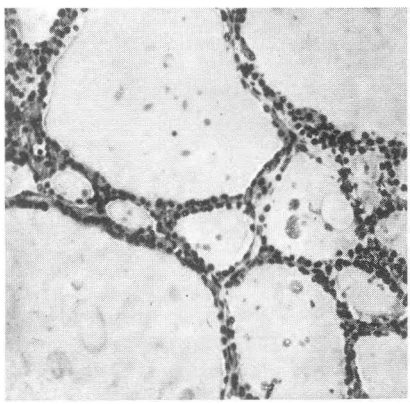


FIG. 653.—Normal thyroid (see text). × 100.

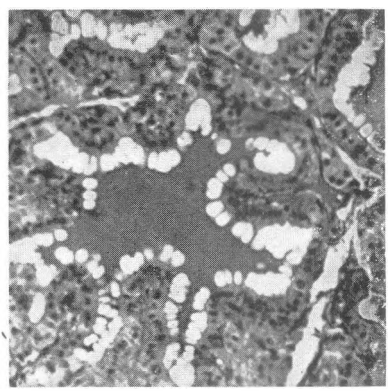


FIG. 654.—Thyrotoxicosis (see text). × 100. (*Dr. Bernard Fox, London.*)

Symptomatology.—Thyrotoxicosis is 8 times commoner in females than in males. It may occur at any age. Wayne's clinical diagnostic index (p. 626) gives all the important symptoms and signs of thyrotoxicosis and indicates by their score the relative importance of each. The most significant symptoms are loss of weight in spite of a good appetite, a recent preference for cold, and palpitations. The most significant signs are the excitability of the patient, the presence of a goitre, exophthalmos, and tachycardia or cardiac arrhythmia.

The goitre in primary thyrotoxicosis is diffuse and vascular, it may be large or small, firm or soft, and a thrill and a bruit may be present, usually at the upper poles over the superior thyroid arteries. The onset is abrupt, but remissions and exacerbations are not infrequent. Hyperthyroidism is usually more severe than in secondary thyrotoxicosis but cardiac failure is rare. Manifestations of thyrotoxicosis not due to hyperthyroidism *per se*, e.g. orbital proptosis, ophthalmoplegia and pretibial myxœdema may occur in primary thyrotoxicosis.

In secondary thyrotoxicosis the goitre is nodular. The onset is insidious and may present with cardiac failure or atrial fibrillation. It is characteristic

Sir Edward Johnson Wayne, Contemporary. Formerly Regius Professor of Medicine, Glasgow, Scotland.

that the hyperthyroidism is not severe. Eye signs other than lid lag and lid spasm (due to hyperthyroidism) are very rare.

Cardiac rhythm.—A fast heart rate, which persists during sleep, is characteristic. Cardiac arrhythmias are superimposed on the sinus tachycardia as the disease progresses, and they are commoner in older patients with

WAYNE'S CLINICAL DIAGNOSTIC INDEX

Symptom score + Sign score = Diagnostic Index

INDEX—Under 11 = Non-Toxic
11–19 = Equivocal
over 19 = Toxic

Symptoms of Recent Onset and/or Increased Severity	*Present*	
	Score	*Score*
Dyspnœa on effort	+1	
Palpitations	+2	
Tiredness	+2	
Preference for heat (irrespective of duration)		−5
Preference for cold	+5	
Indifferent to temperature	0	
Excessive sweating	+3	
Nervousness	+2	
Appetite increased	+3	
,, decreased		−3
Weight increased		−3
,, decreased	+3	

Signs	*Present Score*	*Absent Score*
Palpable thyroid	+3	−3
Bruit over thyroid	+2	−2
Exophthalmos	+2	
Lid retraction	+2	
Lid lag	+1	
Hyperkinetic movements	+4	−2
Fine finger tremor	+1	
Hands		
Hot	+2	−2
Moist	+1	−1
Casual pulse rate:		
Auricular fibrillation	+4	
Regular rates:		
80 per minute		−3
80 to 90 per minute	0	
90 per minute	+3	

thyrotoxicosis because of the prevalence of coincidental heart disease. Stages of development of thyrotoxic arrythmias are:—

(1) Multiple extrasystoles. (2) Paroxysmal atrial tachycardia. (3) Paroxysmal atrial fibrillation. (4) Persistent atrial fibrillation, not responsive to digoxin.

Myopathy.—Weakness of the proximal limb muscles is commonly found if looked for. Severe muscular weakness (Thyrotoxic myopathy) resembling myæsthenia gravis, occurs occasionally. Recovery occurs as hyperthyroidism is controlled.

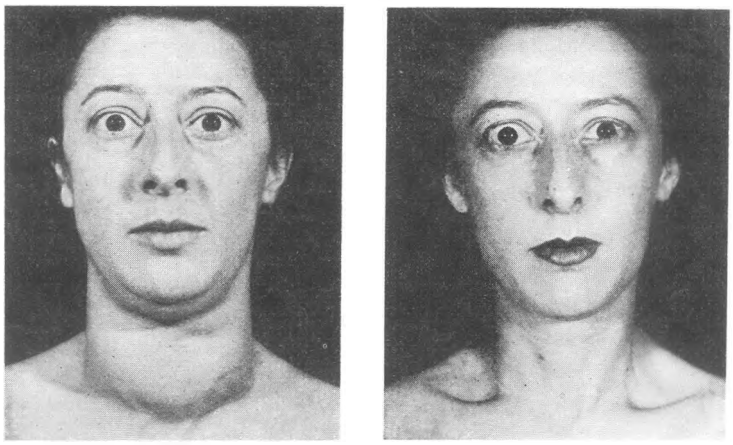
Exophthalmos.—Some degree of exophthalmos is common. It may be unilateral. True exophthalmos is a proptosis of the eye, caused by infiltration of the retrobulbar tissues with fluid and round cells, with a varying degree of retraction or spasm[1] of the upper eyelid. This results in widening of the palpebral fissure (the inner and outer canthus) so that the sclera can be seen clearly above the upper margin of the iris and cornea (above the ' limbus ').

Spasm and retraction usually disappear when the hyperthyroidism is

[1] Lid spasm occurs because the levator palpebræ superioris muscle is partly innervated by sympathetic fibres.

controlled. They may be improved by β-adrenergic blocking drugs, e.g. Guanethidine eye drops. The condition is aggravated by compression of the ophthalmic veins.

The proptosis can be measured with an exophthalmometer.[1] Œdema of the eyelids, conjunctival injection and chemosis are aggravated by com-



(*a*) (*b*)

FIG. 655.—(*a*) Prior to sub-total thyroidectomy following preparation with carbimazole and potassium iodide; note lid retraction, which is more marked on the right. (*b*) Relapse of thyrotoxicosis. Weight loss; a bilateral partial tarsorrhaphy has been performed because of early exposure keratitis. (*Dr. Bruce Fowler, London.*)

pression of the ophthalmic veins. Weakness of the extra-ocular muscles, particularly the elevators, results in diplopia. In severe cases papillœdema and corneal ulceration occur. When severe and progressive it is known as malignant exophthalmos (fig. 656) and the eye may be destroyed. It has

FIG. 656.—Progressive exophthalmos, with chemosis and exophthalmic ophthalmoplegia. (*The Lahey Clinic, Boston, Mass., U.S.A.*)

been postulated that the syndrome of infiltrative ophthalmopathy is due to a pituitary hormone E.P.S. (Exophthalmos Producing Substance).

Exophthalmos is usually self-limiting and may even regress a little. Sleeping

[1] Exophthalmometry. In a person with normal vision a reading of 19 mm. or above is abnormal. In the short-sighted however, the bulb is elongated and each −3 diopters correction represents an increase of 1 mm.

propped up and lateral tarsorrhaphy will help to protect the eye but will not prevent progression. Hypothyroidism increases proptosis by a few millimetres and must be avoided.

FIG. 657.—Pretibial myxœdema in hyperthyroidism.

Improvement has been reported with massive doses of prednisone and with metronidazole. Intra-orbital injection of steroids is dangerous because of the venous congestion, and total thyroid ablation has not proved effective. When the eye is in danger, orbital decompression may be required.

Pretibial Myxœdema (fig. 657) is a thickening of the skin by a mucin-like deposit, nearly always associated with true exophthalmos, past or present hyperthyroidism, and high levels of L.A.T.S. It is usually symmetrical, and minor degrees are not uncommon but are easily missed. The earliest stage is a shiny red plaque of thickened skin with coarse hair, which may be cyanotic when cold. In severe cases the skin of the whole leg below the knee is involved, together with that of the foot and ankle and there may be clubbing of the fingers and toes. (Thyroid Acropachy.)

Diagnosis of Thyrotoxicosis. — Most cases are diagnosed clinically and the Wayne diagnostic index (p. 626) is an excellent discipline. Difficulty is most likely to arise in the differentiation of mild hyperthyroidism from an anxiety state when a goitre is present. In these cases the thyroid status is determined by the diagnostic tests described on p. 613. If there is still doubt after a routine Thyroid Profile and a ^{131}I uptake test, then a T.R.H. test should be done.

T^3 thyrotoxicosis is diagnosed by estimating the serum T^3. It should be suspected if the clinical picture is suggestive, but routine tests of thyroid function are within the normal range. A thyroid scan is essential in the diagnosis of an autonomous toxic nodule.

Thyrotoxicosis should always be considered in : (*a*) children with a growth spurt, behaviour problems or myopathy. (*b*) Tachycardia or arrhythmia in the elderly. (*c*) Unexplained diarrhœa. (*d*) Loss of weight.

Principles of the Treatment of Thyrotoxicosis.—Non-specific measures are rest and sedation, and in established thyrotoxicosis should be used only in conjunction with specific measures which are the use of antithyroid drugs, surgery and radio-iodine.

Antithyroid Drugs.—Those in common use are Carbimazole and Propyl-Thiouracil. Potassium perchlorate and Thiouracil[1] are infrequently used these days and iodides should only be used as immediate pre-operative drugs. Propranolol (p. 632) may also be used. Antithyroid drugs are used to restore the patient to a euthyroid state and to maintain this for a prolonged

[1] In 1943 Astwood first treated thyrotoxicosis successfully with thiourea.

Edwin Bennett Astwood, Contemporary. Physician, New England Center Hospital, Boston, Mass., U.S.A.

period in the hope that a permanent remission will occur (i.e. that production of Thyroid Stimulating Immunoglobulins (T.S.I.) will diminish or cease). It should be noted that antithyroid drugs can not ' cure ' a toxic nodule. The overactive thyroid tissue is autonomous and recurrence of the hyperthyroidism is certain when the drug is discontinued.

Advantages.—No surgery and no use of radio-active materials.
Disadvantages:—
(*a*) Treatment is prolonged and the failure rate after a course of 1½ to 2 years is at least 50%.
(*b*) It is impossible to predict which patient is likely to go into a remission.[1]
(*c*) Some goitres enlarge and become very vascular during treatment[2]—even if thyroxine is given at the same time.
(*d*) Very rarely there is a dangerous drug reaction, e.g. agranulocytosis[3] or aplastic anæmia.

Initially, 10 mg. of Carbimazole[4] are given three or four times a day, and there is a latent interval of 7–14 days before any clinical improvement is apparent. It is most important to maintain a high concentration of the drug throughout the 24 hours by spacing the doses at 8 or 6 hourly intervals. When the patient becomes euthyroid a maintenance dose of 5 mg. two or three times a day is given for another 12–18 months. If Tri-iodo-thyronine—20 μg 2 to 4 times daily, or Thyroxine—0·2 mg daily, are given in conjunction with antithyroid drugs, there is less danger of producing iatrogenic thyroid insufficiency or an increase in the size of the goitre.

Surgery. (p. 632).—In diffuse toxic goitre and toxic nodular goitre with overactive internodular tissue, surgery cures by reducing the mass of overactive tissue. It seems that a cure is probable if the thyroid tissue can be reduced below a critical mass.[5] In the autonomous toxic nodule, and in toxic nodular goitre with overactive autonomous toxic nodules, surgery cures by removing all the overactive thyroid tissue: this allows the suppressed normal tissue to function again.

Advantages.—The goitre is removed, the cure is rapid, and the cure rate is high if surgery has been adequate.
Disadvantages.—A recurrence of thyrotoxicosis occurs in about 5 per cent. of cases. Every operation carries a morbidity and mortality. However, with suitable preparation and an experienced surgeon the mortality is negligible. Although post-operative thyroid insufficiency occurs in about 20 per cent of cases this is rarely due to the operation itself (p. 634). The only significant complication due to the operation is parathyroid insufficiency (p. 634).

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[1] It has been suggested that drug responsive patients can be identified after 6 months treatment by a positive response to a T^3 suppression test. The 20 minute uptake of intravenous ^{132}iodine is measured so that treatment with Carbimazole need not be discontinued. This test has not yet been fully evaluated.
[2] This is probably due to T.S.I. stimulation during the prolonged course of treatment and not a direct effect of the drug.
[3] Agranulocytosis—The patient should be instructed to discontinue treatment if she develops a sore throat until the white cell count has been checked.
[4] Carbimazole = proprietary name Neomercazole, 5 mg. tablets.
[5] This may result in a reduction of T.S.I. production, or it may be that the circulating T.S.I., however high its level, can only produce limited hypertrophy and hyperplasia when the mass of thyroid tissue is small.

Radio-iodine.—Radio-iodine[1] destroys thyroid cells and, as in thyroidectomy, reduces the mass of functioning thyroid tissue to below a critical level.

Advantages.—No surgery and no prolonged drug therapy.
Disadvantages.—(*a*) Isotope facilities must be available. (*b*) There is a high and progressive incidence of thyroid insufficiency which may reach 75–80 per cent. after 10 years.[2] (*c*) An indefinite follow-up is essential.

There is no convincing evidence that radio-iodine has been responsible for genetic damage, leukæmia, damage to the fœtus if given inadvertently in early pregnancy, or carcinoma in the adult. In some North American Clinics radio-iodine is given to almost all patients over the age of 25—when development is complete. In this country, on account of the real and potential risks, most clinicians are unwilling to advise it under the age of 45. The dose of radio-iodine varies with the size of the goitre, and a suggested dosage is 160 μCi per 1 g of thyroid tissue. Thus, for a gland estimated at 60 g, between 9 and 10 mCi would be prescribed.[3] Response is slow, but a substantial improvement is to be expected in 8 to 12 weeks. Accurate dosage is difficult and, should there be no clinical improvement after 12 weeks, a further dose is given (Macgregor). Two or more doses are necessary in 20–30 per cent. of cases.

The Choice of Therapeutic Agent.—Each case must be considered individually. Below are listed guiding principles as to the most satisfactory treatment for a particular toxic goitre at a particular age: these must however be modified according to the facilities available and the personality, intelligence and wishes of the individual patient, her business or family commitments and any other co-existent medical or surgical condition.

1. *Diffuse Toxic Goitre.*—Over 45—Radio-iodine.
Under 45—Surgery for the large goitre—Antithyroid drugs for the small goitre.

Large goitres are uncomfortable and remission with antithyroid drugs is less likely than in the small goitre.

2. *Toxic Nodular Goitre.*—Surgery.

Toxic nodular goitre does not respond as well or as rapidly to radio-iodine or antithyroid drugs as does a diffuse toxic goitre, and the goitre itself is often large and uncomfortable.

3. *Toxic Nodule.*—Surgery or Radio-iodine.

Resection is easy, certain and without morbidity. Radio-iodine is, however, the treatment of choice over the age of 45 because the suppressed thyroid tissue does not take up iodine and there is thus no risk of delayed thyroid insufficiency.

4. *Recurrent Thyrotoxicosis after adequate surgery*—over 45 radio-iodine,—under 45 antithyroid drugs.

5. *Failure of previous treatment with antithyroid drugs or radio-iodine.*—Surgery or thyroid ablation with ^{131}I.

In advising treatment, the age of 45 must not be taken as a rigid dividing line. Intelligence is important: unintelligent patients cannot be trusted to

[1] Radio-active iodine was first used in the treatment of thyrotoxicosis by Hertz and Roberts in 1942. Isotope—Greek, *isos* = equal, *topos* = place.

[2] This is due to sublethal damage to those cells not actually destroyed by the initial treatment and this eventually causes failure of cellular reproduction.

[3] A more sophisticated method is to give a dose of 7,500 rad, calculated from the peak uptake of ^{131}I in the thyroid and the length of time the ^{131}I remains in the gland.

Saul Hertz, Contemporary. Director of Radioactive Isotope Research Institute, Boston, Mass., U.S.A.
A. Roberts, Contemporary. Isotope Department, Massachusetts Institute of Technology, Boston, Mass., U.S.A.
Alastair Goold Macgregor, 1919–1972. Regius Professor of Materia Medica and Therapeutics, Aberdeen, Scotland.

take drugs regularly if they feel well, and are unlikely to attend follow-up clinics indefinitely, which is essential after radio-iodine therapy.

Special Problems in Treatment

1. **Pregnancy.**—Radio-iodine is absolutely contraindicated because of the risk to the fœtus. The danger of surgery is miscarriage; and that of antithyroid drugs is of inducing thyroid insufficiency in the mother, and because both T.S.H. and antithyroid drugs cross the placenta, of the baby being born goitrous (fig. 658) and hypothyroid. The risk of either surgery or antithyroid drugs in competent hands is very small and the choice is exactly as in an uncomplicated case.

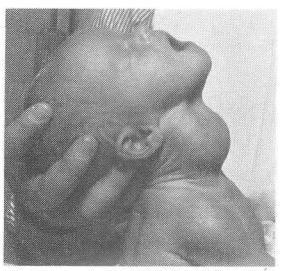


FIG. 658.—Transmitted thiouracil goitre.[1]

2. **Children.**—Radio-iodine is absolutely contraindicated because of the risk of inducing thyroid carcinoma. There is an increased risk of recurrence after thyroidectomy because thyroid cells are highly active in the young. It is probably best to treat children and adolescents with antithyroid drugs until the late teens. If thyroidectomy is to be undertaken it must be very radical.

3. **The Thyro-cardiac.**—This is a patient with severe cardiac damage due wholly or partly to hyperthyroidism. The patient is usually middle aged or elderly with secondary thyrotoxicosis and the hyperthyroidism is not very severe. The cardiac condition is far more significant than the hyperthyroidism, but this must be rapidly controlled to prevent further cardiac damage.

Thyroidectomy is the treatment of choice after control of the cardiac condition. In the elderly, however, or when the operative risk is unacceptable, radio-iodine is given, and treatment with antithyroid drugs is started 48 hours later and continued until the radio-iodine has had effect.

4. **High Pre-operative Titres of Thyroid Antibodies.**—Their presence indicates lymphatic infiltration of the goitre—i.e. a diffuse or focal thyroiditis—and a liability to thyroid insufficiency after thyroidectomy. These patients are best treated conservatively unless the goitre is large, vascular and uncomfortable.

5. **Proptosis of Recent onset.**—It is considered unwise to terminate thyrotoxicosis abruptly by thyroidectomy or radio-iodine if proptosis is recent, because malignant exophthalmos may be induced. There is no real proof of this, but it is advisable to treat these patients with antithyroid drugs, until the proptosis has been static for six months.

Hyperthyroidism due to other causes

1. *Thyrotoxicosis factitia.*[2] Hyperthyroidism can be induced by taking l-thyroxine, but only if the dosage exceeds the normal requirements of 0·3–0·4 mg. a day. Doses below the normal requirements simply suppress normal hormone production by the thyroid.

2. *Jod-Basedow.*[3] *Thyrotoxicosis.* Large doses of iodide given to a hyperplastic endemic goitre which is iodine avid may produce temporary hyperthyroidism, and very occasionally a persistent hyperthyroidism.

3. *In subacute or acute forms of autoimmune thyroiditis or of de Quervain's thyroiditis* (p. 640), mild hyperthyroidism may occur in the early stages due to liberation of thyroid hormones from damaged tissue.

4. *A large mass of secondary carcinoma* will rarely produce sufficient hormone to induce mild hyperthyroidism.

5. *Neonatal thyrotoxicosis* occurs in babies born to hyperthyroid mothers or to euthyroid mothers who have had thyrotoxicosis. High T.S.I. titres are present in

[1] This does not occur if T^3 is given with antithyroid drugs as it too crosses the placenta.

[2] Usually seen in health cranks or those given thyroid extract as 'a tonic'.

[3] Jod—German for iodine + Basedow. In European countries Diffuse Toxic Goitre is often called Basedow's disease.

Carl Adolph von Basedow, 1799–1854. General Practitioner, Merseburg, Germany. Published his account of exophthalmic goitre in 1840.

both mother and child because T.S.I. can cross the placental barrier. The hyperthyroidism gradually subsides in 3 to 4 weeks' time as the T.S.I. titres fall in the baby's serum (p. 613).

Surgery for Thyrotoxicosis

Pre-operative Preparation (fig. 659).—The patient must be euthyroid or near euthyroid at operation. The thyroid state is determined by clinical assessment, i.e. by improvement in previous symptoms and by objective signs such as gain in weight and lowering of the pulse rate, and by serial estimations of the thyroid profile.

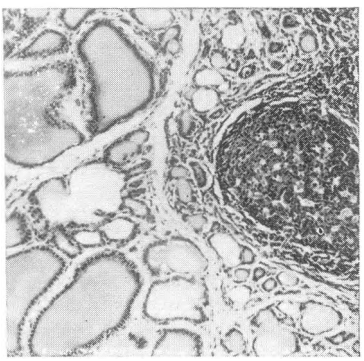


FIG. 659.—'Prepared' thyrotoxicosis. The acini are lined by low cuboidal epithelium and contain more colloid. Compare with figs. 653 and 654. Note the lymphocytic infiltration with lymph follicle formation, which is a feature of the thyrotoxic gland. × 100. (*Dr. Bernard Fox, London.*)

Preparation is as an outpatient and only rarely is admission to hospital necessary on account of severe symptoms at presentation, or failure to control the hyperthyroidism. Failure to control with antithyroid drugs is unusual but may be due to uneven dosage—i.e. not taking the drug at 6 or 8 hourly intervals.

Carbimazole[1] 30–40 mg a day is the drug of choice for preparation. When euthyroid—after 8–12 weeks—the dose is reduced to 5 mg 8 hourly : it is unwise to stop Carbimazole completely prior to operation. Iodides[2] are now very rarely used alone because, if the patient needs pre-operative treatment, a more effective drug should be given. Iodides may be given with Carbimazole for 2–3 weeks immediately prior to operation, but their use is of doubtful value and has been given up in many centres.

Propranolol 40 mg t.d.s. is used by some to prepare less severe cases. Because it acts on the target organs and not on the thyroid gland itself, it does not interfere with the synthesis of thyroid hormones, so that hormone levels remain high during treatment and for some days after thyroidectomy. It is, therefore, important to continue to give the drug for 7 days post-operatively.

Propranolol controls symptoms very rapidly so that its principal value is in combination with Carbimazole in the immediate treatment of patients with very severe hyperthyroidism.

Subtotal Thyroidectomy.—Pre-operative investigations to be carried out and recorded are—(*a*) Indirect laryngoscopy—a symptomless cord paralysis is

[1] Potassium perchlorate—200 mg. 6 hourly may be used pre-operatively but Lugol's iodine cannot be used with it, as perchlorate interferes with the trapping of iodide. To stop potassium perchlorate and give iodides is highly dangerous and may promote thyroid crisis at operation.

[2] Lugol's Iodine—Dose 30 minims (drops) t.d.s. It is helpful to the patient to prescribe in the old-fashioned minims as one minim = 1 drop. The value of iodide is to reduce the vascularity of the goitre and make it softer and easier to handle. Potassium iodide tablets N.F. 60 mg t.d.s. may be used instead of Lugol's iodine.

Jean Guillaume Auguste Lugol, 1786–1851. Physician, Hôpital Saint-Louis, Paris. Henry Stanley Plummer, 1874–1937, Physician, Mayo Clinic, Rochester, U.S.A. was the first to use iodine for preparing thyrotoxic patients for operation.

present in 3 per cent. of cases, without any previous thyroid surgery. (*b*) Thyroid antibodies (p. 640). (*c*) Serum calcium estimation. (*d*) A scan prior to pre-operative preparation is essential in patients with toxic nodular goitre. The surgeon should know which nodules, if any, are autonomous and active in order to ensure their resection. A scan is of no value in diffuse toxic goitre when uptake, for practical purposes, is uniform. The diagnosis of a single toxic nodule can only be made by demonstrating that the nodule is active and the remaining thyroid tissue suppressed.

Technique.—General anæsthesia is administered by an endotracheal tube; local anæsthesia is rarely advisable. A crease incision is made, and flaps of skin, subcutaneous tissue and platysma are raised upwards to the superior thyroid notch and downwards to the suprasternal notch. Adequate exposure entails mobilisation of the sterno-mastoid muscles by incising the fascia along their medial border, midline incision of the cervical fascia, and transverse division of the pretracheal muscles to the carotid sheath. If the pretracheal muscles are not divided, safe ligation of the superior thyroid arteries may be difficult. The thyroid lobes are mobilised by division between ligatures of the middle thyroid veins, the superior thyroid vessels, and inferior thyroid veins. The inferior thyroid arteries are ligated in continuity or divided between ligatures. The recurrent laryngeal nerve should be identified in its course in the operative field. It should first be sought below the level of the inferior thyroid artery as it passes obliquely upwards and forwards. This course (fig. 660), oblique to the trachea and œsophagus is accentuated by mobilisation of the thyroid lobe: only rarely does the nerve lie in the groove between the trachea and œsophagus. The nerve often lies between the branches of the inferior thyroid artery so that identification is helped by traction on a ligature on the trunk of the artery, and if not seen it can frequently be palpated as a taut strand. The nerve passes into the larynx immediately behind the inferior cornu of the thyroid cartilage which is therefore a very important landmark. If the right nerve cannot be found in its usual course, an anomalous nerve, present in 1% of cases, should be suspected: this arises from the vagus trunk and usually passes from behind the carotid sheath curving medially, forwards, and upwards and may be mistaken for the inferior thyroid artery. The parathyroid glands are protected by careful inspection of the goitre before resection and by avoiding ligatures and sutures close to the hilum of identified glands. Subtotal resection of each lobe is carried out leaving a remnant of 4 to 5 g on each side.[1] Absolute hæmostasis is secured by ligation of individual vessels and by suture of the thyroid remnants to the tracheal fascia (fig. 661). The pretracheal muscles and cervical fascia are sutured and the wound closed with suction drainage to the deep cervical space.

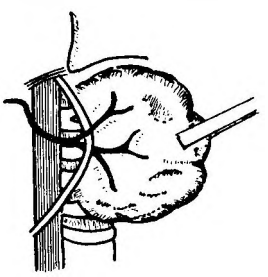


FIG. 660. — Identification of the recurrent laryngeal nerve (see text).

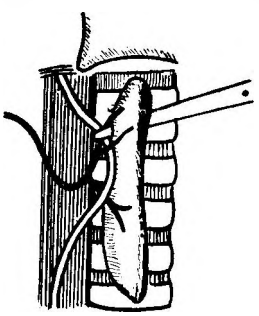


FIG. 661.—On obtaining hæmostasis special care should be exercised on the medial side of the thyroid remnant as the nerve can easily be damaged.

Post-operative Complications

1. **Hæmorrhage.**—A tension hæmatoma deep to the cervical fascia (fig. 662), is usually due to slipping of a ligature on the superior thyroid artery: occasionally hæmorrhage from a thyroid remnant or a thyroid vein may be responsible. It may be necessary to open the wound in

[1] The critical mass for the thyroid remnant is arbitrarily estimated at one-third of the size of a normal thyroid gland (20–25 g).

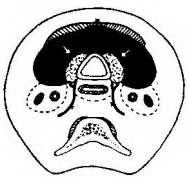


FIG. 662.—Tension hæmatoma deep to the pretracheal muscles. (*After V. H. Riddell, F.R.C.S., London.*)

the ward in order to relieve tension before taking the patient to theatre to evacuate the hæmatoma and to tie off a bleeding vessel if one can be found. A small subcutaneous hæmatoma or collection of serum may form and should be evacuated or aspirated.

2. **Respiratory Obstruction.**—Is very rarely due to collapse or kinking of the trachea. Most cases are due to laryngeal œdema. The most important cause of laryngeal œdema is a tension hæmatoma. However, trauma to the larynx by anæsthetic intubation and surgical manipulation are important contributory factors—particularly if the goitre is very vascular—and may cause laryngeal œdema without a tension hæmatoma. Unilateral or bilateral recurrent nerve paralysis will not cause immediate postoperative respiratory obstruction unless laryngeal œdema is also present, but they will aggravate the obstruction.

If releasing the tension hæmatoma does not immediately relieve airway obstruction, the trachea should be intubated at once. An endotracheal tube can be left in place for several days; steroids are given to reduce œdema and a tracheostomy is rarely necessary. Intubation in the presence of laryngeal œdema may be very difficult and should be carried out by an experienced anæsthetist; repeated unsuccessful attempts may increase obstruction and cause serious cerebral anoxia. In an emergency it is far safer for the inexperienced surgeon to perform a needle tracheostomy as a temporary measure, a Medicut 12G needle (diameter 2·3 mm) is highly satisfactory.

3. **Recurrent Laryngeal Nerve Paralysis** may be unilateral or bilateral, transient or permanent (p. 707). Transient paralysis occurs in about 3% of nerves at risk and recovers in 3 weeks to 3 months. Permanent paralysis is extremely rare if the nerve has been identified at operation.

4. **Thyroid Insufficiency** (p. 617).—This usually occurs within two years, but it is sometimes delayed for 5 years or more. It is often very insidious and difficult to recognise. The incidence is considerably higher than used to be thought and figures above 20 per cent have been reported after operations on diffuse toxic goitres and toxic nodular goitres with internodular hyperplasia. It represents a change in the auto-immune response from stimulation to destruction of thyroid cells and is only rarely a result of operative removal of too much thyroid tissue. Thyroid Insufficiency is rare after surgery for a toxic adenoma, because there is no auto-immune disease present.

5. **Parathyroid Insufficiency**[1] (p. 646). Most cases present dramatically 2–5 days after operation, but very rarely the onset is delayed for 2–3 weeks, or a patient with marked hypocalcæmia is asymptomatic.

6. **Thyrotoxic Crisis** (**Storm**) is an acute exacerbation of hyperthyroidism. It occurs if a thyrotoxic patient has been inadequately prepared for thyroidectomy, and is now extremely rare.[2] Symptomatic treatment is for dehydration, hyperpyrexia and restlessness. Specific treatment is by Propranalol (β adrenergic blocking drug) 40 mg 6 hourly. Iodides and antithyroid drugs are slow acting and therefore of no immediate value.

7. **Wound Infection.**—A subcutaneous or deep cervical abscess should be drained.

8. **Keloid Scar** is more likely to form if the incision overlies the sternum. Intradermal injections of corticosteroid should be given at once and repeated monthly if necessary.

Post-operative Follow-up.

(1) Indirect laryngoscopy should be carried out routinely before leaving hospital.

(2) Screening for parathyroid insufficiency by serum calcium estimation at 6 weeks.

(3) Observation at 6 monthly and then yearly intervals for recurrence of hyperthyroidism—which occurs in 5–10 per cent. of cases.

(4) Observation at 6 monthly and then yearly intervals for delayed thyroid in-

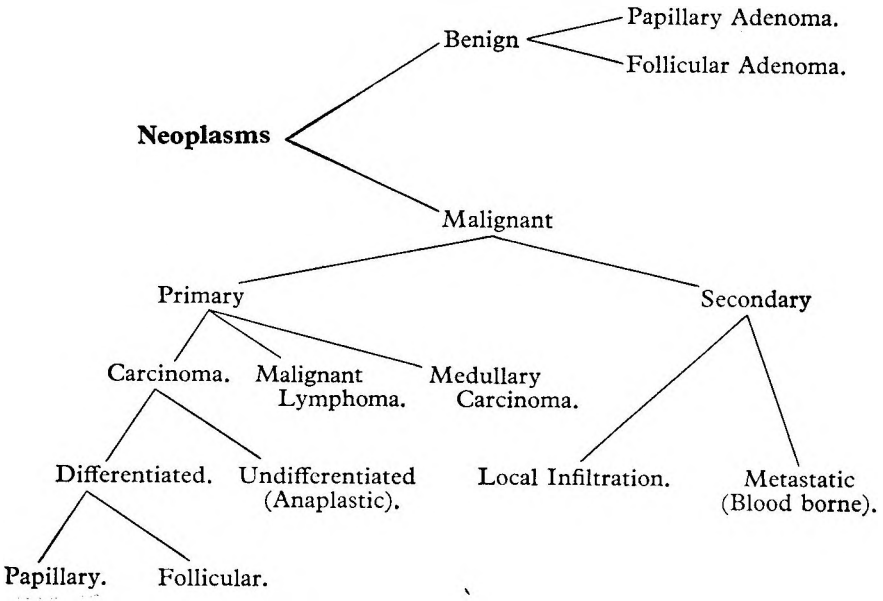
[1] Parathyroid insufficiency is due to removal of parathyroid glands, or infarction through damage to the parathyroid end-artery: often both factors occur together. Vascular injury probably far more important than inadvertent removal.

[2] Very rarely a thyrotoxic patient presents in a crisis and this may follow an unrelated operation.

sufficiency, particularly if the resected goitre showed lymphocytic infiltration or if pre-operative thyroid antibody titres were high. Follow up should be for life.

NEOPLASMS OF THE THYROID

Classification of Thyroid Neoplasms



Benign Tumours.—Follicular adenomas present as clinically solitary nodules and frequently the distinction between a follicular carcinoma and an adenoma can only be made by histological examination: in the adenoma there is no invasion of the capsule or of pericapsular blood vessels. Treatment is, therefore, by wide excision—preferably a lobectomy. The remaining thyroid tissue is normal so that prolonged follow-up is unnecessary. It is doubtful if there is such an entity as a papillary adenoma and all papillary tumours should be considered as malignant even if encapsulated. In all groups the tumour tends to be T.S.H. dependant.

Malignant Tumours.—Secondary growths are rare. Blood borne metastases occur from primary carcinomas of breast, colon, kidney and from melanomas. The vast majority of primary growths are carcinomas (fig. 663). Dunhill classified them histologically as differentiated and undifferentiated : we now subdivide the differentiated carcinomas into follicular

Relative Incidence	(%)
Papillary Carcinoma	60
Follicular Carcinoma	17
Anaplastic Carcinoma	13
Medullary Carcinoma	6
Malignant Lymphoma	4

FIG. 663.—Relative incidence of primary malignant tumours of the thyroid gland.

Sir Thomas Peel Dunhill, 1876–1957. Surgeon, St. Bartholomew's Hospital, London.

and papillary. Clinico-pathological studies have demonstrated a pattern of behaviour according to histological type, which has an important bearing on treatment.

Papillary Carcinoma.—Most papillary tumours contain a mixture of papillary and colloid filled-follicles, and in some the follicular structure predominates. Nevertheless, if any papillary structure is present, the tumour will behave in a predictable fashion as a papillary carcinoma.

Woolner has divided papillary tumours into three subgroups according to their size and extent (fig. 664). Multiple foci may occur in the same lobe as the primary tumour or, less commonly, in both lobes. They are probably due to lymphatic spread in the rich intrathyroidal lymph plexus, rather than to multicentric growth. Spread to the lymph nodes is common but blood-borne metastases are most unusual unless the tumour is extrathyroidal.[1]

CLASSIFICATION BY SIZE AND INFILTRATION.

	A *Occult < 1·5 cm*	*B* *Intrathyroidal*	*C* *Extrathyroidal*
Incidence	31%	53%	15%
Encapsulation	Rare	—	—
Lymph spread	33%	55%	60%
Blood spread	—	—	Late
Death due to growth	Nil/240	9/354	10/68

FIG. 664.—Subgroups of papillary carcinoma and their behaviour. (*after Woolner*)

The primary aim of treatment is to eradicate the growth. The secondary aim is to prevent local recurrence, because a long and comfortable life is possible, even when metastases are present, provided the trachea and larynx are not involved. Because of the high incidence of multiple foci, a total thyroidectomy should be carried out and, in the extrathyroidal tumours, this may entail resection of part of the trachea or a laryngectomy. Because of the high incidence of lymph metastases, even in occult tumours, the pretracheal and paratracheal nodes on the side of the primary tumour should be resected: any other involved nodes should be removed individually and only rarely is any form of block dissection necessary. After operation T.S.H. production must be suppressed by full doses of thyroxin—0·3–0·4 mg daily.

If properly treated, prognosis is extremely good. The presence of lymphatic metastases is of no significance, and only in the extrathyroidal tumours and in those which present over the age of 40 is the expectation of life lower than in a control group.

Follicular Carcinoma.—These appear to be macroscopically encapsulated but microscopically there is invasion of the capsule and of the vascular spaces in the capsular region. Follicular tumours in which invasion is minimal are termed ' non-invasive ' and those in which invasion is moderate or marked are termed ' invasive ' (fig. 665). The prognosis depends on the histological picture. ' Non-invasive ' cases have as good a prognosis as the

[1] The term extrathyroidal indicates that the primary tumour has infiltrated through the capsule of the thyroid gland.

Lewis B. Woolner, Contemporary. Pathologist, The Mayo Clinic, Rochester, Minnesota, U.S.A.

intrathyroidal papillary tumours. On the other hand, 50 per cent of patients with ' invasive ' tumours develop a local recurrence or secondaries in bone or lung, and have a mean survival time after surgery of six years.

Because multiple foci are rare, wide excision, as by lobectomy, is good treatment, and the advantage of total thyroidectomy over lobectomy is very slight.

	A *Non-invasive*	*B* *Invasive*
Incidence	50%	50%
Apparent encapsulation	All	Most
Lymph spread	V. rare	Uncommon
Blood spread	Uncommon	Common
Death due to growth within 6 years	3/104	51/104

FIG. 665.—Subgroups of follicular carcinoma and their behaviour. (*after Woolner*)

Lymph nodes rarely require excision and, although not particularly hormone dependent, full doses of thyroxin should be given post-operatively.

A whole body scan is advisable after surgery if the tumour is of the invasive type, because blood-borne metastases are likely and if detected treatment can be undertaken at an early stage. In this case ablation of all remaining thyroid tissue, by surgery or by ^{131}iodine, is essential.

Isolated secondaries may be irradiated directly, but ^{131}I therapy offers the only prospect of success when metastases are multiple. All normal thyroid tissue must have been ablated by surgery or by radio-iodine and the patient must be hypothyroid when the isotope is given.

Anaplastic Carcinoma.—Local infiltration is an early feature of these tumours with spread by lymphatics and by the blood stream. They are extremely lethal tumours and survival for more than four years after presentation is most unusual. In most cases death occurs within months rather than within years. An attempt at curative resection is only justified if there is no infiltration through the thyroid capsule and no evidence of metastases. Radiotherapy should be given in all cases and may provide a worthwhile period of palliation.

Medullary Carcinoma.—These are tumours of the parafollicular (C-cells) which are derived from the ultimo branchial body and not from the cells of the thyroid follicle as are other primary thyroid carcinomas. The cells are not unlike those of a carcinoid tumour and there is a characteristic amyloid stroma. High levels of serum calcitonin[1] are produced by many medullary tumours. These levels fall after resection of a tumour and will rise again if the tumour recurs. Diarrhœa is a feature in 30 per cent of cases and this may be due to 5HT or prostaglandins produced by the tumour cells.

Some tumours are familial and present early in life: the family is often of Scandinavian origin and the tumour may be associated with phæochromatocytomas and with a parathyroid adenoma or hyperplasia (i.e. the M.E.N.[2]

[1] Calcitonin, undetectable in the serum of most normal individuals, is never above 0·1 ng/l.
[2] M.E.N. = Multiple Endocrine Neoplasia

syndrome). Sporadic cases occur at any age and are sometimes associated with pigmented areas in the skin, and neuromas of the skin, tongue and mucosa of the lip and cheek: in some cases there is bone dysplasia. Involvement of lymph nodes occurs in 50–60 per cent of cases and blood borne metastases are common. As would be expected, tumours are not hormone dependent and do not take up radio-iodine. The course of the tumour is unpredictable: in general, life expectancy is good as long as metastases are confined to the cervical lymph nodes and poor once blood borne metastases are present. Treatment is by total thyroidectomy and resection of involved lymph nodes. Because calcitonin is a marker for this tumour, close relatives of patients with medullary carcinoma should be screened by estimating the serum calcitonin level. If serum calcitonin is present a prophylactic thyroidectomy should be carried out.

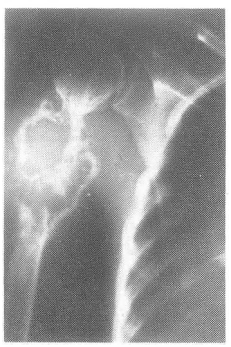


FIG. 666.—Metastasis from a carcinoma of the thyroid in a humerus. (*Dr. S. Devadatta, Vellore, South India.*)

Malignant Lymphoma.—In the past many malignant lymphomas were diagnosed as small round cell anaplastic carcinomas. Response to irradiation is good and radical surgery is unnecessary once the diagnosis is established by biopsy. The prognosis is good if there is no involvement of cervical lymph nodes. Rarely is the tumour part of widespread malignant lymphoma disease and the prognosis in these cases is worse.

Ætiology of Malignant Thyroid Tumours.—Papillary carcinoma has frequently followed accidental irradiation of the thyroid in childhood—never in the adult. The incidence of follicular carcinoma is high in endemic goitrous areas, possibly due to T.S.H. stimulation. Malignant lymphomas sometimes develop in auto-immune thyroiditis so that the lymphocytic infiltration in the auto-immune process may be an ætiological factor.

Clinical Features.—The annual incidence is about 20 per million of the population[1] and the sex ratio 3 females to 1 male. The commonest presenting symptom is a goitre and a 5-year history is far from uncommon in differentiated growths. Recurrent nerve paralysis, enlarged cervical lymph nodes, pathological fractures (fig. 666), or a pulsating bone tumour (fig. 667), may also be the presenting feature.

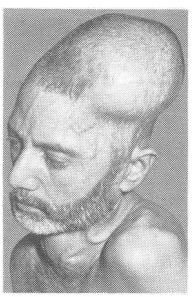


FIG. 667.—Metastasis in the left parietal bone from a carcinoma of the thyroid. (*Professor A. K. Toufeeq, Lahore, Pakistan.*)

Anaplastic growths are usually hard, irregular and infiltrating. A differentiated carcinoma is usually suspiciously firm and irregular, but may resemble a simple nodule. Small papillary tumours may be impalpable—

[1] In the Annual Report of the Chief Medical Officer, Ministry of Health (1965), thyroid carcinoma accounted for 298 female and 86 male deaths. As a registered cause of death the incidence is approximately that of carcinoma of the tongue and of ulcerative colitis.

even when lymph metastases are present.[1] Pain, often referred to the ear, is frequent in infiltrating growths.

Diagnosis.—Diagnosis is obvious on clinical examination in most cases of anaplastic carcinoma, although Riedel's thyroiditis (p. 641) is almost indistinguishable. The localised forms of granulomatous thyroiditis and lymphadenoid goitre may simulate carcinoma. It is not always easy to exclude a carcinoma in a multinodular goitre, and solitary nodules, particularly in the young male, are always suspect. Failure to take up radioiodine is characteristic of almost all thyroid carcinomas,[2] but occurs also in degenerating nodules and all forms of thyroiditis. Thyroid antibody titres are often raised in carcinoma. Thus, no diagnostic test is certain, and exploration with excisional biopsy and frozen section is essential when in doubt. Incisional biopsy may cause seeding of cells and local recurrence, and is most inadvisable in a resectable carcinoma. In an anaplastic and obviously irremovable carcinoma, however, incisional or needle biopsy is justified.

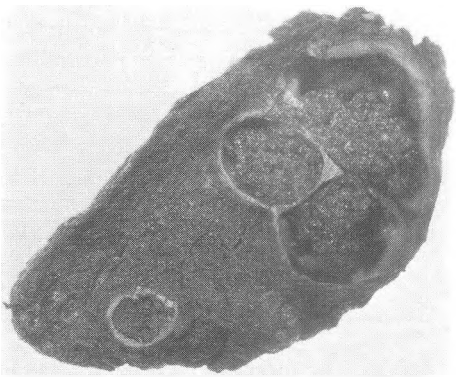


FIG. 668.—Papilliferous carcinoma of the thyroid, showing an intra-glandular seedling. This specimen demonstrates how necessary it is to remove the whole lobe, if not the whole gland. (*F. F. Rundle, F.R.C.S., Sydney, Australia.*)

Histological Surprise.—Not infrequently a differentiated carcinoma of the thyroid is unsuspected until the histological report on the nodule is received several days after thyroidectomy. If the tumour is a follicular carcinoma, further surgery is rarely necessary if the nodule has been excised with a healthy margin of thyroid tissue. However, if it is a papillary carcinoma re-exploration with total thyroidectomy and excision of juxta-thyroid lymph nodes is essential if multiple foci are present in the resected lobe, because their presence indicates intrathyroidal lymphatic spread and a likelihood of foci in the opposite lobe. If the growth is solitary, conservative treatment as for a follicular carcinoma is justified as the risk of local recurrence can be accepted provided that a careful follow up is carried out. In all cases long-term observation and full replacement doses of thyroxin are essential.

Local Recurrence of Differentiated Carcinoma

This may be due to—1. Tumour foci in the thyroid remnant if total thyroidectomy was not performed. 2. Tumour in affected juxta-thyroid nodes not cleared at the initial operation. 3. Spillage of cells from an ill-advised incisional biopsy.

Recurrence may occur 10 or more years after the initial operation.

Total Thyroidectomy

Near-total is a better term than total thyroidectomy, because surgical removal of all thyroid tissue is almost impossible and a little thyroid tissue may have to be left to protect the parathyroid artery. At least one parathyroid gland should be identified and preserved: this is usually the superior gland on the side opposite to the primary

[1] So called Lateral Aberrant Thyroid (p. 642).

[2] Only very rarely will differentiated carcinoma (primary or secondary) take up ^{131}I in the presence of normal thyroid tissue.

growth. The risks of post-operative complications—recurrent nerve injury and parathyroid insufficiency—have been exaggerated, and do not contraindicate total thyroidectomy when it is advisable.

THYROIDITIS

1. Granulomatous Thyroiditis. 2. Auto-immune Thyroiditis. 3. Riedel's Thyroiditis.

1. **Granulomatous Thyroiditis** (Subacute Thyroiditis—de Quervain's Thyroiditis). This is due to a virus infection.[1] In a typical subacute presentation there is pain in the neck, fever, malaise and a firm, irregular enlargement of one or both thyroid lobes. There is a raised E.S.R., absent thyroid antibodies, the serum T^4 is a high normal or slightly raised, and the ^{131}I uptake of the gland is low. The condition is self-limiting and in a few months the goitre has subsided: subsequent hypothyroidism is rare. In 10% of cases the onset is acute, the goitre very painful and tender and there may be symptoms of hyperthyroidism. 35% of cases are asymptomatic but for the presence of the goitre. If diagnosis is in doubt, it can be confirmed by the rapid symptomatic response to prednisone: but if symptomless a biopsy may be necessary. The specific treatment for the acute case with severe pain is to give prednisone—10 to 20 mg. daily—for seven days and the dose is then gradually reduced over the next month.

2. **Auto-immune Thyroiditis.**—This is much more common than was previously thought. It is usually associated with raised titres of thyroid antibodies (see table) and not infrequently there is a family history of other auto-immune diseases, e.g. pernicious anæmia, auto-immune gastritis, vitiligo and thyrotoxicosis.

Classification.—1. Focal thyroiditis. 2. Diffuse non-goitrous thyroiditis (Primary Myxœdema, p. 618). 3. Diffuse goitrous thyroiditis (Hashimoto's disease).[2]

Focal Thyroiditis is commonly seen on histological examination in association with other thyroid disease—notably toxic goitre. Its presence is a warning that post-operative hypothyroidism is likely.

Diffuse Goitrous Thyroiditis.—The histological picture is of interfollicular plasma cell and lymphocytic infiltration, lymph follicles, a varied epithelial change—ranging from destruction to hyperplasia, and a tendency to eventual fibrosis.

THYROID ANTIBODIES

Site of Antigen	*Antibody*	*Test*	*Significant Titres*
Thyroid Cytoplasm	Microsomal Antibody	1. Complement Fixation Test (C.F.T.) 2. Immunofluorescent.	1 in 64
Thyroid Colloid	1. Thyroglobulin Antibody	1. Precipitin	+VE ≡ 1:25,000 T.R.C.
		2. Tanned Red Cell (T.R.C.)	1:2,500
	2. Second Colloid Antibody.	Immunofluorescent	

Thyroid antibodies may be present in normal persons and in those with goitres of all varieties. Low titres are not diagnostic of auto-immune disease. Tests normally available are C.F.T., T.R.C. and Precipitin.

[1] An epidemic reported from Israel was due to a mumps virus.

[2] The concept of Hashimoto's disease as an auto-immune thyroiditis was first defined by Roitt and his co-workers in 1956.

Fritz de Quervain, 1868–1940. Professor of Surgery, Berne, Switzerland. Described this form of thyroiditis in 1902.
Hakaru Hashimoto, 1881–1934. Director of the Hashimoto Hospital, Mie, Japan. Described this disease in 1912.
Ivan Maurice Roitt, Contemporary. Professor of Immunology, Middlesex Hospital, London.

Clinical Features.—As might be expected from the varied histological picture (above), the onset, the thyroid status and the type of goitre vary profoundly from case to case. The onset may be insidious and asymptomatic, or so sudden and painful that it resembles the acute form of granulomatous thyroiditis. Mild hyperthyroidism may be present initially, but hypothyroidism is inevitable and may develop rapidly or extremely slowly. The goitre is usually lobulated, and may be diffuse or localised to one lobe. It may be large or small, and soft, rubbery or firm in consistency—depending upon the cellularity and the degree of fibrosis. The disease is commonest in women at the menopause, but may occur at any age. Papillary carcinoma and malignant lymphoma are very occasionally associated with Hashimoto goitre.

Diagnosis. — Chemical tests of thyroid function vary with the thyroid status and are of diagnostic value only if hypothyroidism is present. A radioactive iodine uptake test is often helpful.[1] Significantly raised titres of one or more thyroid antibodies are present in over 85 per cent of cases. Nevertheless, differential diagnosis from nodular goitre, carcinoma and malignant lymphoma of the thyroid is not always easy and needle or open biopsy may be necessary (fig. 669).

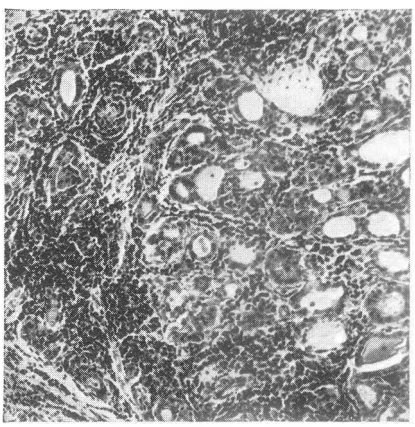


FIG. 669. — Hashimoto's disease (struma lymphomatosa). × 100. Intense lymphocytic-plasma cell infiltration, acinar destruction, and fibrosis. Compare with the normal histology (fig. 653). (*Dr. Bernard Fox, London.*)

Treatment. — Full replacement dosage of thyroxine should be given in every case, because eventual hypothyroidism is inevitable, and because some early Hashimoto goitres (under T.S.H. stimulation) may subside with hormone therapy. Subtotal thyroidectomy may be necessary if the goitre is large and causes discomfort.

3. Riedel's Thyroiditis

This is very rare, accounting for 0·05% of goitres. Thyroid tissue is replaced by cellular fibrous tissue which infiltrates through the capsule into adjacent muscles, paratracheal connective tissue and carotid sheath. It may occur in association with retroperitoneal and mediastinal fibrosis and is most probably a collagen disease. The goitre may be unilateral or bilateral and is very hard and fixed. The thyroid profile and radio-iodine uptake are within normal limits and the scan shows no uptake over the tumour. The differential diagnosis from anaplastic carcinoma can only be made with certainty by biopsy when a wedge of the isthmus should also be removed to free the trachea. If unilateral, the other lobe is usually involved later and subsequent hypothyroidism is common.

[1] A low uptake by the thyroid at 4 hours and 48 hours together with a high 48-hour P.B.^{131}I. in the absence of a previous thyroidectomy or treatment with radio-active iodine is very suggestive.

Bernhard Moritz Carl Ludwig Riedel, 1846–1916. Professor of Surgery, Jena, Germany. Described this form of thyroiditis in 1896.

ECTOPIC THYROID AND ANOMALIES OF THE THYROGLOSSAL TRACT

Ectopic and Aberrant Tissue

Some residual thyroid tissue along the course of the thyroglossal tract is not uncommon, and may be lingual, cervical or intra-thoracic. Very rarely the whole gland is ectopic.

Lingual Thyroid.—Forms a rounded swelling at the back of the tongue at the foramen cæcum (fig. 670) and it may represent the only thyroid tissue present. It may cause dysphagia, impairment of speech, respiratory obstruction or hæmorrhage. It is best treated by full replacement with l-thyroxin when it should get smaller, but excision or ablation with radio-iodine is sometimes necessary.

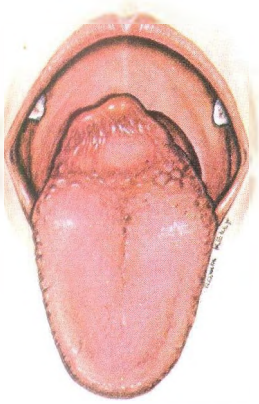


FIG. 670.—Lingual thyroid. *(H. Wapshaw, F.R.C.S., Glasgow.)*

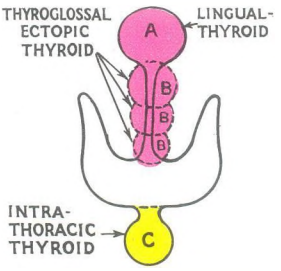


FIG. 671.—Ectopic and aberrant thyroids. A and B are ectopic thyroids, C intrathoracic aberrant thyroid (nearly always acquired).

Median (Thyro-glossal) Ectopic Thyroid.—Forms a swelling in the upper part of the neck (fig. 671B) and is usually mistaken for a thyroglossal cyst. Again, this may be the only normal thyroid tissue present.

Lateral Aberrant Thyroid.—There is no evidence that aberrant thyroid tissue ever occurs in a lateral position (Willis). 'Normal Thyroid Tissue' found laterally, separate from the thyroid gland, must be considered and treated as a metastasis in a cervical lymph node from an occult thyroid carcinoma, almost invariably of papillary type.

Struma Ovarii.—Is not ectopic thyroid tissue, but part of an ovarian teratoma. Very rarely carcinogenic change occurs or hyperthyroidism develops.

Anomalies of the Thyroglossal Tract

Thyroglossal Cyst.—May be present in any part of the thyroglossal tract (fig. 672). The common situations, in order of frequency, are beneath

Rupert Allan Willis, 1898–1980. Professor of Pathology, Leeds, England.

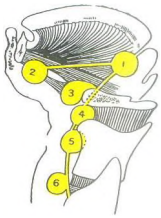


FIG. 672.—Possible sites of a thyroglossal cyst: (1) Beneath the foramen cæcum. (2) In the floor of the mouth. (3) Suprahyoid. (4) Subhyoid. (5) On the thyroid cartilage. (6) At the level of the cricoid cartilage.

the hyoid (fig. 673), in the region of the thyroid cartilage (fig. 674), and above the hyoid bone (fig. 675). Such a cyst occupies the middle line, except in

FIG. 673. — Subhyoid thyroglossal cyst. The commonest variety.

FIG. 674.—Thyroglossal cyst in relation to the thyroid cartilage.

FIG. 675.—Suprahyoid thyroglossal cyst.

the region of the thyroid cartilage, where the thyroglossal tract is pushed to one side, usually to the left.

A thyroglossal cyst should be excised because infection is inevitable (fig. 676) due to the fact that the wall contains nodules of lymphatic tissue which

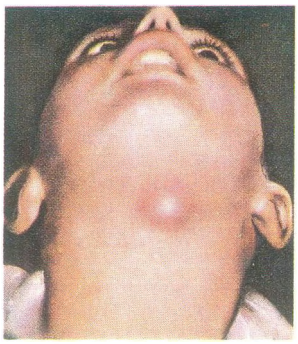


FIG. 676.—Inflamed thyroglossal cyst.

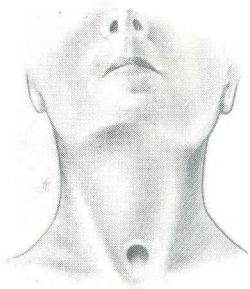


FIG. 677.—A long-standing thyroglossal fistula. The semilunar fold is characteristic.

communicate by lymphatics with the lymph nodes of the neck. An infected cyst is often mistaken for an abscess and incised, which is one way in which a thyroglossal fistula arises.

Thyroglossal Fistula.—Is never congenital: it follows infection or inadequate removal of a thyroglossal cyst. Long-standing fistulæ are inclined to be situated low down in the neck, and fig. 677 shows an example that had been present for twenty years. The hood of skin, with its concavity downwards (due to uneven rates of growth of the neck as a whole and that of the thyroglossal tract), is characteristic. A thyroglossal fistula is lined by columnar epithelium, discharges mucus, and is the seat of recurrent attacks of inflammation.

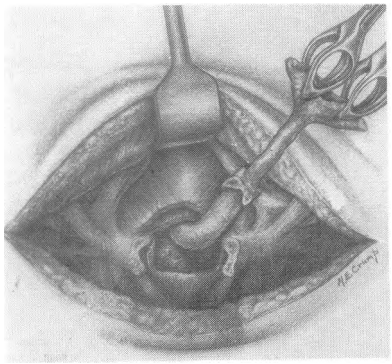


FIG. 678.—Complete extirpation of the thyroglossal tract. A portion of the body of the hyoid bone has been removed, and the dissection is proceeding towards the foramen cæcum.

Treatment.—Because the thyroglossal tract is so closely related to the body of the hyoid bone, this must be excised, together with the cyst or fistula, or recurrence is certain. If the thyroglossal tract can be traced upwards towards the foramen cæcum, it must be excised with a central core of lingual muscle (Sistrunk's operation) (fig. 678).

Walter Ellis Sistrunk, Jr., 1880–1933. Professor of Clinical Surgery, Baylor University College of Medicine, Dallas, Texas, U.S.A.

CHAPTER 36

THE PARATHYROID AND ADRENAL GLANDS

THE PARATHYROID GLANDS

Anatomy and Physiology.—The parathyroid glands, 4 in number are small, oval in shape, about 0·5 to 1·0 cm in size, yellowish-brown in colour and arranged in pairs —closely applied to the thyroid gland, but outside its capsule. The upper pair are fairly constant in position and found on the posterolateral border of the thyroid immediately above the point of entry of the inferior thyroid artery. The lower pair, more variable in position, are usually found at the lower pole of the thyroid, but may be found anywhere along a line from this situation downwards to the upper pole of

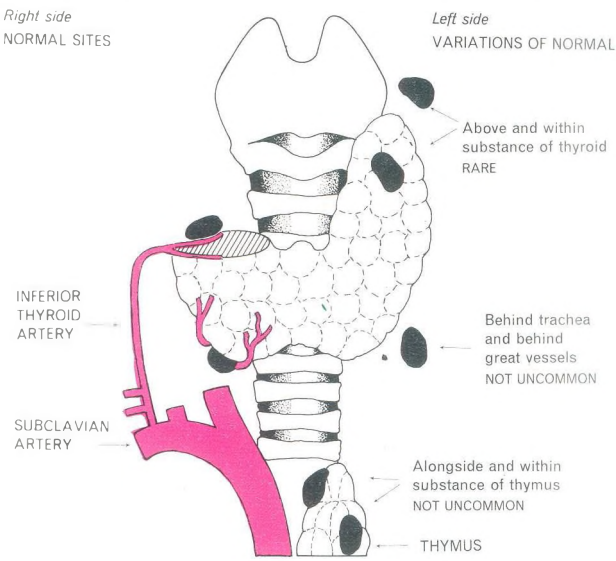


FIG 679.—Parathyroid gland. Normal and abnormal sites.

the thymus (fig. 679). Approximately 5% of parathyroid glands are found within the upper anterior mediastinum. Each gland has a smooth capsule and is supplied by a single leash of blood vessels which can be clearly seen running in the subcapsular plane (fig. 680).

Histology.—The stroma consists of a rich sinusoidal capillary network with islands of secretory cells interspersed with fat cells. The glandular cells are of two types. The 'chief' or 'principal' cells are small with vesicular nuclei and poorly staining cytoplasm. 'Water-clear' cells, derived from the chief cells, are found in the hyperplastic and neoplastic glands. The 'oxyphil' cells are less numerous and larger, with granular cytoplasm and deeply staining nuclei.

Function.—The chief cells of the parathyroids produce parathormone, the hormone being released directly into the blood-stream. The circulating level of parathormone can be measured by radio-immuno-assay. It is sufficiently reliable to distinguish between high and low levels. Facilities for obtaining the estimation are available everywhere in the United Kingdom through the Supra-Regional Assay Service.

Parathormone.—(i) Stimulates osteoclastic activity, thereby increasing bone resorption by mobilizing calcium and phosphate.

(ii) Increases the re-absorption of calcium by the renal tubules, thus reducing the urinary excretion of calcium.

(iii) Augments the absorption of calcium from the gut.

(iv) Reduces the renal tubular re-absorption of phosphate, thus promoting phosphaturia.

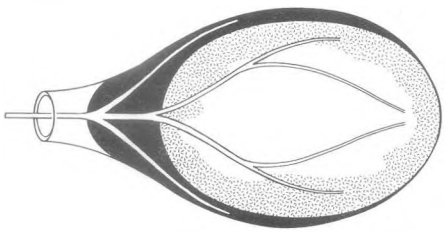


FIG. 680.—The blood supply of a parathyroid gland.

Calcitonin (Copp), was once thought to be the second parathyroid hormone, but is now known to be secreted by the thyroid (Thyrocalcitonin). It lowers the serum calcium and effects calcium storage in bones; quite the opposite action to parathormone.

HYPOPARATHYROIDISM

Parathyroid tetany is a rare complication of subtotal thyroidectomy (less than 1%). At this operation some of the parathyroid glands may be removed or have their blood supply temporarily embarrassed. Symptoms usually appear on the second or third postoperative day, and are temporary. Milder forms of hypoparathyroidism have been described in the follow-up of thyroidectomised patients. Permanent hypoparathyroidism, most commonly encountered following radical thyroidectomy for cancer, requires constant supervision and treatment. Tetany in the new-born may occur within the first few days of life in the child born of a mother with undiagnosed hypoparathyroidism.

Clinical Features.—The first symptoms are tingling and numbness in the face, fingers and toes. In extreme cases cramps in the hands and feet are very painful; the extended fingers are flexed at their metacarpophalangeal joints, with the thumb strongly adducted. The toes are plantar flexed and the ankle joints extended—the so-called carpopedal spasm. Spasm of the muscles of respiration not only result in pain and stridor, but dread of suffocation. In infancy, the symptoms of tetany may be mistaken for epilepsy, though there is no loss of consciousness.

Latent tetany may be demonstrated by:

(i) *Chvostek's sign.*—Tapping over the branches of the facial nerve at the angle of the jaw will produce twitching at the corner of the mouth, the ala of the nose and the eyelids.

(ii) *Trousseau's sign.*—A sphygmomanometer cuff applied to the arm and inflated above the systolic blood pressure for not more than two minutes will produce carpal spasm (fig. 681).

FIG. 681.—The 'obstetrician's hand' seen in parathyroid tetany.

Treatment.—In acute cases the symptoms can be quickly and effectively relieved by the slow intravenous injection of 10–20 ml of a 10% solution of calcium gluconate, and this may be repeated as required until the patient's circulating calcium level has been stabilised. Absorption of calcium is encouraged by the oral administration of Calciferol (vitamin D_2), and calcium lactate. An initial dose of 400,000 units of Calciferol may be followed by 100,000 units daily for

Douglas Harold Copp, Contemporary. Professor of Physiology, University of British Columbia, Vancouver, B.C., Canada.
Frantisek Chvostek, 1835–1884. Physician, Josefsakademie, Vienna.
Armand Trousseau, 1801–1867. Physician Hôtel Dieu, Paris.

several days, according to the patient's need. The only reliable guide to dosage is the level of the serum calcium, which should be estimated daily.

HYPERPARATHYROIDISM

Symptoms of overactivity of the parathyroid glands may result from single or multiple adenomas, hyperplasia of all 4 glands, or carcinoma. A single adenoma is the commonest finding (multiple in 6%). The whole gland is usually considerably enlarged, darker in colour, firmer and more vascular than normal; in some a rim of normal parathyroid tissue can be seen surrounding the adenoma. The histological appearances are the same as in hyperplasia, with a predominance of chief and water-clear cells. Carcinoma of the parathyroids is extremely rare, less than 1%. It tends to invade locally and recur after operation. Blood-borne metastases have been described.

A parathyroid adenoma may occasionally co-exist with secreting adenomas, or hyperplasia of the pituitary, the pancreas or adrenal glands. This association is known as *multiple endocrine adenomatosis*, and may account for a most unusual pattern of symptoms.

Clinical Features.—Hyperparathyroidism, rarely found in the first decade of life, is commoner in women than men, and most commonly found between the ages of 20 and 60 years. The clinical features vary enormously, even when the biochemical changes are similar. The disease has been described as one of *bones, stones, abdominal groans and psychic moans.*

Bone Disease.—There may be generalised decalcification of the skeleton, as in osteitis fibrosa cystica (von Recklinghausen's disease), single or multiple cysts, or pseudotumours of any bone. The latter are particularly common in the jaw bones. Early X-ray changes first appear in the skull and in the phalanges, with loss of density and subperiosteal erosions. Many patients presenting with vague pains in the bones and joints are mistakenly diagnosed as rheumatism.

Renal Stones.—The formation of renal stones is much the commonest result of hyperparathyroidism, and this diagnosis must be considered in every patient presenting with renal tract stone or nephrocalcinosis (fig. 682), and even in those cases of renal colic where no stone can be demonstrated.

Dyspeptic Cases.—Patients with nausea, vomiting and anorexia are relatively common. Peptic ulcer and pancreatitis are not infrequently found in association with hyperparathyroidism, but the relationship is not as yet fully understood.

Psychiatric Cases.—Are not uncommon; women, complaining of tiredness, listlessness and with obvious personality changes, are often wrongly labelled 'neurotic' or 'menopausal'. Patients have been admitted to mental institutions because of irrational behaviour.

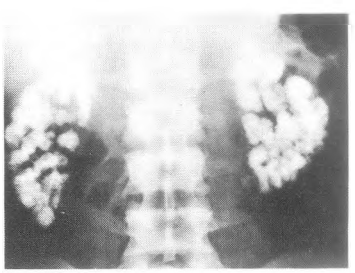


FIG. 682.—Nephrocalcinosis.

Acute Hyperparathyroidism.—This diagnosis is difficult and only too often made

Friedrich Daniel von Recklinghausen, 1833-1910. Professor of Pathology, Strasbourg, France.

after death. Nausea and abdominal pain is followed by severe vomiting, dehydration, oliguria, and finally coma. The serum calcium is very high, up to 17 mg/100 ml (4·2 mmol/l) or more. Treatment is urgent.

Asymptomatic Cases.—Since the advent of routine biochemical screening of all patients attending hospital, more and more cases of hyperparathyroidism fall into this group.

Clinical Examination and Investigation.—Except in severe bone cases with skeletal deformities, swellings or pathological fractures, clinical examination is usually negative. A parathyroid tumour is rarely palpable in the neck. The diagnosis is confirmed with the following biochemical findings:

(i) Elevation of serum calcium (upper limit of normal 10·9 mg/100 ml (2·6 mmol/l)).
(ii) Diminution of serum phosphorus (lower limit of normal 3·0 mg/100 ml (0·8 mmol/l)).
(iii) Increased excretion of calcium in the urine (upper limit of normal 250 mg (6·2 mmol) per 24 hours for females, 300 mg (7·5 mmol) for males).
(iv) Elevation of the serum alkaline phosphatase in bone cases.
(v) Elevation of serum parathyroid hormone concentration (upper limit of normal 0·5μg/l. Note that parathyroid hormone is always detectable in patients with hyperparathyroidism, in many normal people it is undetectable.

Differential Diagnosis.—Other cases of hypercalcæmia must be remembered and excluded. They are:

(i) Secondary cancer in bone (breast, prostate, bronchus, kidney and thyroid).
(ii) Carcinoma with endocrine secretion (bronchus, kidney and ovary).
(iii) Multiple myeloma.
(iv) Vitamin D intoxication.
(v) Sarcoidosis.
(vi) Thyrotoxicosis.

The differential diagnosis presents *no* problem if the parathyroid hormone level is estimated. In none of the above-mentioned conditions will parathyroid hormone be detectable in the blood.

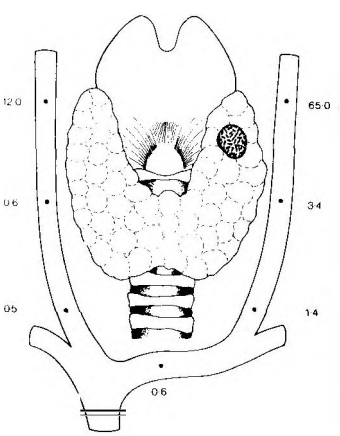


FIG. 683.—Selective venous sampling of parathormone levels to detect site of tumour.

Treatment.—The only corrective treatment is surgical removal of the overactive gland or glands.

Pre-operative treatment is not usually necessary. In acute cases, rapid correction of dehydration and electrolyte imbalance is necessary, with a careful daily check on the serum calcium. Where there is extensive skeletal decalcification, the patient should be given one tablet of calcium lactate and 500 international units of vitamin D daily for a few days prior to operation, and for several weeks afterwards to prevent postoperative tetany.

As there is no pre-operative method for determining the likely pathology, the surgeon at

operation must identify all 4 glands, study each carefully, both macroscopically, and, if need be, microscopically after biopsy. Adenomas, single or multiple, are removed; in hyperplasia three glands should be removed with half of the fourth; in carcinoma, a radical local resection is required.

In approximately 10% of cases even the most experienced surgeon in this field may find difficulty in locating a parathyroid adenoma. Selective venous sampling with estimation of parathyroid hormone levels at these points is of great help to the surgeon in locating the tumour, especially after a first neck exploration has failed (fig. 683).

Prognosis.—With successful surgery, bones will recalcify and pseudotumours resolve. Renal stones will not disappear, but the incidence of recurrence after surgical removal is reduced, and deterioration in renal function is prevented. Psychiatric patients show an early, and often remarkable, recovery. In a small minority of cases hyperparathyroidism recurs after several years, and may warrant further surgery.

Secondary Hyperparathyroidism.—In patients with a low serum calcium, such as one finds in *chronic renal failure* and *malabsorption syndromes*, there is a compensatory parathyroid hypertrophy affecting all 4 glands. Few of these will require surgical treatment and only when bone pain and skeletal decalcification is severe.

THE ADRENAL GLANDS

Surgical Anatomy.—At birth, the adrenal glands have attained nearly adult proportions. Fully developed, each weighs about 4 g, but the left is a little larger than the right. A deeper yellow colour and a firmer consistency enables the gland to be distinguished from the adjacent fat. Each rests on the superior, anterior, and medial aspects of the superior pole of the corresponding kidney, and presents the appearance of a French Liberty cap worn at a rakish angle.

Although intimately related anatomically, the adrenal cortex and the adrenal medulla are quite separate internal secretory glands.

The adrenal glands are supplied by several adrenal arteries, rendering them remarkably vascular, but only one vein drains each gland. On the right side the adrenal vein is short and enters the inferior vena cava just distal to the hepatic vein, while on the left it empties into the left renal vein (which communicates through the azygos vein with the left intercostal internal mammary, and vertebral veins (Anson)). This dissimilarity of the right and left venous flow determines, to some extent, the location of metastases from malignant tumours of these glands (p. 657).

Diagnostic Radiology

(i) Non-invasive methods of detecting adrenal swellings are isotope scanning and CT scanning.

(ii) Invasive techniques include selective retrograde venography (especially for left-sided tumours) and selective arteriography.

(iii) *Calcification* in an adrenal gland is difficult to interpret in a radiograph. It is liable to be confused with a renal calculus. Areas of calcification in the adrenal glands may be present in Addison's disease (p. 652).

THE ADRENAL CORTEX

The adrenal cortex is made up of the following layers from without inwards; the zona glomerulosa, the zona fasciculata, and the zona reticularis.

Physiology.—At least fifty steroid compounds have been isolated from the adrenal cortex. These hormones exhibit various types of activity which, for practical purposes, can be arranged in three groups.

Barry J. Anson, Contemporary. Research Professor, Department of Otolaryngology and Maxillofacial Surgery College of Medicine, University of Iowa, Iowa City, U.S.A.

1. **Salt Regulating Hormones** which are concerned in the maintenance of water and electrolytic balance. A deficiency of these hormones produces sodium diuresis, potassium retention and dehydration; an excess results in hypertension, œdema, cardiac dilatation, and hypopotassæmia. **Aldosterone** is the most important of these ' salt regulating ' hormones (see Conn's syndrome, p. 655).

2. **The cortisones** are concerned with the metabolism of proteins and carbohydrates, favouring the formation of the latter from the body's storehouse of the former. This conversion is known as gluconeogenesis. Consequently hormones belonging to this group are called glucocorticoids. The best known of these are **hydrocortisone** (also known as **cortisol**), and **cortisone** (which is converted in the body to hydrocortisone). The therapeutic application of these hormones falls into two headings:

(*a*) *In Endocrine Deficiencies.*—Cortisone is the logical need in adrenocortical insufficiency (p. 652) and after bilateral adrenalectomy (p. 656).

(*b*) *In Non-endocrine Disease.*—Cortisone is used in the treatment of a diversity of diseases, including allergic conditions, granulomatous disorders, blood diseases and the collagenoses. Hydrocortisone is used in the treatment of hypocorticism and shock (p. 82) and is an effective anti-allergic agent in a number of skin diseases and eye conditions.

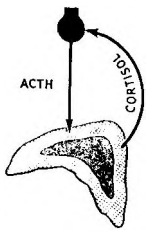


FIG. 684.—Pituitary ACTH stimulates the adrenals. Cortisol of the adrenals inhibits the output of ACTH.

3. **Sex Hormones.**—Androgenic and œstrogenic hormones are produced by the adrenal cortex. Excessive secretion of androgens due to hydroylase or hydrogenase deficiencies causes virilism in females or, rarely, excessive secretion of œstrogens brings about effeminacy in males.

Inter-hormonic Action.—The anterior lobe of the pituitary gland secretes adrenocorticotropic hormone (ACTH) which stimulates the adrenal cortex, whereas the cortisol of the adrenal cortex inhibits the secretion of ACTH (fig. 684).

Tests of Adrenal Cortical Activity

(i) *Estimation of the 24-hour output of urinary steroids (cortisol metabolites).*—The 17-oxogenic steroid and the 17-oxosteroid excretions are similar—10–20 mg/h (35–70 μmol/24h)[1] for men, 5–15 mg (17–42 μmol/24h)[1] for women, and 2–4 mg (7–14 μmol/24h) for a child of six to eleven years. Adult levels are reached at about eighteen years. The average excretion is lower in coloured than in white males. After the age of forty, urinary steroid excretion gradually falls and at seventy is approximately halved.

(ii) *Plasma cortisol level.*—Normally 8–28 μg/100 ml (220/780 nmol/l).

(iii) *Metyrapone test.*—Normally the levels in (i) and (ii) are increased but not if the adrenals or pituitary are diseased.

(iv) *ACTH stimulation test.*—A single intramuscular injection of 250 μg of a synthetic corticotropin (Synacthen) is given. Normally this will double the plasma cortisol level in the space of thirty minutes. Patients with adrenal hyperplasia are said to be very responsive and may give a level of 20–60 mg/24h (70–210 μmol/24h) above the prestimulation control levels of the two urinary steroids mentioned in (i).

(v) *Adrenal suppression.*—Injections of cortisol or dexamethasone will suppress adrenal function (fig. 684), as shown by the depression of the urinary steroids, especially in patients with adrenal hyperplasia. Adrenal tumours, however, are *autonomous*, so the excretion of urinary steroids in these cases is not suppressed.

It must be remembered that, as a rule, the result of each test alone is not a clear indication of the state of the adrenals, and therefore any conclusions must be drawn from the results of all the tests.

Disorders of Adrenocortical Function

The two most common disorders of adrenocortical function are chronic insufficiency (Addison's disease, p. 652) and chronic over-activity (Cushing's syndrome, p. 653).

[1] Estimated as dehydro-epiandrosterone.

Acute Hypocorticism:

1. **Adrenal Apoplexy in the Newborn.**—Extensive hæmorrhage into one (fig. 685) or both adrenals can be a cause of death in infants within the first few days of birth. The condition may occur after long and difficult labour, and particularly when resuscitative procedures have to be employed to combat asphyxia neonatorum. The hæmorrhage into the adrenals follows necrosis of the innermost layer of the cortex, which always occurs at birth, possibly as a result of sudden withdrawal of the female sex hormone (œstrogen). Adrenal crisis in the newborn produce signs of profound shock. A mass may be palpable in one or both renal regions. Intravenous fluid therapy with hydrocortisone, or, failing the latter, cortisone intramuscularly, offers the only hope.

FIG. 685.—Kidney and adrenal of a newly born infant showing hæmorrhage into the adrenal gland.

2. **Waterhouse-Friderichsen Syndrome.**—Massive bilateral adrenal cortical hæmorrhage, occurs in cases of fulminating meningococcal septicæmia and in some cases streptococcal, staphylococcal, or pneumococcal septicæmia. Most of cases occur in infants and young children, but it can happen in adults. The onset is catastrophic, with rigors, hyperpyrexia, cyanosis, and vomiting. Petechial hæmorrhages into the skin which coalesce rapidly into purpuric blotches are a constant feature. Profound shock follows, and before long the patient passes into coma. The condition is one of overwhelming sepsis that pursues a galloping course, death occurring in most cases within forty-eight hours of the onset of symptoms unless correct treatment is given without delay.

Unilateral hæmorrhage causing a lesser degree of systemic upset and not associated with infections has been described. This type of case resembles a perinephric abscess or other upper abdominal acute condition.

Confirming the Diagnosis.—It is futile to await the result of a blood culture. Bilateral tenderness 5 cm. below the costal margin, clear urine (oliguria is often present), and an absence of signs in the lungs help to call attention to the adrenal glands. In meningococcal infection the diplococcus can be demonstrated by smears obtained from a punctured petechial spot in the skin.

Treatment.—Antibiotic therapy must be given intensively by the intravenous route. In addition, an intravenous infusion of dextrose-saline solution containing hydrocortisone 100 to 200 mg. per litre is given. In the absence of hydrocortisone, cortisone can be given intramuscularly, but its action is slower and less certain. Oxygen should also be administered. Following such treatment, improvement often sets in within three hours, and a number of patients have recovered.

3. **Crises of Infantile Hypercorticism** (p. 652).

4. **Following Bilateral Adrenalectomy.**—If precautions are taken (p. 655), acute hypocorticism is unusual in the post-operative period. In the months that follow, crisis may occur if the correct dose of cortisone replacement is not given. The treatment is to support the blood pressure with D.O.C.A. (desoxycorticosterone acetate, 5 to 20 mg. intramuscularly), and then administer 3 litres of dextrose-saline solution per twenty-four hours, 50 to 200 mg. of hydrocortisone being added to each flask. Failing hydrocortisone, give cortisone intramuscularly.

5. **Postoperative Adrenal Hæmorrhage.**—Adrenal hæmorrhage can be an unexpected cause of deterioration and sudden death in the postoperative period. In some cases the left adrenal gland is damaged during radical gastrectomy for carcinoma (Fox). In other cases when adrenal hæmorrhage is bilateral, there is no evidence of operative injury, they are usually associated with intra-abdominal sepsis, pneumonia, coagulation defects and cancer. Thrombosis of the adrenal veins is the cause of infarction of the glands.

The diagnosis of postoperative adrenal hæmorrhage is difficult. It should be suspected when there is sudden collapse, with hypotension, particularly if associated with upper abdominal pain and tenderness. Other evidence of acute adrenal failure are vomiting, diarrhœa and mental confusion. The most useful laboratory finding

Rupert Waterhouse, 1873–1958. Clinical Pathologist and Physician, Royal United Hospital, Bath, England. Described this syndrome in 1911.
Carl Friderichsen, Contemporary. Formerly Medical Superintendent, Children's Department, Sundby Hospital, Copenhagen. Wrote his account of this syndrome in 1918.
Bernard Fox, Contemporary. Pathologist, Charing Cross Hospital, London.

is a low plasma cortisol, although unexplained blood eosinophilia is very suggestive of adrenal failure. The classical changes of hyperkalæmia and hyponatræmia often take several days to appear and may be obscured by intravenous therapy. If the diagnosis is suspected, then treatment should not be withheld for lack of laboratory confirmation.

Treatment.—A therapeutic trial of at least 100 mg of intravenous hydrocortisone hemisuccinate in 500 ml of isotonic saline as quickly as possible, and repeated in about an hour, is indicated. If there is a positive response then hydrocortisone (50 mg every 6 hours) should be given. When the patient's condition has sufficiently improved, then treatment with a synthetic oral steroid should be substituted. Occasionally there may be a deficiency in mineral corticoids so that treatment with fludrocortisone is necessary. Adrenal hæmorrhage may result in chronic adrenal insufficiency (see below).

Chronic Hypocorticism (Addison's disease)

This is due to adreno-cortical insufficiency consequent upon progressive destruction with lymphocytic infiltration of the zona reticularis, the zona fasciculata, the zona glomerulosa, and the medulla of the adrenal glands, in that order. In about 60% of cases the condition is believed to be due to an autoimmune disease, sometimes in association with Hashimoto goitre (p. 640) and pernicious anæmia. Tuberculosis, metastatic carcinoma and amyloidosis account for the remaining 40%.

Clinical Features.—Addison's disease usually commences in the third or fourth decade. Sometimes it is the terminal event in cases of adrenogenital hyperplasia. The sex distribution is about equal. The leading features are muscular weakness and a low blood pressure. Irregular dusky pigmentation of the skin, due to deposits of melanin, appears at points of pressure (e.g. garter, belt) and in the flexion creases. Pigmentation of mucous membranes, particularly of the mouth, is often striking. When fully established, the course of the disease is punctuated by crises of acute adrenocortical insufficiency (see above).

Confirmatory Diagnostic test.—The 24-hour urinary excretion of the 17-hydroxycorticosteroids by a patient with Addison's disease are usually less than 5 mg./24 hrs if the patient is a man and less than 1 mg./24 hrs in the case of a woman. The excretion fails to increase after ACTH administration (p. 650).

Especially during a crisis, the plasma sodium is decreased, the chlorides low, and the potassium elevated above 5 mEq (mmol)/litre.

Treatment is medical, using cortisone, and relevant cases should include chemotherapy for tuberculosis.

Prognosis.—By the use of cortisone, the expectation of life of a patient suffering from Addison's disease has been extended from up to three years to at least seven years.

Hypercorticism

The various forms of adrenal cortical hyperfunction are classified according to the age of onset. (1) Infantile. (2) Pre-pubertal. (3) Adult, otherwise known as Cushing's syndrome—the commonest type. (4) Post-menopausal. (5) Primary aldosteronism (Conn's syndrome) can occur at any age (p. 655).

1. **Infantile Hypercorticism.**—Androgenic excess during intrauterine life is one form of pseudo-hermaphrodism in the *female child.* The condition is present at birth; sometimes the enlarged clitoris and a varying degree of hypospadias

Thomas Addison, 1793–1860. Physician, Guy's Hospital, London. Described the effects of disease of the suprarenal capsules in 1849.

make it difficult to determine the infant's sex. The 17-ketosteroid content of the urine may be sufficiently elevated to substantiate a diagnosis of a female with adrenal hyperfunction. If this is not the case, it is justifiable to perform sex determination by a skin biopsy before the age of one year. Female pseudohermaphrodism with virilism is invariably associated with disease of the adrenal cortex, usually bilateral hyperplasia of the cortex. Hormonal studies have shown that there is a congenital failure of the adrenal glands to synthesise gluco-corticoids, mainly compound F. Due to this lack, these infants are liable to acute phases of adrenal insufficiency during stress or infection, or to suffer from periodic hypoglycæmic attacks. They need cortisone, not only in the emergency, but as long-term therapy, thereby inhibiting the secretion of excessive androgens. In the absence of such treatment the epiphyses join early, the patients are dwarfed, menstruation does not occur, and the breasts do not develop. These tendencies are corrected by cortisone given orally, 25 mg. or more daily, the dose being determined by 17-ketosteroid estimations (Leonard Simpson). Hirsutism is moderated, but not necessarily abolished. The treatment should be commenced early if good results are to be obtained.

2. **Pre-pubertal Hypercorticism.**—There is never any doubt as to the sex of the infant at birth, and during the very early years of life the child is normal. The symptoms commence about the age of five or six years.

In the Female.—Pubic and axillary hair appear, but there is no gross enlargement of the clitoris. The child is short in stature, the legs being especially stunted, but she looks much older than she is. Puberty is often precocious, menstruation, if it occurs, being scanty. There is a deepening of the voice at this time.

In the Male.—The term ' infant Hercules ' is descriptive. He is extremely short, muscular, and hirsute. The genitalia assume adult proportions, and spermatozoa are often present in the seminal fluid.

In both sexes, 17-ketosteroid content of the urine is increased. A very high reading supports the diagnosis of an adrenocortical tumour, which must always be excluded. In both males and females, with a later onset or the passage of time, the features of Cushing's syndrome become super-added.

Treatment.—This is identical with that of Cushing's syndrome.

3. **Post-pubertal or Adult Hypercorticism (Cushing's syndrome)** is due to an excessive production of glucocorticoids, mainly hydrocortisone (compound F). It is the commonest type of hypercorticism. Bilateral hyperplasia is the cause in some 70% of cases, and in one third of these a basophilic tumour of the anterior pituitary gland—often microscopic in size—is present. An adrenal adenoma accounts for 15% and carcinoma (which can be bilateral) in 5%. In the remainder there is no discernible structural alteration in the glands and the condition is due to an ectopic source of an ACTH-like substance being secreted by an undifferentiated carcinoma of the bronchus, or a tumour of the thymus or ovary. Alcoholism also must be considered.

In its most typical form, Cushing's syndrome is seen in patients treated with large doses of cortisone over long periods for non-endocrine diseases, particularly rheumatoid arthritis, and in patients receiving transplants.

Clinical Features.—The female : male ratio is at least 3 : 1. The great majority of cases (excluding those induced by cortisone therapy) occur in females between fifteen and thirty years of age, in whom it produces highly characteristic features. Although the patient's weight is not necessarily increased, there is a deposition of fat in certain situations. The face becomes rubicund, rounded like a full moon, and the lips are pursed. The abdomen becomes protuberant the neck thick, the supraclavicular fossæ obliterated, and a roll of fat appears over the region of the vertebra prominens

Samuel Leonard Simpson, Contemporary. Consulting Endocrinologist, St. Mary's Hospital, London.
Hercules, one of the most famous of the heroes of Greek mythology, was noted for his prodigious strength.
Harvey Cushing, 1869–1939. Professor of Surgery, Harvard University, Boston, Mass., U.S.A. (1912–1932), (see footnote, p. 75). Described this syndrome in 1932.

(buffalo hump). The arms, and especially the legs, are relatively thin, the muscular development is poor, and the patient complains of increasing weakness. As the disease progresses, so the general contour becomes more and more that of a lemon on match-sticks.→ Consequent upon the inhibitory effect of the hypercorticism on fibrous tissue, the skin becomes of tissue-paper consistency, and inelastic. Exceedingly characteristic are purple-red striæ distentiæ, mostly on the abdomen (fig. 686), of a texture that can be likened to an over-stretched garter. Ecchymoses are frequent and bruising occurs on the slightest trauma. Acne is common and there is a low resistance to skin infections. Often there is increased growth of lanugo hair, but hirsutism is usually absent. Amenorrhœa is usual or, in the male, impotence. Due to a negative calcium balance, the matrix of bone becomes thin, and severe osteoporosis results. Pathological fractures, particularly compression fracture of a vertebra, are common, and this is sometimes the first reason for the patient seeking advice. Mild glycosuria is often present. Hypertension is frequent, and eventually congestive heart failure supervenes. In about 60 per cent. of cases various psychoses occur.

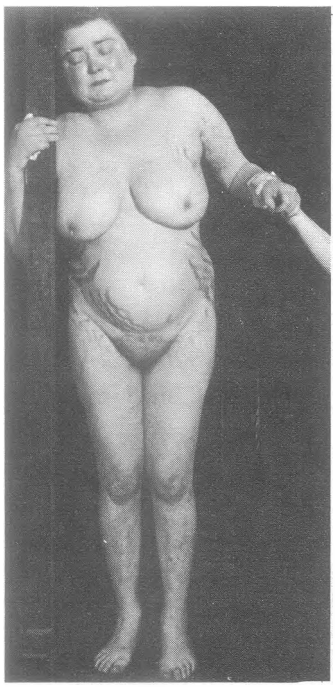


FIG. 686.—Cushing's syndrome in a woman aged twenty-three years. Adrenal hyperplasia. (*Dr. Leonard Simpson, London.*)

Cushing's syndrome is rare in children; when it occurs, the patient is nearly always a female and an adrenal tumour is usually the cause.

A sub-group, probably due to an excessive secretion of adrenal androgens (*adreno-genital syndrome*), commences between the ages of fifteen and twenty-five and is confined to females. One of the first indications of its onset is amenorrhœa or oligo-menorrhœa. There follows an excessive growth of hair on the face (fig. 687), acne, atrophy of the breasts, alteration in bodily contour and muscular development, deepening of the voice, and enlargement of the clitoris. Jewish and Spanish women are more prone to this affliction than those of other races.

FIG. 687.—Adreno-genital syndrome in a woman of twenty-eight. (*Dr. Leonard Simpson, London.*)

Arrhenoblastoma of the ovary.—This rare condition occurs between puberty and the menopause and also causes hirsutism. It may also arise in a suprarenal 'rest'.

Laboratory Findings.—Polycythæmia, lymphopænia, and eosinopænia are common. A fasting

eosinophil count over 30 per ml. of blood is good evidence against the diagnosis. The basal metabolic rate is low and the serum cholesterol is elevated. The dextrose tolerance is impaired, and the insulin tolerance test reveals a resistance to the action of insulin. Urinary 17-ketosteroids are somewhat high, and urinary ketogenic steroids are above normal levels. There is a negative calcium balance and radiography of the skeleton reveals osteoporosis, most marked in the spine and pelvis.

Treatment.—In cases of cortical hyperplasia, total adrenalectomy on one side and resection of seven-eighths of the adrenal gland on the other side (usually after an interval) is the best course. Obviously the only treatment for neoplastic cases is excision of the adrenal gland bearing the tumour.

If a pituitary tumour causes oculomotor symptoms or enlargement of the pituitary fossa as seen on X-ray, it should be removed surgically (p. 464); in other circumstances destruction of the gland can be achieved by the implantation of pellets of radio-active Yttrium 90 (Forrest).

Very rarely, the *adreno-genital syndrome* appears in youths and men. Owing to excessive production of œstrogenic hormones by the adrenal cortex, gynæcomastia, atrophy of the testicles, and psychic signs of effeminacy appear (Adrenal Feminism).

4. **Post-menopausal hypercorticism** is usually characterised by the growth of a beard (the bearded woman of the circus), and is often accompanied by mental aberration. A lesser degree of hirsutes is almost a natural accompaniment of the aging process, particularly in dark-haired females, and it is difficult to draw the line between the normal and the pathological. Thus it is that operative treatment is usually disappointing.

Primary aldosteronism (Conn's syndrome) is due to a rare adreno-cortical tumour producing aldosterone (p. 650). Excess of this leads to sodium retention and a fall in serum potassium. The latter causes the typical features of the syndrome, namely episodes of muscular weakness associated with polyuria and polydipsia. Hypertension may occur. The plasma sodium is high and the potassium is low, but simple administration of potassium does not relieve the condition. Renin and angiotensin levels are depressed. Effective treatment involves removal of the causative tumour, but there is the usual difficulty in ascertaining on which side it lies (see under operation, p. 656).

Secondary aldosteronism is associated with cirrhosis of the liver, and renal artery stenosis with high levels of renin and angiotensin.

ADRENALECTOMY FOR HYPERCORTICISM

It is essential that all patients who are to be subjected to extirpation of adrenal cortical tissue be prepared adequately for the operation, and supported post-operatively by adrenocortical hormone replacement therapy, irrespective of the extent of adrenal resection.

Cortisone Therapy

Pre-operative	Cortisone acetate, 100 mg intramuscularly twice a day for two days. One dose is given two hours before operation.
During the Operation and Immediate Post-operative Period	Hydrocortisone, 100 mg slowly by intravenous drip. Then a similar quantity during the subsequent twelve hours, to be followed by cortisone 50 mg intramuscularly six-hourly. If the blood-pressure remains below 100 systolic, then blood-transfusion and/or noradrenaline is given (p. 657).

Andrew Patrick McEwen Forrest, Contemporary. Regius Professor of Surgery, Edinburgh.
Jerome W. Conn, Contemporary. Professor of Internal Medicine, University of Michigan, Ann Arbor, Michigan, U.S.A.

Post-operative
- **Day 1.**—Cortisone 50 mg intramuscularly six-hourly.
- **Days 2 and 3.**—Cortisone 50 mg intramuscularly eight-hourly.
- **Days 4 and 5.**—Cortisone 50 mg intramuscularly every twelve hours.
- **Days 6 and 7.**—Cortisone 25 mg by mouth every eight hours.

Thenceforward cortisone by mouth should be reduced slowly to maintenance level in cases of total adrenalectomy, or to zero in subtotal adrenalectomy. Fludrocortisone 0·1 mg daily (replacing aldosterone) may be necessary to regulate fluid and salt balance.

N.B.—If hydrocortisone is not available, 200 mg of cortisone instead of 100 mg should be administered intramuscularly twelve hours before operation. Cortisone is cheaper than hydrocortisone.

After total adrenalectomy the patient should always carry a card stating the dosage of cortisone she is receiving. Any stress (a further operation or infection) is an indication to increase the dosage.

Operation: (*a*) When an adrenal tumour has been demonstrated preoperatively, excision of that adrenal gland alone is carried out. (*b*) If a tumour has not been demonstrated, the patient is prepared for bilateral exploration.

Because the difficulties are usually less on the left than on the right side, exploration of the left adrenal gland is undertaken first. If a tumour is found, adrenalectomy is carried out. If the left gland is found to be atrophic, it is highly probable that there is a tumour on the right side, which should be explored forthwith. Should the gland be hyperplastic or normal, subtotal (90 per cent.) adrenalectomy is indicated. If, after this has been performed, it is apparent that the patient will not tolerate a bilateral operation well, exploration of the contralateral side should be postponed.

Technique.—An ample postero-lateral incision, such as is used for exploration of the kidney (Chap. 56), is commonly employed. After subperiosteal resection of the twelfth rib the lower border of the pleura is defined and protected. The incision is extended through the bed of the twelfth rib to reveal the perinephric fat, within which the adrenal gland is identified, as described below. Sometimes an approach through the bed of the eleventh rib, reflecting the pleura upwards is preferred. (See also the abdominal approach, below).

On the right side the suprarenal vein is short and may be torn from the vena cava if it is not identified and ligated at an early stage of the dissection. By finger and gauze dissection, keeping close to the gland, the gland is freed from below and behind, upwards, ligating and dividing bleeding vessels as they are encountered, until it is suspended only by its main vascular pedicle near its apex. If subtotal adrenalectomy is to be performed, the gland is cut across with scissors so as to leave a small triangular fragment of the apex well supplied with blood-vessels. Bleeding should be controlled by swab pressure, as diathermy coagulation leads to necrosis.

Bilateral total adrenalectomy for inoperable carcinoma of the breast or prostate, or their metastases.—In somewhat less than 50 per cent of patients, total adrenalectomy results in a surprising degree of reduction of pain, a feeling of well-being, occasional radiographic evidence of recalcification of osseous metastases, and apparent resorption of metastases in other situations. In the case of carcinoma of the breast, the operation may be combined with removal of both ovaries (p. 684). Hormone therapy is employed before, during, and after adrenalectomy as described above. The majority of patients tolerate a single-stage bilateral adrenalectomy exceedingly well.

Operation.—The anterior route is to be preferred in these cases, and the adrenal glands are approached through either a curved transverse incision or a long paramedian incision. *The left adrenal gland* is approached first by cutting along the lateral leaf of the lieno-renal ligament and then curving downwards and medially, so as to enable a wide peritoneal flap to be reflected. By retracting the spleen downwards and medially, the adrenal gland comes into view. The fascia over its lateral border is incised, and by gauze dissection the blood-vessels of the gland are defined, ligated, and divided, thus freeing the gland, which is removed. Alternative approaches can

be made by an ' up and under ' dissection of the mesocolon and pancreas, or through the lesser sac. *The right adrenal gland* is more deeply situated. The peritoneum is incised lateral to the duodenum and above the upper pole of the kidney. The flap of peritoneum is raised to expose the anterior surface of the adrenal gland as it lies against the bare surface of the liver. The fascia covering the lateral surface of the gland is incised. A finger can then be inserted above the upper pole of the gland into the space between the two layers of fascia enclosing the gland (fig. 688). This prevents the gland from becoming displaced upwards, which otherwise it is prone to do. The anterior fascial layer is then incised transversely and the gland can be dissected under vision, as on the left side. After removal, each gland should be inspected to check its completeness, and each adrenal bed must be searched for the presence of accessory adrenal tissue, which is present in 32 per cent of cases. If this important step is omitted, failure of the operation is not unlikely. The abdomen is closed with small drainage tubes passing to the adrenal fossæ on either side.

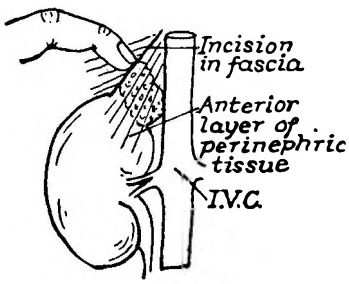


FIG. 688.—Incision in the fascia lateral to the right adrenal gland. Insertion of the finger into the space above the gland prevents its upward displacement. (*After J. C. McKeown and A. Ganguli.*)

THE ADRENAL MEDULLA

The medulla of the adrenal glands (chromaffin tissue), which is developed, together with sympathetic nerves, from ectoderm, is grey in colour and connected intimately, both anatomically and functionally, with splanchnic nerves. Chromaffin tissue is so-called because the large polyhedral cells of which it is composed contain granules that stain yellow with chromic acid. These granules are the internal secretion of the adrenal medulla itself, for they can be observed being extruded *in toto* into radicles of the adrenal vein. The secretion consists of adrenaline and noradrenaline. In health, 80 per cent. of the output is adrenaline, and 20 per cent. is noradrenaline. However, in hyperfunctioning medullary tumour (phæochromocytoma) this ratio is completely reversed. Fear, anger, pain, and effort give rise to an increased output in response to the stimuli received via the splanchnic nerves.

It would appear that one of the principal functions of chromaffin tissue is to liberate quickly hormones that permit a more rapid response of the body to noxious stimuli. Noradrenaline causes an over-all vasoconstriction, thereby raising the blood pressure. Its action is short-lived.

TUMOURS OF THE ADRENAL MEDULLA

Neoplasms of the sympathetic neurones < Ganglioneuroma / Neuroblastoma (sympatheticoblastoma)

Neoplasm of chromaffin cells = Phæochromocytoma

Those occurring at any age:

A ganglioneuroma is relatively benign. This neoplasm is symptomless, grows to a large size, and constitutes one of the varieties of retroperitoneal ' sarcoma ' (Chap. 48). Only 15 per cent. involve the adrenal, the remainder occurring in any position along the sympathetic chain. If removed completely at a comparatively early stage, a cure can be expected.

Those occurring in infants and children:

Neuroblastoma of the adrenal medulla is a reddish-grey tumour that is highly malignant. It soon breaks its confines and invades neighbouring organs, e.g. the kidney and the pancreas, and metastasises by lymphatics, and even more frequently by the blood-stream.

Clinical Features.—Distributed equally between the sexes, 80 per cent. of these comparatively rare tumours occur below the age of five years. Usually the child is

brought on account of an abdominal swelling. Pallor and loss of appetite are frequent accompaniments. The knobbly contour of an adrenal neuroblastoma helps to differentiate it from a Wilms' tumour (Chap. 56), which remains smooth even after it has attained a great size. Although unilateral, the growth, as it enlarges, extends across the middle line. About 60 per cent. of patients have metastases by the time they are seen (fig. 689).

Pepper's type of the disease.—The primary and secondaries are on the right side, with large liver metastases. In *Hutchison's type*, a left-sided primary spreads upwards by lymphatics and deposits are found in the orbit and skull. Metastases in the skull mimic the spicular osteogenic sarcoma; those in the long bones resemble Ewing's tumours. (p. 264)

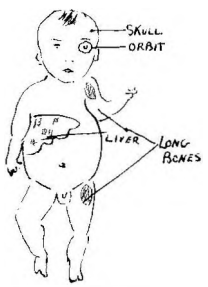


FIG. 689.—The common sites for metastases from neuroblastoma of the adrenal. Bones are involved more frequently than the liver.

Investigations should include a complete radiographic examination of the skeleton (skeletal survey).

Treatment.—Unless there are secondary deposits, exploration (before or after radiotherapy) should be undertaken; in comparatively early cases the tumour can be removed completely. When total removal is not feasible, as much as possible of the neoplasm should be excised, followed later by a course of deep radiotherapy.

If complete excision is possible, the prognosis is enhanced, but an extraordinary feature of these cases is that from time to time a patient survives when the tumour is found to be so advanced that only a piece is removed for section, and no treatment of any kind is given; sometimes even secondary deposits disappear. There is some evidence that cases which are histologically borderline between ganglioneuroma and neuroblastoma become converted to the former with high doses of *folic acid* with subsequent betterment of the prognosis. With *operation*, followed by *X-ray therapy*, about 25 per cent. of the patients recover, and if they are free from recurrence by the end of one year, it is almost certain that they are cured permanently (Gross).

Those occurring in adults (rare in children):

Phæochromocytoma is a soft brownish benign tumour, usually less than 5 cm. in diameter, composed of large differentiated sympathetic ganglion cells, and a few fibres enclosed in a delicate capsule. It owes its name to the presence of chromaffin granules. In about 15 per cent. of cases the tumour is bilateral. This tumour, occurs in both sexes, usually during early adult life or middle age. It produces, either intermittently or continuously, an excess of adrenaline, and especially of noradrenaline: the ratio of the latter to the former often being as high as 20:1 causing *hypertension* which is either *paroxysmal* or *persistent*. The latter predominates statistically and probably indicates a late stage of the disease. Consequently all patients under sixty years of age who suffer from sustained arterial hypertension deserve routine tests to confirm or exclude a phæochromocytoma. While not more than 0·5 per cent. of cases of hypertension are caused by a phæochromocytoma, at the Mayo Clinic, where routine diagnostic procedures are undertaken to confirm or exclude the presence of this tumour in all cases of hypertension, the percentage has been stated to be nearly 3 per cent. Untreated, it progresses to a fatal termination.

Clinical Features.—The most common symptoms, in order of frequency, are: headache (55 per cent.), palpitation, vomiting, sweating, dyspnœa,

William Pepper (Junior), 1874–1947. Dean, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, U.S.A.
Sir Robert Hutchison, 1871–1960. Physician, The London Hospital.
James Ewing, 1866–1943. Professor of Pathology, Cornell University, New York, U.S.A.
Robert Edward Gross, Contemporary. Ladd Professor of Children's Surgery, Harvard University, Boston, Massachusetts, U.S.A.
The Mayo Clinic, Rochester, Minnesota, U.S.A. was founded in 1889 by William Worrall Mayo (1819–1911) and his two sons William James Mayo (1861–1939) and Charles Horace Mayo (1865–1939).

weakness, pallor—i.e. the symptoms of adrenaline overdosage. A typical attack is associated with hypertension. When the blood pressure is persistently high the patients are liable to all the complications of hypertension and may present with them. Ten per cent. of the patients have diabetes mellitus.

Diagnostic Tests.—(i) *Vanillyl mandelic acid (VMA)* estimations carried out on three consecutive 24-hour urine collections in a hypertensive phase. Normal excretion is less than 7 mg/24 h, and one is alerted if it is 8 mg/24 h or over. Modern methods of thin layer chromatography have improved this screening method. Another method is the 24-hour metanephrine excretion.

(ii) *Radiology.*—by the non-invasive CT scan, or by the invasive use of angiography which shows a vascular ' blush ' at the site of the tumour. It also reveals the vascular state and size of the kidneys. If a ' blush ' is not seen and one kidney is diseased, this is the likely explanation of the hypertension.

Operation.—Twenty to 40 mg (for an adult) of Rogitine[1] (1 to 2 tablets) should be given after meals t.d.s., while the patient is being prepared for operation. The hazardous phases in the operation are: during the induction of anæsthesia, positioning of the patient on the operating table, when the tumour is manipulated, and immediately after removal of the tumour. Five mg of Rogitine are given intravenously, prior to commencing the anæsthetic. With an intravenous drip infusion of dextrose-saline running, the operation is commenced. If pre-operative investigations have not revealed the side on which the tumour is situated, the procedure detailed on p. 656 is carried out. As some 10 per cent. of phæochromocytomas are ectopic there is something to be said for exploration via the anterior route (p. 656). The blood-pressure rises sharply when the affected adrenal gland is handled, and to counteract this rise another 5 mg of Rogitine is given into the tube of the intravenous drip. Should it be necessary, yet another dose is given. As soon as the adrenal gland has been extirpated, a considerable fall in the blood pressure is to be expected, and is combated by giving an injection of noradrenaline (p. 657), which, during the first week following the operation, should always be at hand for immediate intravenous injection in case of need. With these precautions, the earlier operative mortality has been lowered. If symptoms persist after unilateral adrenalectomy, a tumour in the contralateral gland is highly probable. In this instance the second tumour, which is usually well-defined, must be dissected from the healthy portion of the gland. Some consider this to be the better technique for all cases, particularly because these tumours are almost always benign and the operation is curative.

The excised specimen, when fixed in bichromate solution, stains brown.

Hyperplasia of the adrenal medulla, although often more in evidence on one side than the other, is usually bilateral. Paroxysmal hypertension, clinically identical with that produced by a phæochromocytoma, is present. Unilateral adrenalectomy brings about amelioration, but for a cure of the condition, the remaining adrenal should be removed, after an interval.

[1] Rogitine (Ciba) (phentolamine B.P.), a substance that suppresses temporarily the pressor activity of the secretion of chromaffin tissue.

CHAPTER 37

THE BREAST

Comparative and Surgical Anatomy.—Mammals are distinguished and so-called because they are provided with mammary glands. The cow, sheep, goat, mare and the elephant have an udder surmounted by teats, while other animals are furnished with breasts, the number of pairs of which vary with the species, and is related to the average number of offspring in each litter. Thus the sow has six to nine pairs, rodents six or seven pairs, while, like man, the anthropoid apes, the lioness, the sea-cow and, as John Hunter first noted, the whale, have but a single pair.

In anatomical works the protuberant part of the human breast is generally described as overlying the second to the sixth ribs, and extending from the lateral border of the sternum to the anterior axillary line. Actually a thin layer of mammary tissue extends considerably farther on all sides, viz: to the clavicle above, to the seventh or eighth ribs below, to the mid-line medially, and to the edge of the latissimus dorsi posteriorly. This fact is of importance to a surgeon when he seeks to remove the whole breast. The full extent of the breast is apparent in cases of milk engorgement.

The axillary tail of the breast is of considerable surgical importance. In some normal cases it is palpable, and in a few it can be seen in the pre-menstrual phase and during lactation. A well-developed axillary tail is sometimes mistaken for a mass of enlarged lymph nodes or a lipoma.

The lobule is the basic structural unit of the mammary gland. In the human breast the number and size of the lobules vary exceedingly: they are largest and most numerous during early womanhood. From ten to more than a hundred lobules empty by means of ductules into a lactiferous duct, of which there are from fifteen to twenty. Each lactiferous duct is lined by a spiral arrangement of contractile myo-epithelial cells and is provided with a terminal ampulla—a little reservoir for milk or abnormal discharges.

The ligaments of Cooper are hollow conical projections of fibrous tissue filled with breast tissue, the apices of the cones being attached firmly to the superficial fascia and thereby to the skin overlying the breast. These ligaments account for the dimpling of the skin overlying a scirrhous carcinoma, or other lesions of the breast accompanied by fibrosis.

The Areola.—The subcutaneous tissue contains involuntary muscle arranged in concentric rings as well as radially. The areolar epithelium contains numerous glands of three kinds—sweat glands, sebaceous glands, and accessory mammary glands. The sebaceous glands (known as the glands of Montgomery) enlarge strikingly during pregnancy and serve to lubricate the nipple during lactation. The accessory mammary glands are minute, inconstant, and possess ducts that open on the areola.

The Nipple is covered by a thick and rather crinkled skin. Near its apex, and very difficult to see because of the cutaneous corrugations, lie the orifices of the lactiferous ducts. The nipple and the areola of a nullipara are pink; with succeeding pregnancies they become pigmented by deposits of melanin. The nipple contains smooth muscle fibres arranged concentrically and longitudinally; thus it is an erectile structure and, for the convenience of the infant in arms, points forwards and *outwards*.

Lymphatics.—The lymphatic vessels of the breast drain into (1) nodes that lie between the greater and lesser pectoral muscles; (2) the thoracic chain of lymph nodes along the lateral thoracic artery; (3) the subscapular chain that extends from the lateral thoracic wall to the axillary vein, and thence along the axillary vein, most of the lymph nodes being on the caudal aspect of that vessel; (4) the central group, which is the largest, lying at the apex of the axilla. The highest of these lymph nodes is sheltered beneath the clavicle; (5) The internal mammary group which lie along the internal mammary vessels.

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London.
Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London.
William Fetherston-Haugh Montgomery, 1797–1859. Obstetrician, Dublin.

It is highly important to know that: (*a*) there is free communication between the subclavicular and the supraclavicular lymph nodes; (*b*) that the postero-cervical chain of lymph nodes is linked with those of the axilla, and that (*c*) the lymph nodes along the internal mammary artery are involved in about half the cases in which the axillary lymph nodes are implicated (Handley).

Investigation of the Breast.—Although an accurate history and clinical examination are still the most important methods of detecting breast disease there are a number of investigations which can assist in the diagnosis:

Mammography.—Soft tissue X-rays are taken by placing the breast in direct contact with fine-grained film and exposing it to low voltage high amperage X-rays for a prolonged time. The standard views are lateral and supero-inferior. Mammography is a relatively accurate method of demonstrating the soft tissue architecture of the breast, but it should always be combined with clinical examination.

Galactography.—Soft tissue X-rays combined with the injection of radio-opaque material into a major lacteal duct may be used to demonstrate a duct tumour.

Thermography.—A technique by which the heat emission from the surface of the breast in the form of infra-red radiation can be recorded. The information is usually displayed on a photographic plate or a cathode tube comparable to a television screen. The heat intensity varies with the vascularity and metabolism of the tissues and is increased in inflammatory conditions and some cancers. Unfortunately the technique is as yet relatively inaccurate and has little clinical value.

THE NIPPLE

Absence of the nipple is rare, and usually associated with amazia (p. 663).

Supernumerary nipples occasionally occur along a line extending from the anterior fold of the axilla to the fold of the groin. This constitutes the milk line of lower mammals.

Retraction of the nipple is of two important varieties: (*a*) that occurring at puberty (remote), and (*b*) that occurring during womanhood (recent).

(*a*) **Retraction occurring at puberty** is due in most instances to a developmental abnormality; the nipple, for some unknown reason, does not develop *pari passu* with the breast. It is possible that in a few instances the retraction is an aftermath of mastitis of infancy. In about one-quarter of the cases the condition is bilateral.

Non-protuberance of the nipple hinders an infant suckling at the breast. Clinical experience shows that a breast with long-standing retraction of its nipple is prone to local excoriation by the retained sebaceous gland secretion and, especially during lactation, to infection and abscess formation.

Treatment.—During and soon after puberty: if the patient draws out the nipple between finger and thumb daily for about three weeks the condition is usually remedied.

(*b*) **Recent Retraction.**—The importance of long-standing retraction of the nipple is dwarfed by the ominous diagnostic significance of recent retraction, which is a frequent accompaniment of scirrhous carcinoma (p. 677). Therefore the all-important question to put to the patient is: ' How long has this nipple been retracted ? '

Cracked Nipple.—Want of care in the preparation for lactation and neglect of the hygiene of the nipple during lactation are the chief causes of this not uncommon condition. Its main importance lies in the fact that the crack may be a forerunner of acute infective mastitis.

Prophylaxis.—During the last two months of pregnancy the nipples and

Richard Sampson Handley, Contemporary. Consulting Surgeon, Middlesex Hospital, London.

their areolæ should be washed, dried, and anointed with a little lanoline each day. The same routine may be continued, with advantage, after suckling.[1]

Treatment.—At the first sign of soreness the nipple is rested for twenty-four to forty-eight hours and the breast emptied with a breast-pump, as necessary. The nipple is washed and a mild antiseptic cream applied. When the soreness ceases, the baby is put to the breast for one minute at first and normal feeding is gradually resumed.

FIG. 690.—Papilloma of the nipple.
(*R. R. Deshmukh, M.S., Dhantoli, Nagpur, India.*)

Papilloma of the nipple (fig. 690) presents the features of a cutaneous papilloma. Sometimes it grows to the size of a cherry, but the pedicle is always narrow. The treatment is excision together with a tiny disc of the skin from which it grows.

Retention Cyst of a Gland of Montgomery.—These glands, situated in the areola, secrete sebum and, as a result of an orifice of one of the glands becoming blocked, a sebaceous cyst forms.

Chancre of the Nipple. — The majority of chancres of the nipple occur by infection from a syphilitic buccal mucous patch in the mouth of a member of the opposite sex. Although wet-nurses are now seldom, if ever, employed in this country, when this form of infant feeding was used the nipple was sometimes infected from the mouth of a syphilitic baby. The mother of such an infant is immune to re-infection from her own child.

Eczema of the nipple is a rare condition and is bilateral, and presents features common to eczema elsewhere.

Paget's disease of the nipple (p. 678) must be distinguished from the foregoing.

ABNORMAL DISCHARGES FROM THE NIPPLE

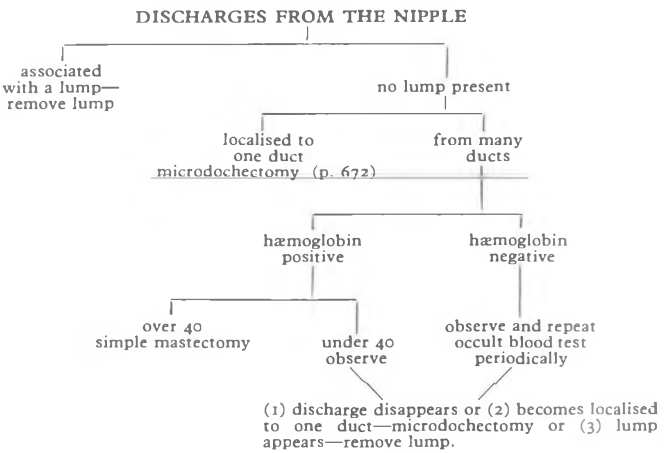
The discharge occurs from one (rarely more) of the lactiferous ducts. The management depends on the presence of a lump, or of occult blood, or whether it is localised to one duct. Mammography is usually unhelpful except to identify a grossly distended duct or a circumscribed tumour.

A clear serous discharge is associated usually with a retention cyst, consequent upon fibroadenosis, but may signify an underlying carcinoma.

A blood-stained discharge is pathognomonic of duct papilloma or duct carcinoma. The former condition is more common than the latter.

A black or green discharge may be due to altered blood from the foregoing, but is much more frequently an accompaniment of ductal changes and the secondary retention cysts of fibroadenosis. In cases of extensive

[1] In some maternity hospitals the nipple is cleansed before and after suckling by wiping it with a sponge removed from 70 per cent. alcohol solution kept in a jar labelled with the patient's name.


DISCHARGES FROM THE NIPPLE
associated with a lump—remove lump
no lump present
localised to one duct microdochectomy (p. 672)
from many ducts
hæmoglobin positive
hæmoglobin negative
over 40 simple mastectomy
under 40 observe
observe and repeat occult blood test periodically
(1) discharge disappears or (2) becomes localised to one duct—microdochectomy or (3) lump appears—remove lump.

Scheme for management of discharges from the nipple.
(*After Sir Hedley Atkins, Past P.R.C.S., London.*)

dirty green discharge, when the breast is removed it is usually found to be riddled with cysts containing the same material.

Common causes of discharge are *Duct Papilloma* (p. 672), *Mammillary Fistula* (p. 667), and *Duct Ectasia.*

Duct Ectasia.[1]—A primary dilatation of the major ducts of the breasts which is commonly associated with discharge from the nipple in middle aged women. The ducts become distended with amorphous debris and crystalline material probably lipid in nature and the discharge is of similar composition. The initial presentation is of a worm-like swelling extending radially from the nipple. As the condition progresses there is an associated periductal inflammation with lymphocytes and plasma cells at which stage there may be local pain and tenderness and ultimately a hard mass with local skin tethering and nipple retraction (Plasma cell mastitis). The condition is benign but may be mistaken for either cancer or an abscess (Haagensen, Hadfield). Provided a tumour can be excluded the treatment should be conservative but symptomatic relief can be gained by local removal of the duct and affected segment of the breast.

THE BREAST

CONGENITAL ABNORMALITIES

Amazia.—Congenital absence of the breast may occur on one (fig. 691) or both sides. It is sometimes associated with an absence of the sternal portion of the pectoralis major. Amazia, which is rare, is more common in males.

Polymazia.—Accessory breasts have been recorded in the axilla, groin, buttock, and thigh, the most frequent site being the axilla. They have been known to function during lactation (fig. 692).

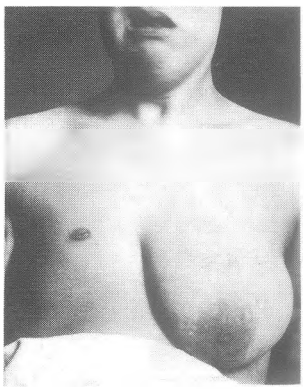


FIG. 691.—Congenital absence of the right breast.

[1] John Birkett, 1815–1904, surgeon to Guy's Hospital gave an early account of this curious condition in 1850. He was a founder member of the Pathological Society of London.

Cushman D. Haagensen, Contemporary. Surgeon, Columbia University College of Physicians and Surgeons, New York, U.S.A.
Geoffrey John Hadfield, Contemporary. Surgeon, Stoke Mandeville Hospital, Aylesbury, England

DIFFUSE HYPERTROPHY

Diffuse hypertrophy of the breasts occurs sporadically in otherwise healthy girls at puberty and, much less often, during the first pregnancy. The breasts attain enormous dimensions (fig. 693), and may reach below the knees when the patient is sitting. This tremendous overgrowth of the mammary glands is due, apparently, to their

FIG. 692.—Functioning axillary breast. *(M. Kumar, F.R.C.S., Calicut, S. India.)*

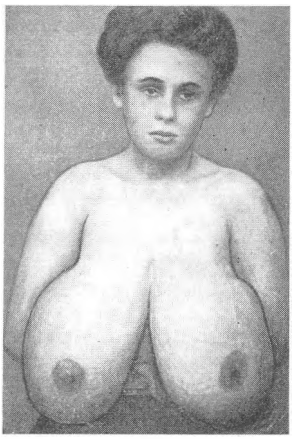


FIG. 693. —Diffuse hypertrophy (virginal).

extreme sensitivity to œstrogenic hormone. Sometimes the hypertrophy is unilateral.[1] Unilateral hypertrophy may be easily mistaken for a breast which is pushed forward by a large lipoma or other tumour of the retromammary area. If diffuse hypertrophy causes real distress, a plastic procedure in the form of a reduction mammoplasty is indicated.

INJURIES OF THE BREAST

Injuries of the breast were rare until the introduction of the diagonal motor car seat belt but are comparatively unimportant.

Hæmatoma, particularly a resolving hæmatoma, gives rise to a lump which, in the absence of overlying bruising, is difficult, if not impossible, to diagnose correctly until an exploratory incision has been made.

Traumatic fat necrosis may be acute or chronic, and usually occurs in stout, middle-aged women. Following a blow, or even indirect violence (e.g. contraction of the pectoralis major), a lump, often painless, appears. In the absence of a definite lead, the swelling, which is often attached to the skin, is usually diagnosed as a carcinoma, though mammography will provide the diagnosis. A definite history of injury should bring the condition to the clinician's mind. On incising the lump a chalky white area of necrotic fat is found resembling necrosis seen in cases of subsiding acute pancreatitis.

ACUTE AND SUBACUTE INFLAMMATIONS OF THE BREAST

Mastitis of infants is at least as common in the male as in the female. On the third or fourth day of life, if a breast of an infant is pressed lightly, a drop of colourless fluid can be expressed; a few days later there is often a slight milky secretion, which finally disappears during the third week. This is popularly known as ' witch's milk '. The explanation of this phenomenon

[1] In tropical countries this must be distinguished from filarial elephantiasis of the breast.

is that the hormone which stimulates the mother's breast reacts also upon the mammary tissue of the fœtus. Thus it is essentially physiological.

Mastitis of puberty is encountered rather frequently, usually in males. The patient, aged about fourteen, complains of pain and swelling in the breast. In 80 per cent. the condition is unilateral but the opposite breast may be affected later. The breast is enlarged, tender and slightly indurated. Suppuration never occurs. The tenderness subsides in fourteen days or so, but induration often persists for several weeks. In some instances enlarged tender breasts may persist in males for a prolonged period even up to years. In such circumstances it may be justifiable to recommend local mastectomy.

Mastitis of mumps is usually unilateral, and more common in females.

Mastitis from local irritation, which is commonly subacute, may be produced from a too tight elastic brassière. The condition is by no means rare in men (p. 686).

Mastitis from milk engorgement is liable to occur about weaning time ; and sometimes in the early days of lactation when one of the lactiferous ducts becomes blocked with epithelial debris. In the latter instance a sector only of the breast becomes indurated and tender.

Bacterial mastitis, which is by far the most common variety of mastitis, nearly always commences acutely. Although often referred to as mastitis of lactation, it is incorrect to assume that acute mastitis in women is necessarily lactational. Of a hundred consecutive cases of breast abscess, thirty-two occurred in women who were not lactating (De Jode); probably some were due to infection of a hæmatoma. In almost every case the infecting organism is a staphylococcus. In cases where the infection is acquired in hospital no less than 90 per cent of the infecting staphylococci are insensitive to penicillin.

Ætiology.—In maternity wards, staphylococcal infections of the breast sometimes assume almost epidemic proportions, due, no doubt, to a staphylococcal carrier among the nursing staff. Usually the intermediary is the infant; after the second day of life 50 per cent. of infants harbour staphylococci in the nasopharynx.

' Cleansing the baby's mouth ' with a swab is also an ætiological factor. The delicate buccal mucosa is excoriated by the process; it becomes infected, and organisms in the infant's saliva are inoculated on to the mother's nipple.

There seems little doubt that in the great majority of cases the precursor of intramammary mastitis is failure of secretion to escape because one (rarely more) of the lactiferous ducts becomes blocked with epithelial debris—a hypothesis that is strenghtened by the fact that, whether they are lactating or not, intramammary mastitis and abscess of the breast are relatively frequent in women with a retracted nipple. While stasis in some part of the lactiferous tree is a major factor in the production of this condition, undoubtedly the older hypothesis—ascending infection from a sore or an infected cracked nipple—must not be spurned. Once within the ampulla of the duct, staphylococci cause clotting of milk. Within the clot organisms multiply rapidly.

Clinical Features.—The affected breast, or more usually mainly one part

Louis Rene Julien De Jode, Contemporary. Surgeon, Whipps Cross Hospital, Leytonstone, London.

of it, presents the classical signs of acute inflammation, and what is aptly called ' the cellulitic stage ' of a breast abscess has been reached.

Treatment during the Cellulitic Stage.—The patient must be confined to bed. A broad spectrum antibiotic such as ampicillin 500 mg 6-hourly for seven days should be started immediately. Support to the breast and local heat will help to relieve the pain, and permit examination of the inflamed breast daily, which is essential.

To wean or not to wean is a question of great importance. Except when the patient has been suckling the child for over nine months, undoubtedly it is better not to wean. Feeding is continued from the uninfected breast, and the infected breast is emptied after each feed by manual expression, carried out by the patient herself; this is more effective than a breast pump, which does not always clear obstructed ducts. The milk expressed from the inflamed breast is boiled for five minutes before being given to the child in a feeding bottle. In patients with bilateral acute mastitis the baby can be fed entirely on the milk obtained by expression and boiled; this is not detrimental to the baby.

In cases where the child has already been breast-fed too long, or the mother finds manual expression too painful, weaning is advisable. Stilbœstrol 10 mg three times a day or as a single dose, is given after the breasts have been emptied and this usually inhibits lactation completely.

Formation of an 'Antibioma'.—It is absolutely essential that an antibiotic should *not* be given if pus is already present. In such circumstances, if an antibiotic is given the pus in the abscess frequently becomes sterile and a large brawny œdematous swelling remains in the breast and takes many weeks to resolve. Sometimes there is excessive fibrosis and this, with the absence of tenderness, has caused the diagnosis to be revised in favour of a carcinoma. It is better to explore the mass with a wide-bore aspirating needle than to cause an ' antibioma ' with its attendant pain, chronicity, and ill health. Most ' antibiomas ' are due to late, inadequate, and ineffective antibiotics.

Indications for Operation.—The breast should be incised when, after emptying, an area of tense induration is felt and/or when œdema of the overlying skin is found. In contrast to the majority of localised infections, fluctuation is a late sign and incision must not be delayed until it appears. Usually the area of induration is sector-shaped, and in early cases about one-quarter of the breast is involved (fig. 694); in many later cases the area is more extensive (fig. 695).

Drainage of an Intramammary Abscess.—The usual incision is sited in a radial direction over the affected segment. One which follows the cutaneo-areolar margin has a better cosmetic value and does permit access to the affected area. The best cosmetic result, however, follows the most adequate drainage rather than a particular incision. The incision passes through the skin and the superficial fascia. A long hæmostat is then inserted into the abscess cavity. Every part of the abscess is palpated against the point of the hæmostat, and its jaws are opened. All loculi that can be felt are entered. Finally, the hæmostat having been withdrawn, a finger is introduced and any remaining septa are disrupted. Unless the abscess cavity is situated at the very highest sector of the breast a counter-incision should be made at the most dependent part of the breast and a drainage tube inserted. In this, almost

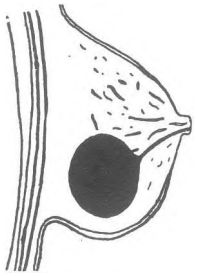


FIG. 694.—Intramammary breast abscess.

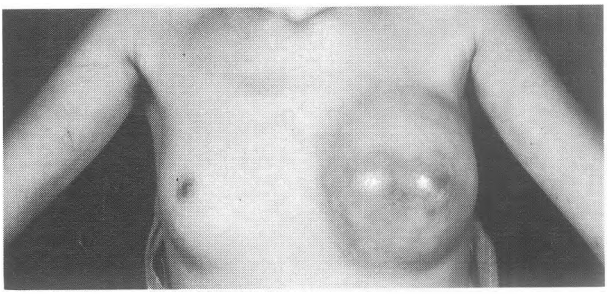


FIG. 695.—Intramammary breast abscess. The abscess should always be drained before it becomes subcutaneous. (*T. A. Bouchier-Hayes, F.R.C.S.I., Dublin.*)

more than any part in the body, *dependent* drainage is essential. Dressings are carried out aseptically on the second day, when the tube is removed, and subsequently every other day. Antibiotic therapy is unnecessary with adequate drainage but if started should be continued for the full course.

Subareolar mastitis is not a true mastitis but results from an infected (sebaceous) gland of Montgomery, or from a furuncle on or near the areola. The inflammation develops insidiously, usually without constitutional symptoms. When the patient presents early, there is often an area of induration no larger than a pea. No matter how small, if a lump can be felt, pus is present (fig. 696), and the abscess should be drained without delay. Spontaneous rupture, if allowed to occur, does not cure the condition; it merely results in recrudescence or chronicity.

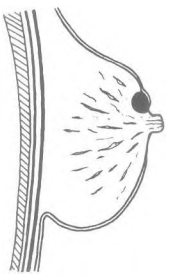


FIG. 696.—Subareolar abscess.

Chronic intramammary abscess which follows inadequate drainage or injudicious antibiotic treatment is often a very difficult condition to diagnose: when encapsulated within a thick wall of fibrous tissue, the condition cannot be distinguished from carcinoma without the histological evidence from a biopsy.

FIG. 697. — Milk-fistula originating in a chronic subareolar abscess.

Chronic Subareolar Abscess (leading to a *milk-fistula* (syn. *Mammillary fistula*)).—A recurrent subacute or a chronic abscess may occur apart from lactation in women of the child-bearing age. The condition is a frequent complication of long-standing retraction of the nipple the infection being restricted to a single obstructed duct system. The abscess ruptures and subsides, only to repeat the cycle over and over again at intervals of a few months when it forms a chronic milk-

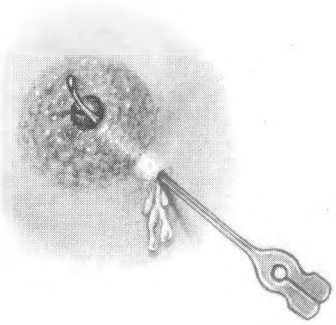


FIG. 698.—Slitting up a chronic subareolar abscess. In this case the patient has a retracted nipple of long standing. (*After H. T. Caswell and A. W. E. Burnett.*)

fistula (fig. 697). A non-infective inflammation such as duct ectasia (p. 663) may also result in fistula formation.

Treatment.—Antibiotic therapy followed by incision and drainage is useless. The fistula must be treated in the same way as a fistula-in-ano, i.e. the track is laid open and saucerised (fig. 698).

Tuberculosis of the breast, which is comparatively rare among Western races but more common in Asia and some other parts of the world, is, as a rule, associated with active pulmonary tuberculosis, or tuberculous cervical adenitis.[1]

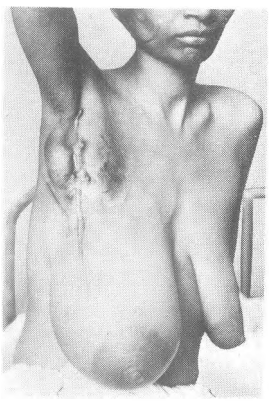


FIG. 699.—Tuberculosis of the breast with secondary suppurating axillary lymph nodes. *(Professor A. K. Toufeeq, Lahore, Pakistan.)*

Tuberculosis of the breast (fig. 699) occurs more often in women who have borne one or more children and usually takes the form of multiple chronic abscesses with multiple sinuses and a typical bluish attenuated appearance to the surrounding skin. The diagnosis rests on bacteriological and histological examination. Treatment with antituberculous chemotherapy should be given. Healing is usual though often delayed, and mastectomy should be restricted to patients with persistent residual infection.

Actinomycosis of the breast is rarer still, except among peasant women who work in the fields of Europe. The lesions present the essential characteristics of faciocervical actinomycosis (p. 39). If healing does not occur as a result of the systemic treatment of actinomycosis recommended on p. 39, the affected breast should be amputated.

Syphilis of the Breast.—A primary chancre of the nipple has been referred to on p. 662. Secondary lesions of syphilis in the form of mucous patches are sometimes found in the submammary fold. In the second stage also both breasts may become swollen and painful, the condition being known as diffuse syphilitic mastitis. Gumma of the breast is very rare, and almost impossible to diagnose unless there is other evidence of syphilis.

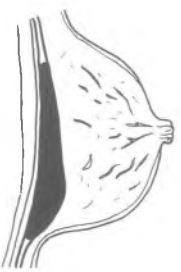


FIG. 700.—Retromammary abscess.

Retromammary Abscess (fig. 700).—Here the pus is situated in the cellular tissues behind the breast, and in the great majority of cases the abscess has no connection with the breast proper. Usually a retromammary abscess originates from a tuberculous rib, infected hæmatoma, or possibly from a chronic empyema necessitatis (p. 719), and treatment must be directed to the relief of these conditions. A submammary incision allows the breast to be retracted as necessary from the field of operation.

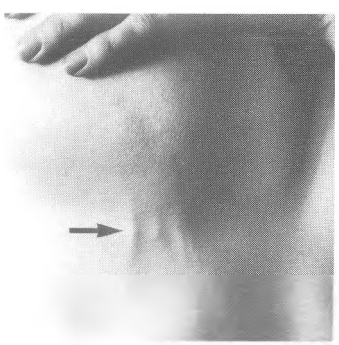


FIG. 701.—Mondor's disease, under the right breast.

MONDOR'S DISEASE

Mondor's disease is thrombophlebitis of the superficial veins of the breast and anterior chest wall (fig. 701) though it has also been encountered in the arm.

[1] In 1829 Sir Astley Cooper, Surgeon to Guy's Hospital, London, described scrofulous swellings in the bosoms of young women, most of whom suffered from tuberculous cervical adenitis.

Henri Mondor, 1885–1962. Surgeon, des Hôpitaux, Paris.

In the absence of injury or infection, the cause of the thrombophlebitis—like that of spontaneous thrombophlebitis in other sites—is obscure. The essential characteristic physical sign is an indurated subcutaneous thrombophlebitic cord about 3 mm. in diameter, of varying length, situated in the subcutis of the breast. Usually it is attached to the skin, and of a consistency that has been likened to that of the vas deferens. When the skin over the breast is stretched by raising the arm, a narrow, shallow subcutaneous groove alongside the cord becomes apparent. The great importance of the condition is that those unfamiliar with Mondor's disease are likely to diagnose the condition as one of lymphatic permeation from an occult carcinoma of the breast. The only treatment required is restricted arm movements, and in any case the condition subsides within a few months without recurrence, complications, or deformity.

FIBROADENOSIS[1]

Ætiology.—Fibroadenosis is an aberration of the physiological changes that occur in mammary tissue at puberty (evolution) and at the menopause (involution). Although it is called an ' aberration ' it may be nothing more than a variation on the normal theme of cyclic proliferation and regression. Some women, however, worry about the lumps or the pain which it sometimes causes and thus it has become a disease ' entity '. Many women are affected without being aware of it.

Pathology.—When sectioned with a knife the affected areas in the breast are white or yellow and of india-rubber consistency, but they never present the grey tones and hard gritty texture of carcinoma. Microscopically the disease consists essentially of five features which vary in extent and degree in any one breast—these are:

1. *Cyst Formation.*—Cysts are almost inevitable and vary much in size. They contain dark mucoid material.

2. *Adenosis.*—There is a budding and multiplication of acini and an overall increase in glandular tissue.

3. *Fibrosis.*—Fat and elastic tissue disappears and is replaced by dense white fibrous trabeculæ. This fibrous tissue compresses the ducts and leads to cyst formation. The interstitial tissue is infiltrated with small round cells and large pink cells.

4. *Epitheliosis.*—Hyperplasia of epithelium in the lining of the ducts and acini may occur.

5. *Papillomatosis.*—The epithelial hyperplasia may be so extensive that it results in papillomatous overgrowth within the ducts.

After decades of discussion there is still no agreement between surgeons and pathologists as to whether fibroadenosis is a premalignant condition. The majority are of the opinion that it is not, while others are equally emphatic that the epithelial hyperplasia when florid is undoubtedly premalignant. Both fibroadenosis and carcinoma of the breast are common conditions; by coincidence, they can coexist, which in point of fact occurs in 3 per cent. of cases of fibroadenosis.

Clinical Features.—Premenstrual pain and tenderness, usually bilateral

[1] Many alternative terms have been applied to this condition. Fibrocystic Disease, Mammary Dysplasia and Chronic Mastitis are in common use, the choice depending on the opinion of the individual surgeon as to the nature of the disease.

and often associated with fine nodularity of the breast, occurs frequently. In some young women the discomfort may be disabling in its severity and may be so prolonged as to lose its cyclic pattern. There are minimal abnormal changes in the breast tissue of these patients and the symptoms are self-limiting requiring no active treatment. Pregnancy and lactation nearly always produce permanent relief. The contraceptive pill will also produce a reduction in symptoms in most women although in others, previously asymptomatic, the pill may stimulate discomfort. True fibroadenosis, though it may occur at any age after puberty, is most prevalent between the ages of forty and fifty-five. Spinsters, childless married women, and multiparous women who have not suckled their children are the usual sufferers. This suggests that the condition is prone to appear in breasts that have been denied their intended function. The patient usually complains of pain in *one* breast, worse before menstruation, or after using the arm. On examination both breasts are inclined to be nodular—the nodules being about the size of rice grains. As a rule it is possible to define the saucer-like edge of the periphery of the breast. When the breast complained of is examined between finger and thumb its texture is unusually firm, more often than not an indefinite lump can be made out, but with the flat of the hand this can be felt only vaguely. The lump is neither adherent to the pectoral fascia nor to the skin. There is no recent retraction of the nipple, but occasionally there is a serous or dark-green discharge therefrom. Fibroadenosis is sometimes more in evidence in one quadrant of the breast than in the remainder. The presence of a palpable cyst or cysts is complementary to the more general clinical findings of fibroadenosis *per se*. Frequently the axillary lymph nodes are slightly enlarged; they are not hard but are often tender.

Treatment.—Reassurance is probably the most important part of treatment. It must be explained that these changes occur at this stage of life, that they are not precancerous and a certain amount of pain is to be expected.

Support for the breast by a firm brassière worn day and night is essential. Diuretics administered during the time of greatest discomfort may reduce local tension pain but simple analgesia is usually the most effective management. When the menopause is complete the pain will cease and the lumps disappear. From the surgeon's point of view it is important to confirm that the palpable lump is not a carcinoma. Mammography (p. 661) may be helpful but an excision biopsy of the local lesion is necessary in doubtful cases.

Indications for Surgery in Fibroadenosis.—Apart from excision biopsy, surgery is only indicated when the patient is persistently disabled by intolerable pain, when there is pathological anxiety or when the degree of epitheliosis is such that there is a risk of early malignancy. The advent of synthetic breast prosthetic implants has meant that a subcutaneous mastectomy is no longer associated with the physical and emotional trauma of the standard mastectomy.

CLASSIFICATION OF CYSTIC SWELLINGS

Cysts of the breast are related to either the ducts or the stroma.

Ducts —Fibroadenosis < Solitary. / Multiple.

Intracystic papilliferous carcinoma.
Galactocele.
Papillary Cystadenoma (p. 673).
Serocystic disease of Brodie (p. 673).
Stroma —Colloid degeneration of carcinoma (p. 676).
Lymphatic cyst.
Hydatid.

(*a*) *Simple Solitary Mammary Cyst.*—The size of the cyst varies widely, the seat of election being the upper and outer quadrant. Macroscopically the solitary variety (fig. 702) presents as a blue-domed fluctuant mass (Bloodgood), the colour being due to its fluid content. Although, clinically, the condition is a solitary cyst, as a rule the breast parenchyma contains a number of tiny satellite cysts. That the swelling is a cyst may sometimes be confirmed by transillumination; aspiration is required to establish the diagnosis.

(*b*) *Multiple Cysts.*—This is the commonest type; the cysts are usually several in number. Sometimes one sector of the breast is affected alone. If a main duct near the nipple is obstructed, a duct papilloma should be suspected.

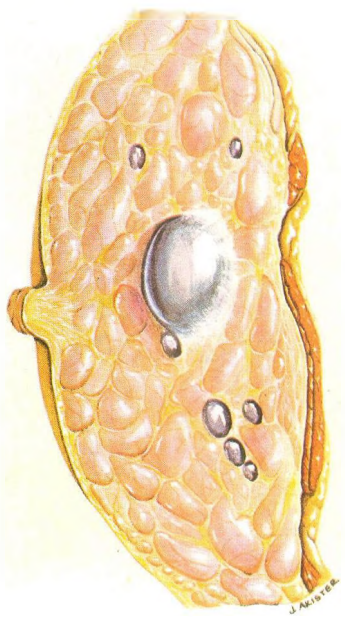


FIG. 702.—The blue-domed cyst of Bloodgood. (*After C. F. Geschickter.*)

Treatment.—*To aspirate or not to aspirate? That is the question!*

Aspiration has its dangers as an intracystic neoplasm may be missed. It is only safe if (1) the cyst does not refill, (2) the fluid withdrawn is not blood stained, (3) there is no residual lump after aspiration (Patey), (4) the cytological examination of the aspirated fluid shows no evidence of malignant cells. If any of these criteria is not fulfilled, biopsy excision must be done, but a preliminary mammogram may be helpful to confirm the diagnosis of cancer or occasionally to support the diagnosis of fibroadenosis. A solitary cyst can be excised through a circumferentially orientated incision over the swelling.

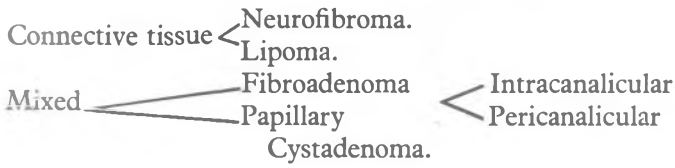
Galactocele, which is extremely rare, usually presents as a solitary, subareolar cyst, and always dates from lactation. It contains milk, liquid or inspissated, and in long-standing cases its walls tend to calcify. It can become enormous (fig. 703).

Lymphatic cyst is a curiosity, and its pathology is similar to the more common lymphatic cyst of the neck (p. 599).

BENIGN NEOPLASMS OF THE BREAST

Epithelial —Duct papilloma.
—Pure adenoma (very rare).

Joseph Colt Bloodgood, 1867–1935. Surgeon, Johns Hopkins Hospital, Baltimore, Maryland, U.S.A.
David Howard Patey, 1899–1976. Surgeon, The Middlesex Hospital, London.



Duct Papilloma.—The majority of these tumours are single, but bilateral examples are not rare, and occasionally two or more ducts of the same breast are the seat of a papillary growth. The usual single papilloma often has a stalk, and is situated in one of the larger lactiferous ducts.

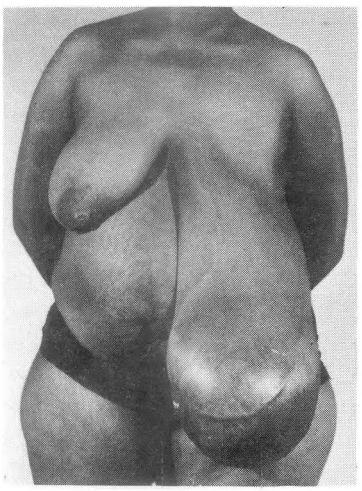


FIG. 703.—Galactocœle. A 27-year-old multipara (3) with progressive breast enlargement following confinement. *(O. O. Ajayi, F.R.C.S., and O. Adekunle, F.R.C.S., Ibadan, Nigeria.)*

Clinical Features.—The condition is rare before the age of twenty-five, and usually occurs in women between thirty-five and fifty. In the majority of cases bright red blood or, less often, a dark blood-stained discharge from the nipple is the only symptom. On examination, a cystic swelling can sometimes be felt beneath the areola; pressure upon it will cause a discharge from the mouth of the affected duct on the nipple. The majority of patients are found to have a solitary papilloma which is benign but identical symptoms are associated with multiple papillomatosis which is undoubtedly premalignant and after months or years will transform into a duct carcinoma.

Treatment.—Amputation of the breast is unnecessary, the removal of the papilloma and the involved duct providing a cure. It is important not to express the blood before operation as it then may be difficult to identify the duct in the theatre.

Microdochectomy (after Hedley Atkins and Brigitte Wolff).—A fine sewing needle, from which the point has been removed, is inserted into the duct from which the discharge is emerging. The skin of the nipple is then fixed to the eye of the needle by fine silk stitches. The skin of the incision is then infiltrated with adrenaline in saline (1 ml. of adrenaline $\frac{1}{1000}$ in 80 ml. of saline). Using a pair of fine pointed scissors, a triangular area is cut 1 mm away from the eye of the needle. Skin flaps are then reflected and the needle together with the duct is excised to produce a mass of breast tissue about 2·5 cm in diameter. The lesion is nearly always situated within 4–5 cm of the nipple orifice. The whole specimen, together with the needle and triangular area of skin, is removed intact. After strict hæmostasis, the wound is closed without drainage.

Nearly all excised specimens are reported upon by the pathologist as "Papilloma. No evidence of malignancy"—a major triumph resulting from a comparatively minor surgical procedure.

Fibroadenoma of the breast is divided into two varieties, the pericanalicular and intracanalicular. They are benign tumours with epithelial ele-

Sir Hedley John Barnard Atkins, Contemporary. Emeritus Professor of Surgery, Guy's Hospital, London.
Brigitte Wolff, Contemporary. Formerly Clinical Assistant, Department of Surgery, Guy's Hospital, London.

ments set in a fibrous tissue stroma. In the pericanalicular type the epithelial part appears as normal ductules within a background of dense fibrous tissue. In the intracanalicular type the connective tissue stroma is less dense and projects into the duct system whose epithelial cells are flattened and form a single covering layer. A well-developed capsule is always present in both types.

Pericanalicular Fibroadenoma.—As a rule the patient is between fourteen and thirty, the peak being about twenty-one years of age, who complains of a lump in the breast. Exceptionally, there is more than one neoplasm present. On examination the lump is smooth, hard, and so freely movable that it has been termed a ' breast mouse '.

Treatment is enucleation of the tumour through an incision which should be periareolar; this is accomplished easily, because this neoplasm is entirely encapsulated.

Intracanalicular fibroadenoma, like the foregoing, is a definitely localised tumour. Much rarer than the hard variety, they occur in women about the age of thirty-five or forty. They are occasionally bilateral and usually lie deeply in the breast. Cystic and sarcomatous changes sometimes supervene.

Treatment.—As an intracanalicular fibroadenoma usually lies at some depth from the surface, it is best removed through a submammary incision (fig. 704).

Papillary cystadenoma is a swelling composed of cysts in which papillomatosis is so extensive that the cysts are almost filled and feel solid. It may cause a bloodstained discharge from the nipple. The condition is entirely innocent and local excision is all that is necessary.

MASSIVE SWELLINGS OF THE BREAST

These are:

1. Diffuse hypertrophy, which is usually bilateral (p. 664).

2. *Giant fibroadenoma* of breast in women (about 35 to 40, but occasionally in teenagers) may cause unilateral hypertrophy. This is usually retromammary, difficult to distinguish from a large firm breast, and can be removed by a submammary incision.

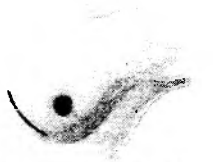


FIG. 704.—Incision for removing a soft fibroadenoma in the medial half of the breast.

3. *Serocystic disease of Brodie* (*syn.* Cystosarcoma Phylloides)[1] (fig. 705) may occur at any time from 40 years onwards. It is really a giant intracanalicular fibroadenoma which rapidly attains such a large size that the overlying skin necroses and the tumour fungates through. It is not malignant even though the term cystosarcoma implies this, but there is an undoubted association between this tumour and both sarcomatous degeneration and breast carcinoma. The surface of the tumour is unevenly bosselated with areas of softening and even fluctuation in the larger convexities. The overlying skin is thin and tense, and large veins can be seen coursing beneath the integument. As a rule the tumour is adherent neither to the skin nor to deeper structures. On occasions there is a serous discharge from the nipple. The axillary lymph nodes are not enlarged, except secondarily to infection.

[1] Phylloides—from the Greek *Φυλλωδηs* = leaf-like. There are branching projections of the tumour tissue into the cystic cavities of this neoplasm. It is not a sarcoma.

Sir Benjamin Collins Brodie, 1783–1862. Surgeon, St. George's Hospital, London. Described this disease in 1840.

Treatment. Local excision of the encapsulated tumour is all that is necessary but simple mastectomy is required for very large tumours.

When the Diagnosis of Carcinoma is in Doubt.—There will always be cases where the clinician cannot be sure whether a particular lump in the breast is a patch of fibroadenosis, a benign tumour, or an early carcinoma. In doubtful cases it is essential to advocate operation without delay. The first step must be to excise that part of the breast containing the lump. In many cases a naked eye inspection will indicate the nature of the mass (fig. 706) but where possible the diagnosis should be confirmed by immediate histological examination by frozen section techniques. In the absence of this facility it is wise for the surgeon to close the incision and await a formal histological report.

FIG. 705.—Cystosarcoma phylloides in a patient aged 26. It weighed 3 kg. (*Dr. Kulin Pandya, M.S., Godhra, India.*)

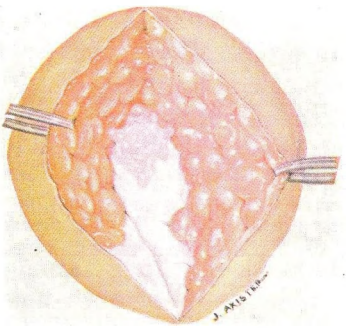


FIG. 706.—Macroscopic examination of a doubtful lump in the breast. In the lower part of the specimen the dead-white appearance of fibroadenosis is evident. In the upper quarter of this area there is a small carcinoma which cuts like an unripe pear, is greyish-white in colour and the surface of which is concave.

Needle Biopsy.—A restricted biopsy using a high-speed drill, an aspiration or a biopsy needle may give valuable information if positive but inaccuracies occur in the interpretation of the very small specimen. It is also possible for the biopsy needle to miss a small tumour deeply set within the breast.

CARCINOMA OF THE BREAST

Breast cancer is the commonest malignant disease of women in England and Wales. It causes 10,000 deaths annually. It is estimated that one in 17 of all female children born will develop the disease during their lifetime. In spite of an immense amount of investigation there is still no known cause and its natural history is obscure. Women between forty-five and fifty-five are its most frequent victims, but many factors are known to influence its frequency.

(*a*) *Racial.*—It occurs commonly in the Western World, England and Wales having the highest incidence. It is a rare tumour in the East.

(*b*) *Genetic.*—It occurs more commonly in women with a family history of breast cancer than in the general population.

(*c*) *Socio-economic.*—The incidence is higher in the lower socio-economic classes IV and V than in the higher I and II.

(*d*) *Dietary.*—In some countries it has been shown that the incidence is lower in areas of overall nutritional deficiencies.

(*e*) *Endocrine.*—It appears to be commoner in nulliparous women than in women who have borne many children and have breast fed. It is also less common in women who have their first child at an early age especially if associated with late menarche and early menopause.

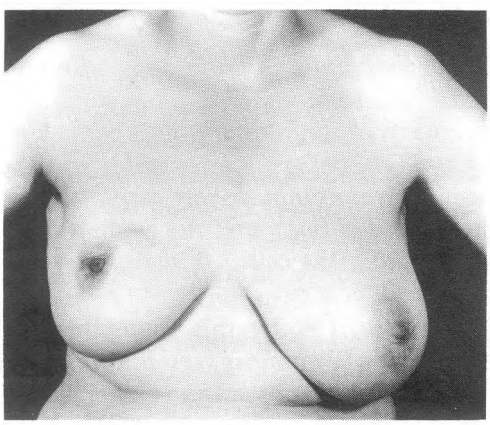


FIG. 707.—Scirrhous carcinoma of the right breast, upper outer quadrant (below) and Stage III (p. 677).

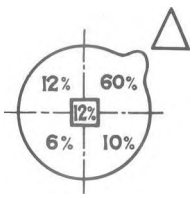


FIG. 708.—The relationship of carcinoma of the breast to the quadrants of the breast. (*Marshall and Higginbotham's statistics.*)

(*f*) *Milk Factor.*—Although an infective agent carried in the milk has been proposed there is as yet no evidence to support this.

While any portion of the breast may be attacked, the disease commences most frequently in the upper and outer quadrant (figs. 707 and 708). Unfortunately, so often the patient states that, although she noticed a lump in her breast while washing herself, she 'took no notice of it' because it was painless. Probably the average time between the patient finding the lump and reporting it is six weeks. Women should be urged to report to their doctors as soon as a lump in the breast is discovered.

Pathological Classification.—Almost all breast cancers arise in duct epithelium.[1] Some are so malignant and rapidly dividing that they transgress barriers readily and are almost everywhere seen to be extraductal and invasive. Such cells are spheroidal or polygonal from mutual compression. Others are less malignant and retain their characteristic columnar form. Such growths have less tendency to transgress barriers and are seen to be mainly intraductal. The invasive growths excite a greater or less degree of stromal reaction, both fibrous and cellular. A prominent feature of the histology is that in any one tumour there is usually an infinite variation in the pattern of the cancer cells, from the atrophic scirrhous with much fibrous tissue to the most cellular with little or none. Furthermore, while the breast lump may be an obvious scirrhous, the deposits in the lymph nodes may be highly cellular. It is possible, however, to distinguish those arising in the *nipple* (Paget's disease, p. 678), those arising in the *ducts* which are frequently papilliferous in form, and those arising in the *breast tissue* itself which are composed mainly of spheroidal cells with varying degrees

[1] Sir Hedley Atkins, p. 672.

of fibrous tissue response. A distinct type is the so-called *colloid* carcinoma where all the neoplastic cells are distended with colloid material. This tends to form a large growth but it is not highly malignant as might be expected.

THE SPREAD OF MAMMARY CARCINOMA

(*a*) **Local Spread.**—The tumour increases in size and invades other portions of the breast. It tends to involve the skin (fig. 711) and to penetrate the pectoral muscles, and even the chest wall.

(*b*) **Lymphatic spread** occurs in two ways: *by emboli*, composed of carcinoma cells, being swept along the lymphatic vessels by the lymph stream; and *by permeation*, that is, actual growth of columns of cancer cells along the lumina of the lymphatic channels. The axillary lymph nodes and the internal mammary lymph nodes are involved comparatively early. Later, the supraclavicular lymph nodes, the opposite breast, and the mediastinum, are possible resting places for itinerant carcinoma cells. Finally, they may be found in lymph nodes even farther afield.

(*c*) **Spread by the Blood-stream.**—It is by this route that skeletal metastases occur (in order of frequency) in the lumbar vertebræ, femur, thoracic vertebræ, and the skull, they are generally osteolytic pathological fractures occurring most often in a rib or a vertebra. In most instances it is by way of the blood-stream that metastases arrive in the liver, lung fields or brain from the breast, but secondary deposits may also be carried to the liver via the lymphatics within the rectus sheath and the falciform ligament. The adrenal glands and the ovaries are also common sites for blood-borne metastases.

Clinical Types of Carcinoma of the Breast.—It is difficult to give any one growth a distinct classification, and the clinical significance may be minimal. However, because the tumour may present in such a wide variety of ways, it is relevant to recognise certain clinical types. Commencing with the most malignant and passing to the least malignant, the following clinical varieties are encountered:

Mastitis Carcinomatosa (2 per cent.).—Most of these cases occur during pregnancy or lactation. The diseased breast is often painful—an uncommon symptom in ordinary carcinoma of the breast. In 50 per cent. of cases there is retraction of the nipple. The reddened skin (fig. 709) feels abnormally warm and cutaneous œdema, which indicates blockage of the subdermal lymphatics with carcinoma cells, usually extends over a considerable area, i.e. over one-third or more of the breast. This galloping form of mammary cancer, which is the result of an unbridled proliferation of cancer cells with very little fibrous reaction, sometimes is mistaken for an abscess, and incised. The main distinguishing features between the two conditions are that in mastitis carcinomatosa there is (1) more widespread œdema, (2) an absence of pyrexia and leucocytosis.

Medullary (*syn.* **anaplastic**) **carcinoma** (17 per cent.) is usually found in women between twenty-five and thirty-five years of age with well-developed breasts. While presenting many characteristics the scirrhous variety, the epithelial element of the growth is in excess of its fibrous stroma. As a consequence the tumour does not feel so hard, and in advanced cases (fig. 710)

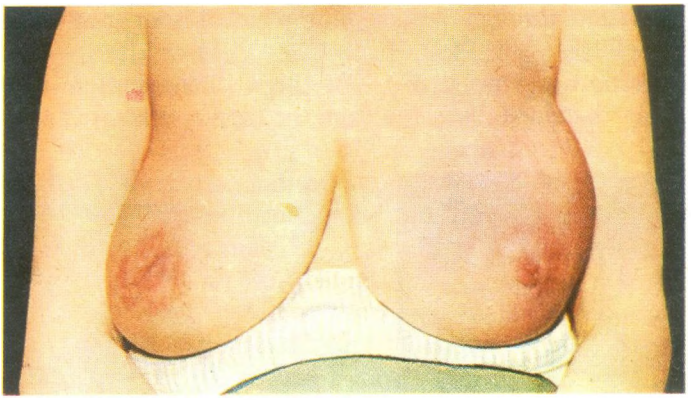


FIG. 709.—Mastitis carcinomatosa of the left breast.

it may present semi-solid areas due to degeneration of masses of ill-nourished cells.

Scirrhous[1] **carcinoma** (63 per cent.) is met with principally in middle-aged and elderly women. Owing to an abundance of fibrous tissue the lump feels very hard, while its contour tends to be irregular. The most important physical sign in breast cancer is attachment of the lump to the skin, underlying muscle, or to the nipple (fig. 711). The clinician must spare no pains to be certain whether the lump he is investigating has any such attachment (fig. 707). The importance of recent retraction of the nipple has been alluded to already (p. 661). *Peau d'orange* of the skin overlying the tumour is a late sign. Likewise is ulceration through the skin and involvement of the chest wall.

FIG. 710.—Medullary carcinoma fungating through the skin.

If a breast containing a scirrhous carcinoma is cut with a knife so as to section the tumour, the following macroscopical characteristics will be noticed:

1. The growth cuts like an unripe pear, and may grate whilst being cut.
2. Usually *both* cut surfaces are found to be concave.
3. The colour of the cut surface is definitely grey (fig. 706), and its appearance has been aptly likened to the interior of an unripe pear. A most important gross finding is small granular whitish streaks within the tumour tissue.
4. On viewing the periphery of the sectioned tumour it will be found that there is not the slightest indication of a capsule. True to its namesake, the crab, its claws have penetrated hither and thither into the breast tissue, and it is impossible to separate the tumour from the breast.

Atrophic scirrhous carcinoma (5 per cent.) is seen principally in aged, thin women with small breasts. The cellular element of the growth is comparatively sparse, its main constituent being the fibrous stroma. Although steadily progressive, the disease runs a very chronic course, perhaps taking

[1] Scirrhous—from the Greek *skiros* = hard.

ten years to ulcerate through the skin, after which it is inclined to grow somewhat more rapidly.

Duct Carcinoma (8 per cent.).—As the leading symptom in both conditions is a blood-stained discharge from the nipple, it is often impossible to

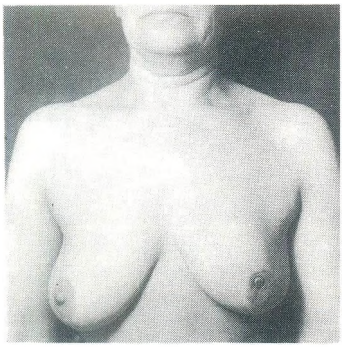


FIG. 711.—Scirrhous carcinoma of the left breast; Stage I. Nipple commencing to retract. (For staging of carcinoma of the breast, see p. 679.)

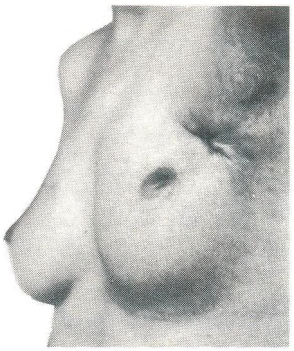


FIG. 712.—Stage IV. Nipple submerged, Skin involved. Growth is adherent to chest wall.

distinguish a duct papilloma from a duct carcinoma without the evidence of a biopsy or occasionally mammography. In the case of a duct carcinoma sometimes a small lump can be palpated behind the nipple or areola, and emerging from this there is usually a sector-shaped area of induration, viz.→

Obviously the cause of the latter is distension of those alveoli drained by the duct that is blocked by the growth.

FIG. 713.—Paget's disease of the nipple. Note also the ' peau d'orange ' due to the underlying carcinoma.

Paget's disease of the nipple (1 per cent.) is a persistent eczema-like condition that usually commences in patients over fifty years of age and does not respond to treatment. The nipple is eroded slowly and eventually disappears. As the disease progresses the areola becomes involved (fig. 713), and the erosion continues to spread peripherally until a carcinoma within the breast becomes manifest. It is agreed that Paget's disease of the nipple is due to a slowly growing duct carcinoma that infiltrates the epithelial covering of the nipple.

Microscopically the affected area is characterised by the presence of large vacuolated cells with small deeply staining nuclei in the epidermis. In the majority of cases an exhaustive microscopic search reveals the presence of malignant changes in the ducts. Sooner or later a carcinoma develops in the breast itself.

Treatment should always be mastectomy. As a rule the prognosis is extremely good. Naturally, if the patient has a palpable lump as well as nipple erosion, the prognosis is less favourable and is comparable to an invasive duct carcinoma without associated nipple change.

The differential diagnosis between simple eczema and Paget's disease depends upon the fact that simple eczema is bilateral and responds to local treatment whereas Paget's eczema is unilateral and does not so respond.

Sir James Paget, 1814–1899. Surgeon, St. Bartholomew's Hospital, London. Described this condition in 1874.

CLINICAL STAGING OF CARCINOMA OF THE BREAST

When the patient is first examined, instead of categorising the growth in such vague terms as ' early ', ' moderately advanced ', or ' advanced ', it is highly desirable to have some conventional method of expressing, in an explicit manner, the stage which has been reached. Not only does this provide a reasonably accurate indication of prognosis but it also provides a basis by which results of treatment can be compared. It is a prerequisite of any staging method that it must be accurate, and symptomless metastasis must be searched for with all available methods. Full radiological survey is essential. Bone scanning by which the uptake of technetium or a similar isotope by the skeleton is examined using a gamma camera will detect occult metastases in the presence of normal X-rays and should be used if available. Liver function tests and a liver scan may be helpful while blood investigations such as alkaline phosphatase and hydroxyproline levels may give an indication of diffuse skeletal disease.

T.N.M. Classification.—The International Union against cancer has recommended a staging system known as T.N.M. (Tumour, Nodes, Metastases) (p. 53). This system is now accepted in most centres as the basis for defining stage on clinical grounds. As a result it is possible to define four stages very comparable to the original Manchester classification, i.e.

Stage I (T1 or T2, No, Mo).—Growth confined to the breast. An area of adherence to, or ulceration of, the skin smaller than the periphery of the tumour does not affect staging. The tumour must not be adherent to the pectoral muscles or the chest wall.

Stage II (T1 or T2, N1, Mo.—Same as Stage I but there are affected *mobile* lymph nodes in the axilla of the same side. Clinical examination is notoriously inaccurate in assessing the significance of axillary lymph glands, for 26% of patients with no palpable glands have histological evidence of involvement and a rather greater percentage of patients with palpable glands have no evidence of tumour.

Stage III (T1 or T2, N2 or 3, Mo, T3 or T4, No–3, Mo).—Skin involvement or *peau d'orange* larger than the tumour but still limited to the breast, tumour fixed to pectoral muscle but not to chest wall. Homolateral axillary lymph nodes matted together or fixed to chest wall, or homolateral supraclavicular nodes mobile or fixed, or œdema of the arm.

Stage IV (T1–4, No–3, M1).—Skin involvement wide of the breast and including ' cancer-en-cuirasse ', distant spread and metastases either blood borne or lymph borne; this includes involvement of the opposite breast or axilla and deposits in bones and viscera such as lungs and liver.

PHENOMENA RESULTING FROM LYMPHATIC OBSTRUCTION IN CASES OF MAMMARY CARCINOMA

Peau d'orange is due to cutaneous lymphatic œdema. Where the infiltrated skin is tethered by the sweat ducts it cannot swell. The characteristic pitted appearance, so well likened to orange peel by French observers, has become a classical physical sign of advanced carcinoma of the breast. But it should be remembered that occasionally the same phenomenon is seen over an abscess, particularly a chronic abscess, of the breast.

Late œdema of the arm (*syn.* elephantiasis chirurgens) is a troublesome complication of radical mastectomy, especially when postoperative radiotherapy is given. The swelling (fig. 714) appears at a time varying from several months to many years after operation and, although not inevitably the case, should be considered as evidence of tumour extension until disproved. Obese patients are specially susceptible to it. It may be associated with a local infection of the arm or hand and if any such is demonstrable, it must be vigorously treated with antibiotics. The only treatment of value is elevation with the use of an elastic armlet from the wrist to the axilla (p. 25). Massage and exercises may give considerable benefit. In obese patients drastic reduction in weight is helpful.

FIG. 714.—Late œdema of the arm following radical mastectomy three years previously.

Brawny arm can result from advanced neoplastic infiltration or unremoved or incompletely removed axillary or supraclavicular lymph nodes. The œdema, which is persistent and brawny (it does not pit), is due to lymphatic blockage, but in some cases venous obstruction is a contributory cause.

A forequarter amputation is merciful in selected cases, especially in order to relieve the intense pain caused by involvement of nerves in the axilla.

Cancer *en Cuirasse*.—Here, in addition to a brawny arm, the affected side of the thoracic wall is studded with carcinomatous nodules and the skin is so infiltrated that it has been likened to a coat of armour. Usually, but not necessarily, the condition appears in cases with local recurrence after amputation of the breast. It may also follow the distribution of irradiation to the chest wall and shoulder. Various palliative measures can be tried, but the main objective is to relieve pain and mental distress for the few remaining months of the patient's life, e.g. Mist. Euphoria (Chap. 62) is strongly indicated.

Lymphangiosarcoma has been recognised recently as a complication of post-mastectomy lymphœdema. Unless there is awareness of the condition, it is likely to be confused with recurrent carcinoma of the breast.

If considered humane and advisable, deep X-ray therapy may postpone the inevitably fatal course of this complication. The only alternative is to perform an interscapulo-thoracic (forequarter) amputation (fig. 715).

FIG. 715.—Lymphangiosarcoma developed 3 years after radical mastectomy. Patient well 6 years after forequarter amputation. (*R. P. Singh, F.R.C.S., Karchana, India.*)

METHODS OF TREATMENT OF CARCINOMA OF THE BREAST

At the present time there is no agreement as to the treatment of choice for early breast cancer. A variety of operations have been described and each has its advocates. They include (i) *segmental resection*, (ii) *simple mastectomy*, (iii) *radical mastectomy to remove the axillary lymph nodes, and* (iv) *extended radical mastectomy to include the internal mammary and/or the supraclavicular lymph glands*. Each of these may be supplemented by radiotherapy or by chemotherapy. There is as yet no strong evidence that any of these methods is superior to the others and the main decision in an individual patient must be to define whether she is potentially curable or not. The presence of distant metastases whether occult or overt means that major mutilating surgery must be avoided, and it is held by authorities that local treatment for breast cancer should be directed to the ablation of the primary tumour (simple mastectomy) alone; more extensive resection (radical mastectomy) has as its objective the avoidance of unpleasant local recurrence rather than to improve overall survival. It must be said that the removal of axillary lymph glands and their subsequent histological

examination means that metastatic deposits can be confirmed and thereby their effect on prognosis assessed. Some authorities believe that the presence of such deposits indicate disseminated disease and have advocated the use of prophylactic chemotherapy with cytotoxic drugs to eradicate these micro-metastases.

In advanced disease, Stages III and IV, the interests of the patient are best served by early palliation with surgery, radiotherapy and hormone control where appropriate. For this reason it is always advisable to screen the patient for metastatic disease before deciding on the optimal treatment. It is also advisable to obtain prior histological confirmation of the tumour preferably by needle or aspiration biopsy.

Simple Mastectomy.—In this operation the excision of the breast is the same as the radical operation, but the pectoral muscles and fascia are not removed and the axilla is not disturbed.

Patey has described and practiced an extension of this operation in which the pectoral muscles are again spared but the axilla is cleared of its nodes. It is increasingly popular, because it combines the virtues of the radical mastectomy with a better cosmetic result.

Radical Mastectomy.[1]—As this operation is still widely used in the world a brief description is deemed necessary: The breast and associated structures are dissected *en bloc*, and the excised mass is composed of:

1. The whole breast.
2. A large portion of skin, the centre of which overlies the tumour, but always includes the nipple. When there is much skin involvement more skin must be sacrificed.
3. The fat and fascia from the lower border of the clavicle to, and including, the upper quarter of the sheath of the rectus abdominis, and from the sternum to the anterior border of the latissimus dorsi.
4. The sternal portion of the pectoralis major and its fascial sheath.
5. The pectoralis minor and its fascial sheath.
6. The costocoracoid membrane.
7. All the fat, fascia, and lymph nodes of the axilla.
8. The fascia over, and a few of the superficial muscle fibres of, the anterior part of the external oblique, serratus anterior, the subscapularis, the exposed portion of the latissimus dorsi, and the upper part of the rectus abdominis (fig. 716).

During the operation every effort should be made to preserve:

1. The axillary vein.
2. The cephalic vein.
3. The long thoracic nerve of Bell (nerve to serratus anterior).

The middle or long subscapular nerve (nerve to latissimus dorsi) can be sacrificed without ill effect, and this should be done without hesitation if its division enables a more thorough dissection to be carried out.

During the operation the exposed chest wall must be protected by towels wrung out in hot saline. At the completion of the operation the wound is drained.

If a wide area of skin has been sacrificed, it may not be possible to approximate the skin edges completely. A deficiency is left which is treated by immediate or subsequent skin grafting.

After operation the arm is supported upon a pillow until the wound has healed, when early movement of the arm is encouraged. The movements of the arm after so extensive a loss of muscle are surprisingly good.

Extended Radical Mastectomy.—As about 50 per cent. of cases of carcinoma in the medial quadrants and 25 per cent. in the lateral quadrants of the breast show metastases in the internal mammary lymphatic chain, a small number of surgeons feel that the internal mammary chain should be included in a radical mastectomy.

THE RÔLE OF RADIOTHERAPY IN THE TREATMENT OF MAMMARY CARCINOMA

Post-operative irradiation is not indicated when the mastectomy specimen reveals no metastases in the axillary lymph nodes. It is usually given

[1] William Stewart Halsted, 1852–1922, Professor of Surgery, Johns Hopkins University, Baltimore, was responsible for evolving the radical amputation of the breast as performed today. The operation is often known as ' a complete Halsted '

David Howard Patey, 1899–1976. Surgeon, The Middlesex Hospital, London.
Sir Charles Bell, 1774-1842. Surgeon, Middlesex Hospital, London, and founder of its Medical School.

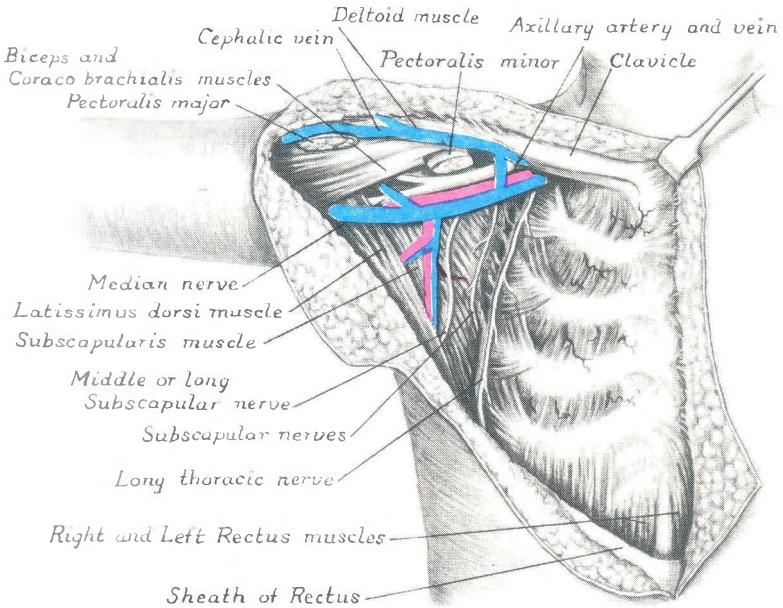


FIG. 716.—Radical mastectomy completed.

to patients who have axillary node involvement as soon as the wound has healed. The internal mammary, supraclavicular and axillary node areas are irradiated to a total dose of 4,000–6,000 rad. The criticism of radiotherapy that it may interfere with the local immune response to the disease has not been substantiated.

Treatment of Recurrences and Secondary Deposits.—Deep X-ray therapy causes disappearance or retards the progress of recurrences and secondary deposits in about one-third of cases. Examples are encountered

RESULTS OF TREATMENT

Recurrence-free Rates (all forms of treatment)

Stage	*5 years*	*10 years*
I	68 per cent.	54 per cent.
II	40 ,, ,,	25 ,, ,,
III	15 ,, ,,	4 ,, ,,
IV	2 ,, ,,	4* ,, ,,

* Includes several patients treated earlier but not accounted for in the 5-year list. (*Statistics from the Royal Marsden Hospital, London.*)

where extensive metastatic deposits are so radio-sensitive that they melt away with this form of therapy, unfortunately only to return in the same, or another site.

Unlike some forms of malignant disease, where if the patient survives five years a permanent cure can be assumed, there is no such criterion in cases of carcinoma of the breast. Patients die of secondary mammary carcinoma

(though rarely) as long as fifteen or even twenty years after operation. It is, however, abundantly clear that when the axillary lymph nodes are not involved, the prospect of many years of freedom from recurrence is much greater.

Screening for Breast Cancer.—Most authorities accept that the disappointing results which follow the treatment of breast cancer, regardless of the method used, are related to the dissemination that occurs in even early disease. It is less generally accepted that very early diagnosis and treatment materially influences the results. In some centres screening programmes which utilise clinical examination and mammography have attempted to diagnose tumours at a preclinical stage, and results are now becoming available relating to these patients. There is no doubt that the survival rates are significantly better than the hitherto accepted figures at comparable times after treatment but it is not yet possible to say whether this improvement will be maintained. It is possible, however, that population screening for high risk groups of women will become an accepted part of the management of breast cancer.

The effects of pregnancy after radical mastectomy for carcinoma are unknown but most surgeons advise against having children within 3 years of operation. Similarly the use of small doses of œstrogens for menopausal symptoms or in the contraceptive ' pill ' may accelerate the growth of any remaining tumour tissue.

THE ENDOCRINE TREATMENT OF INOPERABLE AND RECURRENT CARCINOMA OF THE BREAST

1. **Hormone Responsive.**—About 30 per cent. of mammary carcinomas are hormone responsive; that is to say that their growth is controlled partly by one or more of the hormones involved in normal mammogenesis. When their hormonal milieu is altered to one that is disadvantageous to their continued growth they cease to proliferate or metastasise and many of their component cells perish. Unfortunately this regression following hormone deprivation is never permanent.

2. **Autonomous.**—The remaining 70 per cent. of carcinomas are composed of cells which require no hormonal stimulus to enable them to thrive, multiply and metastasise. Alteration of the hormonal status has an insignificant effect and usually no effect at all on tumour growth. Even if carcinomas belonging to group one are deprived of the hormone or hormones that stimulate them, after a varying number of months or years they, too, become autonomous.

Although the fact that some breast carcinomas are hormone responsive has been known for about seventy years, there is still no direct evidence as to which hormone or group of hormones is responsible. It has always been presumed that the term hormone responsive was synonymous with œstrogen responsive and that these cancers would cease to thrive if they were deprived of naturally occurring œstrogens. Indeed, most of the endocrine treatments for advanced cancer were aimed at those organs (i.e. the ovaries and adrenal glands) that are able to secrete œstrogens. During the past few years, however, since methods have become available for the accurate estimation of steroid hormones and their metabolites and of pituitary hormones it has become apparent that the problem is complex. Attempts to define hormone responsiveness in terms of the pattern of hormone levels in the blood or urine have so far been unhelpful. More recently methods of looking for œstrogen receptors in tumour cells or of

examining the direct effect of various hormones on tumour cell cultures have been more hopeful in providing an answer. At the present state of our knowledge, however, treatment is still on an empirical basis, and although it has been found that certain combinations or sequences of drugs have a beneficial effect, we still do not know why this is so.

Oöphorectomy

This was the first treatment designed to affect the endocrine status of patients with advanced carcinoma of the breast. It was first described in 1896 by Sir George Beatson, of the Western Infirmary in Glasgow, who published an account of two cases treated in this manner. Oöphorectomy or ovarian irradiation is still used extensively as a treatment of recurrent or advanced breast cancer and is beneficial in approximately 20–30 per cent. of cases. It is of value only before the menopause and the remission so produced can last up to two years or longer.

Androgens

The administration of androgens probably accomplishes the same as an oöphorectomy although it could be argued that the former would also counteract the adrenal secretion of œstrogens. The remission rate and duration of remission are similar for the two forms of treatment. Administration is usually by injection, either in the form of testosterone propionate, 100 mg three times a week intramuscularly or one of the anabolic steroids, such as Durabolin 50 mg once a week intramuscularly. The great disadvantage of this form of treatment is the incidence of virilisation which, if severe, can be most distressing. With Durabolin, which has a more powerful anabolic and weaker androgenic effect, the incidence of side-effects is greatly diminished, and only a problem in 10 to 20 per cent. of cases. Androgens can be administered to all age groups, but are usually considered the treatment of choice for patients who are pre-menopausal or within five years of the menopause.

Œstrogens

Although the prescription of œstrogens may seem contradictory to the theories of hormone responsiveness, it is perhaps the most efficacious treatment. It should not be given unless the woman is more than five years post-menopausal. The response to the drug increases with age and may reach 60 per cent. in women over seventy. Over-all the remission rate is approximately 40 per cent. Side-effects do occur however; nausea and vomiting may be troublesome in the early stages and usually subside in time. Fluid retention and vaginal bleeding are a more persistent problem and if severe may mean that the treatment has to be stopped altogether or an alternative type of œstrogen used. It is important that the drug is administered in sufficient amounts, as there is some evidence that small doses may actually increase the rate of growth of the tumour. Stilbœstrol is usually the drug of choice, and can be administered orally in doses up to 50 mg a day. An efficient substitute is ethinylœstradiol 0·5 mg daily by mouth.

Antiœstrogens

Tamoxifen citrate acts by competition at the cellular level and appears to be effective in both pre- and post-menopausal women. It has few significant

Sir George Thomas Beatson, 1848–1933. Surgeon, Western Infirmary, Glasgow, Scotland.

side effects, and is considered the agent of first choice in recurrent disease. It may also have a place in the management of the patient with early disease in whom there is indirect evidence of micrometastases, *dose* 10 mg t.d.s.

Adrenalectomy[1]

There can be little doubt that when successful this operation can give the greatest degree of benefit to a patient. Some 20 to 25 per cent of patients have a worthwhile objective improvement, and in a further 20 per cent there will be relief of pain and subjective improvement. It is usually carried out as a one-stage procedure through either a transverse or a long paramedian abdominal incision (p. 656). In all cases bilateral oöphorectomy must be or have been carried out.

SUMMARY OF TREATMENT OF ADVANCED BREAST CANCER. (J. L HAYWARD) (IN ORDER OF PROCEDURE)

1. *Surgery.*—Localised lesions may be excised if solitary or causing symptoms.
2. *Radiotherapy.*—Especially for metastases in bone and skin.
3. *Hormones.*—(*a*) Up to menopause—ovarian ablation.
(*b*) Up to 5 years postmenopausal—ovarian ablation if there is evidence of continuing ovarian function followed by tamoxifen or androgen therapy.
(*c*) After menopause plus 5 years—tamoxifan or œstrogen therapy.
4. *Hypophysectomy* or *Adrenalectomy.*—For widespread metastases after hormone therapy has failed—corticosteroids if surgery not possible.
5. *Cytotoxins.*—Non-hormonal chemotherapeutic agents are used after hormone therapy and endocrine ablation has failed. The alkylating agent cyclophosphamide is the most frequently used single agent but recently combined treatment with up to four complementary agents (Quadruple chemotherapy) has provided worthwhile responses. Massive chemotherapy of this nature must be handled with care since the distressing side-effects may be poor palliation in patients with terminal disease.
6. *Immunotherapy.*—Non-specific stimulation of the immune defence mechanisms by means of B.C.G. or *corynebacterium parvum* has been used in breast cancer. At this stage there is no firm evidence as to its efficacy.

THE 'FOLLOW-UP' OF CASES OF CARCINOMA OF THE BREAST

It is the duty of the surgeon to examine all his cases periodically for life, so that in event of recurrence he can inaugurate such treatment as is applicable without delay. The following indicates a routine suitable for this periodic examination :

History.—This includes an inquiry as to the physical energy, general health, and the presence of an unexplained cough. Symptoms that the patient usually ascribes to rheumatism, lumbago or sciatica (humerus, spine or femur) must be regarded with suspicion, for often skeletal metastases announce themselves in this way.

Examination.—The operation field is examined for nodules. The axillæ, supraclavicular lymph nodes, and opposite breast[2] are palpated. The hand and arm are examined for œdema. The chest is percussed and auscultated and the abdomen examined for evidence of enlarged liver or ascites. If considered necessary, a rectal or vaginal examination is made in order to detect pelvic or ovarian metastases.

Radiography.—Regular annual mammography of the remaining breast and a routine radiographic examination of the chest is a valuable part of the follow-up, while in doubtful cases X-rays should be taken of any bones suspected of harbouring metastases because of pain.

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[1] Charles Huggins of Chicago was the first to carry out adrenalectomy successfully.

[2] A few surgeons recommend early simple mastectomy on the contralateral breast because 7·5 per cent. of patients with carcinoma of the breast develop carcinoma in the contralateral breast.

Charles Brenton Huggins, Contemporary. Professor of Surgery, Chicago, Illinois, U.S.A.
John Langford Hayward, Contemporary. Surgeon, Breast Unit, Guy's Hospital, London.

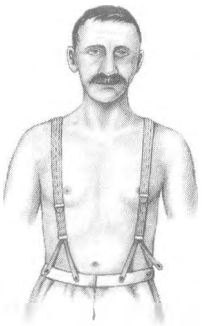


FIG. 717.—A braces button being lost, the remaining button draws the brace inwards, and the buckle rubs against the nipple.

THE MALE BREAST

The mammary tissue of the male is the leading, if not the only, example of an external secretory mechanism that persists without actual or potential function.

Mastitis of puberty is discussed on p. 665.

Mastitis from local irritation is by no means rare in men; in civil life it usually occurs in manual labourers with ill-fitting braces (fig. 717). It is not uncommon among soldiers carrying heavy equipment across their shoulders. The treatment is to remove the cause; substitution of a belt for braces is sound advice.

Gynæcomazia.—(*a*) *Idiopathic.*—Hypertrophy of the male breast may be unilateral or bilateral. The breasts enlarge at puberty, and sometimes present the characteristics of a well-developed female organ. The subjects of this deformity are often virile. Chengwayo, chief of the Zulus (fig. 718), a gynæcomast, at the age of fifty-five, had forty wives and over a hundred children. Tribal tales of a father nurturing his motherless infant with milk from his own breast belong to the realms of mythology, but pseudolactation has been observed in rare instances, and fluid akin to colostrum has been expressed.

FIG. 718.—Chengwayo, from a photograph by Schujelot.

(*b*) *Hormonal.*—Enlargement of the breasts often accompanies stilbœstrol therapy, e.g. for carcinoma of the prostate (Chap. 58); it may also occur as a result of a teratoma of the testis, in anorchism, and after castration. Rarely it may be a feature of ectopic hormonal production in a bronchial carcinoma and in adrenal or pituitary disease.

(*c*) *Associated with Leprosy.*—Gynæcomazia is very common in male persons suffering from leprosy. Possibly this is the result of bilateral testicular atrophy, which is a frequent accompaniment of leprosy (Bowesman).

FIG. 719.—Carcinoma of the male breast.
(*Dr. Y. V. Shah, Vamnagar, India*).

(*d*) *Associated with Liver Failure.*—Gynæcomazia sometimes occurs in patients with cirrhosis due to failure of the liver to metabolise œstrogens.

(*e*) May occur in patients with *Klinefelter's syndrome* (a sexual anomaly giving a chromatin positive male and infertility). Certain drugs such as digitalis, spironolactone, cimetidine and isoniazid may also cause gynæcomazia.

Treatment.—Provided the patient is healthy and comparatively young, the treatment of idiopathic gynæcomazia is mastectomy with preservation of the nipple and areola, for their absence is subject to ridicule.

Fibroadenosis and Fibroadenoma are not exceedingly rare and present the same clinical features as in the female.

Carcinoma of the male breast (fig. 719) accounts for about 0·6% of all cases of carcinoma of the breast, and generally develops in men with a pre-existing gynæcomazia. It has an evil reputation; more than one-third of cases are totally inoperable when the patient first presents. In the first place this is due to the fact that so often he does not seek advice for months or years. Secondly, the breast is so small that the enlarging growth reaches the extramammary tissues much sooner than in the female; this favours earlier metastasis.

Treatment.—Stages I and II are treated as in the female. In inoperable cases bilateral orchiectomy has an even more advantageous effect than oöphorectomy in the female, favourable results having been reported in as high as 70% of cases. Œstrogen

Charles Bowesman. Contemporary. Formerly Surgical Specialist, Lately Colonial Medical Service, Kumasi, Ghana.
Edmund William Klinefelter, Contemporary. Radiologist, Massachusetts General Hospital, Boston, Mass., U.S.A.

therapy sometimes produces amelioration after the good effects of orchiectomy have waned.

Other Tumours of the Breast

Lipoma.—A true lipoma is rare.

Pseudolipoma.—A swelling like a lipoma, if found, commonly indicates underlying breast cancer (Shucksmith and Dossett).

Sarcoma.

Sarcoma of the breast is usually of the spindle-celled variety, and accounts for 0·5 per cent. of malignant tumours of the breast. Some of these growths arise in an intracanalicular fibroadenoma. It may be impossible to distinguish clinically a sarcoma of the breast from a medullary carcinoma, but areas of cystic degeneration suggest the probability of sarcoma. On incising the neoplasm its pale, friable consistency is characteristic. Sarcoma of the breast is met with most often in women between the ages of thirty and forty. Treatment is by simple mastectomy followed by radiotherapy. Surprisingly enough the outlook in this disease is good.

Henry Samuel Shucksmith, Contemporary. Surgeon, General Infirmary, Leeds, England.
John Arthur Dossett, Contemporary. Pathologist, St. James's Hospital, Leeds, England.

CHAPTER 38

THE PHARYNX

TONSILS AND ADENOIDS

Surgical Anatomy.—The lymphadenoid tissue of the nasopharynx is Nature's barrier to bacterial invasion. The ætiology of certain cervical inflammations can be better understood if Waldeyer's inner and outer rings (fig. 720) are studied. *The faucial tonsils* are the largest and most important moieties of the inner ring. The tonsils contain tortuous crypts, which extend throughout the tonsillar substance to the external capsule. These crypts can harbour pus and micro-organisms. Clothing the lateral two-thirds of each tonsil is the capsule, a well-defined structure composed of fibrous and elastic tissue, and muscle fibres. The medial third of the tonsil lies between the pillars of the fauces and, being bereft of covering, is accessible to clinical examination. The tonsil has an exceptionally good blood supply (fig. 721). It is well to bear in mind that a tortuous *facial artery* may be closely related to the lower pole. A vein unaccompanied by an artery—the paratonsillar vein—is often a source of serious venous bleeding following tonsillectomy. When divided, the bleeding end retracts into the upper part of the tonsillar fossa, and must be found and ligated before the patient leaves the theatre.

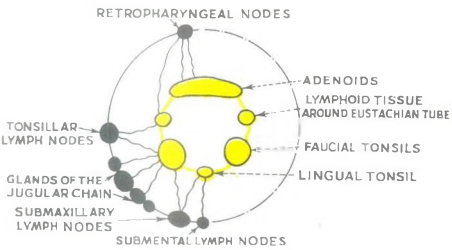


FIG. 720.—Waldeyer's rings. Inner ring—first barrier to infection; outer ring—second barrier.

ENLARGEMENT OF THE TONSILS AND ADENOIDS

Enlarged tonsils are not necessarily infected; a certain amount of hypertrophy is common in early childhood. As adult life approaches, the tonsils, together with other lymphoid tissues, tend to atrophy. Excessive hypertrophy is often bilateral. Occasionally, the tonsils are so large that they almost meet in the middle line.

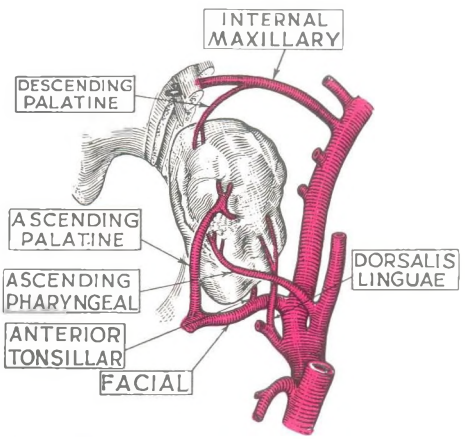


FIG. 721.—The arterial supply of the tonsil from branches of the external carotid. (*After R. H. Fowler.*)

Enlarged Adenoids[1].—The 'nasopharyngeal tonsil' is present at birth but undergoes atrophy at puberty, although remnants of it often persist into adult life. The most common period for hypertrophy is between the ages of

[1] To be exact—there is only one adenoid.

Heinrich Wilhelm Gottfried Waldeyer-Hartz, 1836–1921. Professor of Pathological Anatomy, Berlin.

four and fourteen, and a damp climate favours the development. Enlarged adenoids consist of masses of lymphoid tissue covered by ciliated epithelium and supported by a delicate framework of fibrous tissue.

Considerable adenoid hypertrophy causes the patient to snore loudly at night and to breathe through the open mouth, giving that well-known vacant expression (fig. 722). Added to this, hearing is impaired by the hypertrophied lymph-adenoid tissue obstructing the orifices of the Eustachian tubes, and infections of the middle ear and upper respiratory tract occur frequently.

FIG. 722.—The adenoidal facies. Tonsillar hypertrophy is a usual accompaniment.

Acute follicular tonsillitis is a common condition characterised by pyrexia associated with a sore throat. The cervical lymph nodes are enlarged and tender. Pain occasionally radiates up to the ears. The most usual cause is *streptococcus pyogenes*. On examination the tonsils are swollen, and yellow spots, due to pus exuding from the tonsillar crypts, can often be discerned. The condition can be distinguished from diphtheria by a rapid bacteriological examination of a smear.

Treatment.—Aspirin is administered to relieve pain and gargles of glycerol-thymol are soothing. If the symptoms persist after 24 hours, a throat swab should be taken for culture and penicillin is given until antibiotic sensitivities are known.

Chronic tonsillitis is sometimes associated with hypertrophy. During early childhood chronically inflamed tonsils are usually soft, but by the time puberty has been reached they have frequently become indurated and adherent, due to recurrent attacks of inflammation and subsequent fibrosis. The tonsillar lymph node of the jugular chain is usually palpable. Sometimes pus and debris can be expressed from infected tonsillar crypts to enable a bacteriological examination to be made.

TONSILLECTOMY[1]

Indications.—1. A history of recurrent attacks of acute tonsillitis.
2. One attack of peritonsillar abscess.
3. Chronic tonsillitis (especially if associated with cardiac, renal or rheumatic problems).
4. Biopsy for suspected malignancy.

Removal of the tonsils may also be considered in the treatment of tuberculous and non-tuberculous cervical adenitis.

Operation[2].—Tonsils are removed by dissection. Pre-operative treatment with penicillin, and for several days after operation, is a wise precaution, as 30 per cent. of patients develop bacteræmia and are in danger of developing infective lesions elsewhere in the body (e.g. bacterial endocarditis, pyelonephritis).

[1] Should a recently tonsillectomised patient contract poliomyelitis, he is liable to develop the more lethal bulbar type. Tonsillectomy is therefore contraindicated during an epidemic of this disease. The British Association of Otolaryngologists recommend inoculation against poliomyelitis before tonsillectomy is undertaken. The child should have had at least two inoculations before the operation.

[2] Suggested pre-op. drugs for children—Quinalbarbitone 6 mg./kg. (max. 200 mg.). Atropine 0·02 mg./kg. (max. 0·6 mg.).

Bartolommeo Eustachi, (Eustachius), ?1520–1574. Professor of Anatomy, Rome.

Either local or general anæsthesia can be employed. The mouth is kept open and the tongue depressed with a Davis's gag. The tonsil is seized with vulsellum forceps. An incision is made through the mucous membrane (fig. 723), and the capsule of the tonsil is exposed. The tonsil is removed by dissection, starting at the upper (palatal) pole. When the pedicle is defined, it is severed by a wire snare. Bleeding can be accurately stopped by ligating any bleeding vessels, arteries or veins, so that the patient does not leave the operating table until all bleeding has ceased. Until the patient has recovered consciousness he should be kept with his head low and well over to one side (fig. 724). On no account should he be permitted to lie on his back or be left unattended.

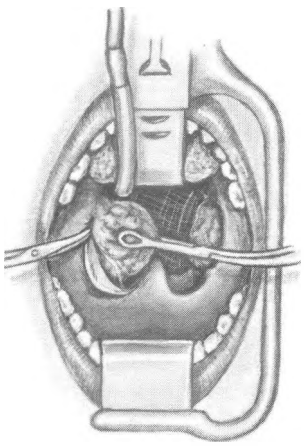


FIG. 723.—Removal of the tonsils by dissection. Davis's gag in place.

Complications after tonsillectomy

1. *Obstruction of the airway*, e.g. blood clot or the tongue falling back.

2. *Inhalation of foreign material*, e.g. blood, vomitus, broken teeth.

3. *Hæmorrhage*. Reactionary hæmorrhage occurs within the first few hours. It may be recognised by blood appearing from the corner of the mouth or nostrils, a rising pulse rate, generalised irritability. Immediate measures to be taken are: (1) Removal of clot from the tonsillar bed, (2) The application of pressure by means of a swab on a holder.

FIG. 724.—Position of patient after tonsillectomy.

If bleeding persists in spite of these measures, an intravenous infusion must be set up and blood taken for grouping and cross-matching (p. 77). The patient should be returned to the operating theatre, and under general anæsthesia the bleeding point sought and ligated. When the bleeding-point cannot be found, coaptation of the pillars of fauces with sutures will arrest the hæmorrhage.[1]

ADENOIDECTOMY

Indications.—Hypertrophic adenoids associated with: (*a*) Recurrent otitis media. (*b*) Post-nasal obstruction. (*c*) Post-nasal discharge. (*d*) Recurrent sinusitis.

Removal of Adenoids.—Removal is undertaken either before dissection of the tonsils, or at the conclusion of guillotine tonsillectomy. Adenoids are removed with a guarded curette (fig. 725) pressed against the roof of the nasopharynx (fig. 726) and then carried backward and downward with a firm sweeping movement. The after-treatment is similar to that described above. Reactionary hæmorrhage may be stopped by sitting the patient bolt upright. As a last resort, the nasopharynx may have to be packed (p. 553).

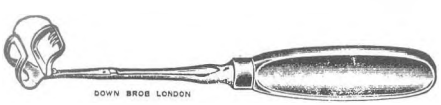


FIG. 725.—St. Clair Thomson's adenoid curette.

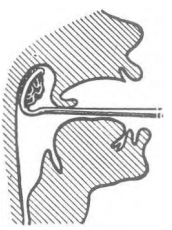


FIG. 726.—Curettage of adenoids.

[1] Morphine must not be given to children suffering from post-operative hæmorrhage. It is a factor in tonsillectomy deaths.

The original Davis's gag was invented by Dr. Davis, of Boston, Mass. Henry Edmund Gaskin Boyle, 1875–1941, anæsthetist, St. Bartholomew's Hospital, London, improved it.
Sir St. Clair Thomson, 1859–1943. Surgeon, Ear, Nose and Throat Department, King's College Hospital, London.

MALIGNANT TUMOURS OF THE TONSIL

Both carcinoma and lymphosarcoma occur in the tonsil. The diagnosis in many instances is not easy. Any unilateral enlargement of the tonsil occurring in adult life should be regarded with suspicion. A biopsy is often required to confirm the diagnosis.

Carcinoma of the Tonsil (85 per cent.).—The patient is commonly an elderly man and pain is the leading symptom. The pain is severe and radiates to the ear, and, unlike that of tonsillitis, is unilateral. The breath is foul. Later, bleeding occurs, and as the ulcer deepens the loss of blood may be copious.

Treatment is by external irradiation of the primary growth and the side of the neck as far as the clavicle. Small residues can be treated with accessory local irradiation. The five years' survival rate after treatment is 25 per cent.

Lymphosarcoma of the tonsil (15 per cent.) has the reputation of being very malignant. While this is true if it is allowed to grow beyond the peritonsillar bed, the condition is by no means hopeless in its early stages. The patient is usually between fifty and sixty years of age, complains of a lump in the throat, which in the early stages is painless. Thick speech is a common symptom, and the tonsil appears large and pale. Later, the growth spreads, and often forms a swelling of the palate, which may be mistaken for a peritonsillar abscess, and incised. Once the barrier formed by the capsule of the tonsil has been breached, the growth extends rapidly into the neck, often forming a swelling behind the angle of the mandible. While the cervical lymph nodes soon become involved, a swelling in this position is likely to be an extension of the primary growth. Eventually bleeding, dysphagia, and dyspnœa foretell that the end is not far distant.

Treatment is by external irradiation of the growth and of the lymph nodes, and some apparently hopeless cases may respond dramatically.

QUINSY (PERITONSILLAR ABSCESS)

As a rule the abscess is unilateral, but it is not uncommon for the contralateral side to become involved a few days later. The condition is rare in children, the incidence being highest in adult males. Extreme pain is experienced in the tonsillar region, radiating to the ear and to the side of the neck. Swallowing is so painful that saliva dribbles from the mouth; speech is thick and muffled. As the patient can open the mouth only to a slight extent, examination is often difficult. With good illumination, a diffuse swelling of the soft palate, mainly near the superior border of the affected tonsil, will be seen. The swelling displaces the œdematous uvula to the contralateral side.

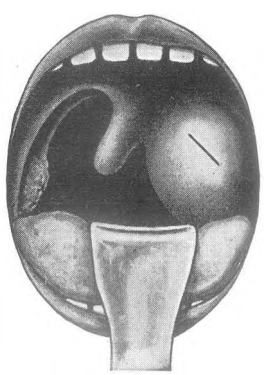


FIG. 727.—Peritonsillar abscess, showing site of incision.

Treatment, in the early stages, is the same as that for acute follicular tonsillitis (p. 689). If suppuration occurs, evacuation of pus in the following manner should not be delayed.

A small scalpel is modified by winding a strip of strapping around the blade so that only 1 cm. of the tip projects. Except in small children, no general anæsthetic is used.[1] The patient sits upright. An incision is made in the position shown in fig. 727, which is usually described as midway between the base of the uvula and the third upper molar tooth. Dressing forceps are now pushed firmly *directly backwards*. As soon as pus is encountered, the forceps are opened widely and withdrawn.

Parapharyngeal abscess is similar to the above, but the maximum swelling is behind the posterior faucial pillar, and there is little or no œdema of the palate. The abscess is opened with a really blunt instrument, such as a tongue depressor. Often the gloved finger will suffice.

[1] In adult patients surface anæsthesia (five per cent. cocaine on a pledget of wool), is the safest.

RETROPHARYNGEAL ABSCESSES

Acute retropharyngeal abscess is due to suppuration of the prevertebral lymph nodes. It is seen most commonly in children and 50% of cases occur under one year of age. The portal of entry is the tonsils, the nasopharynx, or the oropharynx. The condition may be accompanied by rigors, convulsions and vomiting. The neck is held rigidly, usually on one side, saliva dribbles from the child's mouth, and feeds are regurgitated. A croupy cough is common. The cry may resemble a 'squawk'. *Difficulty in breathing is the leading symptom* and this should always be the signal to examine a child's throat. The posterior wall of the pharynx is swollen. On digital examination a localised soft cushion-like projection can be felt on the posterior pharyngeal wall. The only condition with which acute retropharyngeal abscess may be confused is laryngeal diphtheria. A less acute form is seen in older children as a complication of otitis media.

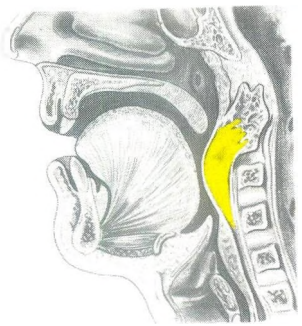


FIG. 728. — Chronic retropharyngeal abscess secondary to tuberculous disease of the cervical vertebræ.

Treatment.—No anæsthetic is used for infants, who must be placed in the prone position, with the head low. A pair of dressing forceps guided by the finger is thrust into the abscess cavity, the contents of which are evacuated. Suction must be available. Suitable antibiotic therapy is prescribed.

Chronic retropharyngeal abscess is sometimes due to an extension of tuberculosis of a cervical vertebra (fig. 728). When the collection of pus is large, in addition to the retropharyngeal swelling, there is a fullness behind the sterno-mastoid on one side. A chronic retropharyngeal abscess must never be opened into the mouth, for such a procedure will lead to secondary infection. The pus should be evacuated by an incision in front of the sternomastoid, to lead into the plane between the carotid sheath and the thyroid gland. The dissection towards the retropharyngeal space is conducted carefully until the abscess is opened. The cavity is then mopped dry and the wound closed. Suitable treatment of the underlying tuberculous lesion must then be instituted (p. 37).

DIVERTICULUM OF THE PHARYNX

Pharyngeal Pouch

Aetiology.—The pouch is a protrusion through Killian's dehiscence, that weak area of the posterior pharyngeal wall between the oblique fibres (thyropharyngeus) and the sphincter-like transverse fibres (cricopharyngeus) of the inferior constrictor muscle (fig. 729). Continued imperfect relaxation of the cricopharyngeus on swallowing and therefore repeated moments of high pressure as a bolus of food is propelled on its way to the œsophagus initiate the pouch. As it enlarges the resistance of the vertebral column behind causes it to turn outwards, usually to the left (fig. 730).

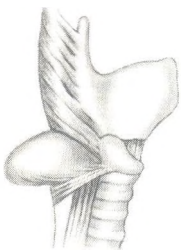


FIG. 729.—Pharyngeal pouch protruding between the thyropharyngeal and cricopharyngeal portions of the inferior constrictor of the pharynx. (*After Sir Victor Negus.*)

Clinical Features.—Patients suffering from this condition are usually elderly, and it is twice as common in men as in women.

There are three stages in the development of symptoms.

Stage 1.—There is a small diverticulum directed towards the vertebral column, viz. →

Usually it is symptomless, and the finding of it is incidental during the course of a barium swallow. Occasionally it gives rise to symp-

Gustav Killian, 1860–1921. Professor of Laryngo-rhinology, Berlin.
Sir Victor Ewings Negus, 1887–1974. Surgeon, Ear, Nose and Throat Department, King's College Hospital, London.

toms identical with those of a foreign body in the throat. At this stage the diverticulum can be ignored.

Stage 2.—The diverticulum is larger and more globular, but its mouth still lies in the vertical plane, viz.

Regurgitation of undigested food at an unpredictable time after a meal, during the swallowing of the next meal, or after turning from one side to the other at night, is the chief complaint. Sometimes the patient is awakened from sleep by a feeling of suffocation, followed by a violent fit of coughing. Infrequently an abscess of the lung results from food inspired from the pouch. Removal of the pouch is indicated.

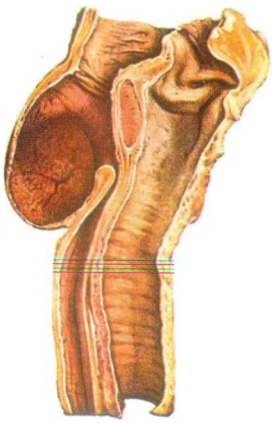


FIG. 730.—A pharyngeal pouch, showing compression of the adjacent œsophagus.

Stage 3.—The pouch has become larger, and what is so important is that its mouth looks horizontally upwards, viz.

The fundus of the pouch has become dependent, and consequently when the pouch is full it compresses the œsophagus. The symptoms of the second stage persist; in addition, there are gurgling noises in the neck, especially when the patient swallows. In about one-third of cases, the pouch is large enough to form a visible swelling in the neck : sometimes such a pouch can be seen to enlarge when the patient drinks. Nevertheless, when this stage has been reached the symptom that transcends all others is increasing dysphagia, and in a large number of instances it is this symptom alone that compels the patient to seek relief. However, even at this stage a few intelligent patients acquire a knack of overcoming their disability. It is recorded that Lord Jeffrey, a Scottish nobleman, was in the habit of emptying his pouch with a large silver spoon after every meal. Other sufferers have found that they can swallow their food better if the pouch is full, and accordingly take porridge as a first course. Notwithstanding these ingenious devices, eventually there is progressive loss of weight due to semi-starvation, and cachexia is sometimes extreme.

Radiography.—If a pharyngeal pouch is suspected, a very thin emulsion of barium should be used for the barium swallow; a thick mixture often requires much washing through a tube to remove the barium from the pouch. Quite often the fundus of the sac will be seen invading the superior mediastinum. Radiologically, the antero-posterior appearance of a barium-filled pouch can be simulated closely by a partial septum obstructing the commencement of the œsophagus. Therefore, if this mistake is to be avoided a semi-lateral view (fig. 731) also must be taken. In this view the overflow of barium emulsion into the œsophagus often can be seen to come from the top of the pouch—not from the bottom, as is the case with an œsophageal web. X-ray of the chest may reveal aspiration pneumonitis.

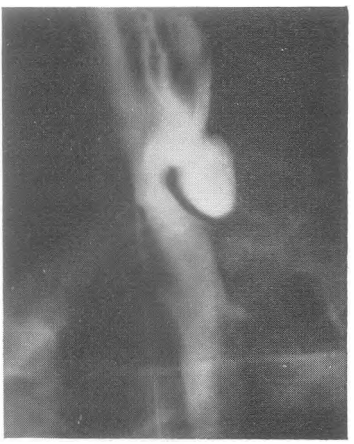


FIG. 731.—Semi-lateral radiograph of a pouch after barium has been swallowed.

Œsophagoscopy or bouginage is unnecessary for diagnosis, and may be dangerous. On many occasions the tip of the instrument has entered the pouch and has perforated its fundus, which is thin and fragile, and mediastinitis has resulted.

Treatment.—When the pouch is of a considerable size, operation is strongly advised, because progressive symptoms are inevitable. When emaciation is extreme a preliminary temporary 'feeding' gastrostomy or jejunostomy may be required.

The operation is performed in one stage. To prevent mediastinitis, antibiotics should be given before and after operation.

Operation.—Prior to skin preparation, the surgeon should inspect the pouch endoscopically, and gently pack it with ribbon gauze. At the same time a stomach tube is carefully passed into the œsophagus. These will act as helpful guides in determining the position of the sac during the dissection. The pouch is approached through either a transverse incision at the level of the cricoid cartilage, or, as many prefer, an oblique incision following the anterior border of the left sternomastoid. The first step is to mobilise the lateral lobe of the thyroid gland, therefore it is necessary to ligate and divide the middle thyroid veins, and sometimes the inferior thyroid artery. When this has been completed, the lateral lobe is retracted forwards, and the carotid sheath is retracted backwards.

The walls of the sac vary in thickness; in some cases they are so thin that great care must be taken not to tear them. Having freed the pouch completely, a cuff of the outer layer of the pouch is dissected from the mucous membrane. This permits the closure of the neck of the sac, which must be performed very accurately in two layers. It is very important not to narrow the upper end of the œsophagus at the point where the pouch is removed. In all cases a cricopharyngeal myotomy dividing the hypertrophied circular muscle must be performed. (cf. Heller's operation, p. 803). The wound is closed with drainage.

After-treatment.—The patient is fed through an in-dwelling transnasal gastric tube for three days. Fluids only are permitted for the next three days. After this, semi-solids are given, and the diet is then increased gradually.

Complications.—1. *Infection.*—Severe infection of the wound and the mediastinum is now infrequent. 2. *Pharyngeal Fistula.*—Usually the fistula closes spontaneously if the upper end of the œsophagus has not been narrowed.

Congenital lateral diverticulum is really a blind internal branchial fistula (p. 598) opening into the fossa of Rosenmüller (the pharyngeal recess). Occasionally such a fistula becomes distended and food lodges within it.

NEOPLASMS OF THE PHARYNX

Surgical Anatomy.—Concerning neoplasms of the pharynx, the clinico-anatomical division into three component parts (fig. 732) is of practical importance.

The nasopharynx (*syn.* post-nasal space, epipharynx) is that portion of the pharynx lying above the level of the soft palate which forms its incomplete floor. With the exception of this floor, the nasopharynx has rigid, immovable walls. Each Eustachian tube opens into the antero-lateral wall of the nasopharynx just behind the posterior end of the inferior turbinate. Above and behind this orifice is the supratonsillar fossa of Rosenmüller.

The oropharynx (*syn.* the mesopharynx) extends from the inferior border of the

Johann Christian Rosenmüller, 1771–1820. Professor of Anatomy and Surgery, Leipzig, Germany.

soft palate to the lingual surface of the epiglottis. In the sulcus between the back of the tongue and the anterior (lingual) surface of the epiglottis lie a median glosso-epiglottic and a right and left pharyngo-epiglottic fold. The corresponding depression on either side of the losso-epiglottic fold is known as the vallecula (fig. 733).

The laryngopharynx is the longest of the three divisions of the pharynx and it diminishes in width from above downwards. It extends from the tip of the epiglottis to end

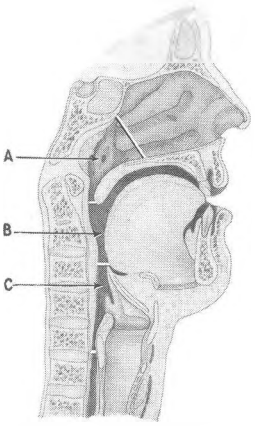


FIG. 732.—The component parts of the pharynx. A. Nasopharynx. B. Oropharynx. C. Laryngopharynx, the antero-superior portion of which contains the inlet of the larynx, and is known as the epilarynx.

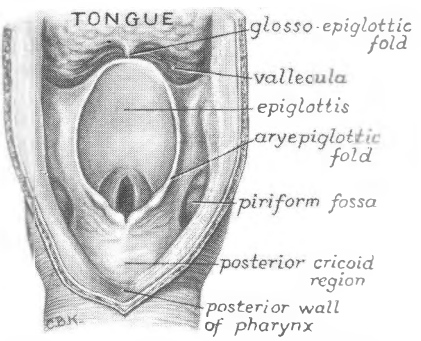


FIG. 733.—The epilarynx: the valleculæ are shown also. In, or on, all the structures labelled a carcinoma can arise.

opposite the body of the sixth cervical vertebra, where it is continuous with the œsophagus. It is convenient to subdivide the laryngopharynx into the *epilarynx* above (fig. 733) and the *hypopharynx* below, the dividing line being at the level of the upper border of the cricoid cartilage (Stanford Cade). The inlet of the larynx faces almost backwards, and is bounded above by the epiglottis, laterally by the aryepiglottic folds, and below by the short interarytenoid fold. On the lateral side of each aryepiglottic fold is a recess—the pyriform fossa.

TUMOURS OF THE NASOPHARYNX

Benign:

Angio-fibroma[1].—Although its local behaviour is the antithesis of benignity, this tumour is not malignant, for it never metastasises, neither does it infiltrate tissues. However, on account of its ability to send tentacles into first one and then the other nasal fossa, and thence into the accessory nasal sinuses, and above all because of its power to cause pressure necrosis of bone, it is a *highly destructive* tumour. As a result of these intrusions the tumour expands the nose, may fill the antra and in turn expand the cheek, and cause the palate to bulge, and at times invades the ethmoid and produces a ' frog face ' appearance.

Nasopharyngeal angio-fibroma is a reddish, firm tumour covered with normal mucous membrane. Ulceration seldom occurs unless the tumour is traumatised. Histologically it is composed of immature fibroblasts and blood-vessels; in the early stages cavernous blood-vessels predominate. In long-standing cases fibrous tissue is more plentiful.

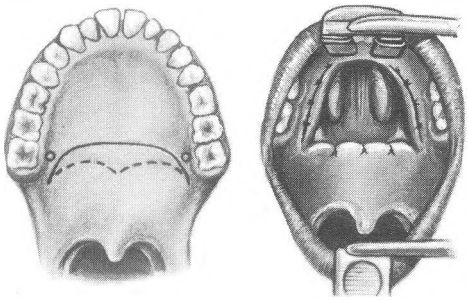
Clinical Features.—This tumour is almost confined to juvenile male patients.

Appearing at the age of puberty, the tumour usually regresses in the early twenties, provided that no secondary complications occur. Although nasopharyngeal angio-fibroma is rare, when a boy presents with progressive nasal obstruction, recurrent epistaxis, a purulent nasal discharge and a firm mass in the nasopharynx, this clinical entity should spring to mind.

Biopsy should be avoided unless there are compelling reasons for undertaking it,

[1] Formerly called 'nasopharyngeal tumour', which is ambiguous.

Sir Stanford Cade, 1895–1973. Formerly Surgeon, Westminster Hospital, London.



A B

FIG. 734.—Transpalatine approach to the nasopharynx. A. Incision. B. Permanent fenestration of the hard palate. (*After the late C. P. Wilson, F.R.C.S.*)

when matched blood must be in readiness and the surgeon must be prepared to deal with hæmorrhage.

Treatment.—Pre-operative external irradiation will shrink the tumour and render it less vascular. Removal of the tumour should follow some six weeks later.

Operation.—The transpalatal route (fig. 734A) is used. The tumour is excised with diathermy and the soft palate is replaced and sutured to the mucoperiosteum of the hard palate. Hypotensive anæsthesia and cryosurgical techniques have facilitated the procedure. Alternatively, in order that early recurrences may be seen, and destroyed by diathermy, a permanent palatal fenestra can be made (fig. 734B). The fenestra is closed by an obturator attached to a denture. The patient can then talk and eat quite normally.

A choanal polypus may give rise to difficulty in differential diagnosis. Ætiologically it belongs to nasal polypi, originating either in the ethmoid region or in the maxillary antrum. If large, it may descend through the posterior nares into the nasopharynx and present behind the soft palate. A choanal polypus may be distinguished from angiofibroma by its mobility due to the long pedicle by which it is attached, avascularity and an elastic rather than firm consistency. It can be easily removed with a snare, although the corresponding sinus may have to be opened for complete removal.

Malignant:

In China, Japan, and Malaysia malignant tumours are more common in the nasopharynx than in any other part of the body save the cervix uteri, a possible cause being the use of smoky kerosene lamps in these countries.

Type of growth	
Carcinoma	67 per cent.
Reticulosarcoma	15 ,, ,,
Lympho-epithelioma[1]	11 ,, ,,
Other tumours, including mixed salivary tumour	7 ,, ,,

(James Ewing)

Fifty per cent. of these growths arise in the lateral wall of the nasopharynx, mostly in the supratonsillar fossa of Rosenmüller (fig. 735 and p. 694); the remainder are divided equally between the roof and the posterior wall.

Clinical Features.—These depend mainly on whether the growth is obstructive (reticulosis), or infiltrating (carcinoma). Usually the *first* symptoms for which advice is sought fall into four groups:

1. *The Nasal Group.*—Slight, intermittent epistaxis and nasal speech are early nasal symptoms; other nasal symptoms, viz. a feeling of obstruction to the airway and a post-nasal discharge, usually are delayed.

2. *Aural Group.*—Unilateral deafness, with pain in the ear, is the usual

[1] A tumour containing many lymphoid elements in addition to carcinomatous elements.

ames Ewing, 1866–1943. Professor of Pathology, Cornell University, New York, U.S.A.

complaint. Obstruction of the pharyngeal orifice of the Eustachian tube by a growth leads to a collection of sero-sanguinous fluid within the middle ear. The deafness that results is relieved by paracentesis of the tympanic membrane and suction, but a fresh accumulation soon occurs. Bleeding on Eustachian catheterisation is a sign of the utmost importance.

FIG. 735.—Papillary carcinoma of the fossa of Rosenmüller, as seen in a laryngeal mirror. (*After W. L. Mattick.*)

3. *Enlarged Cervical Lymph Nodes.*—By the time the diagnosis is established, 70 per cent. of patients with a malignant tumour of the nasopharynx have enlarged cervical lymph nodes, and 40 per cent. present on account of the cervical swelling. The swelling is in the upper jugular chain and the nodes are firm, rather than hard, and may be mistaken for tuberculous adenitis.

Unlike carcinoma of the tongue, metastases more distant than the neck occur eventually in about a quarter of cases.

4. *Cranial Nerve Involvement.*—In Far Eastern patients this is the most common presenting symptom; in British patients 30 per cent. have some cranial nerve involvement when first seen. All suffer implication by the growth either at their exit through their respective foramina at the base of the skull or, less frequently, by intracranial extension of the growth. Pain of trigeminal distribution is an important feature in this group. X-ray of the base of the skull may show destruction of bone by the tumour, but it is not unusual for patients to develop root pains without X-ray changes.

Trotter's Triad.—The three cardinal symptoms of a locally invasive tumour can be summarised as follows: (i) Conductive deafness. (ii) Elevation and immobility of the homolateral soft palate—due to direct infiltration. (iii) Pain in the side of the head—due to involvement of the fifth cranial nerve from infiltration via the foramen lacerum.

Biopsy is necessary to ascertain the histological characteristics of the tumour. Inspection of the nasopharynx is facilitated if a soft rubber catheter is passed through the nose, and withdrawn through the mouth. Gentle traction displaces and immobilises the soft palate, and thereby an uninterrupted view is obtained. If more detailed inspection is desirable, Wilson's approach (fig. 734) through the junction of the hard and soft palate should be used.

Treatment.—Present practice favours supervoltage external irradiation, given to the primary tumour and to the lymph node fields on both sides of the neck at least as far down as the clavicles. Reticulosarcoma, undifferentiated carcinoma, and lympho-epithelioma are very radio-sensitive. If the primary responds satisfactorily, it is feasible to perform a cervical block dissection of the enlarged lymph nodes.

Prognosis.—Owing to their secluded position and consequent late diagnosis, and the inaccessibility of the nasopharynx, the prognosis is poor. The five-year survival rate is about 40 per cent. for the reticulosis group and under 20 per cent. in the case of carcinoma.

Wilfred Batten Lewis Trotter, 1872–1939. Professor of Surgery, University College Hospital, London.
Charles Paul Wilson, 1900–1970. Surgeon, Ear, Nose and Throat Department, Middlesex Hospital, London.

NEOPLASMS OF THE OROPHARYNX

Benign:

Diffuse cavernous angioma involving the pharynx, fauces, and often extending into the neck (where it forms a swelling) has been treated successfully by injecting into the bluish mass in the pharynx 0·5 ml. of 1 : 4 solution of ferrous chloride in sterile water. The injected area immediately turns bright red, remains swollen for a few days, and then cicatrizes. Further injections may be required. Later, if necessary, abnormal tissue is removed by diathermy, with practically no bleeding. Recently cryosurgery (p. 702) has been shown to be most effective in dealing with these lesions.

Malignant:

Usually carcinoma of the oropharynx is of the ulcerative type. The commonest site of origin is the tonsillo-lingual sulcus. There is discomfort at the back of the throat, fœtor, and blood-stained sputum. Pain is absent until the growth is far advanced.

Treatment.—The management is identical to tumours of the nasopharynx of similar histological picture.

Operation.—Although the long-term results are far from good, when possible, extirpation of the growth and the regional lymph nodes holds out a better prospect of success than irradiation.

Lateral pharyngotomy with partial pharyngectomy is performed as follows. A tracheostomy is usually performed as the first step. Then a block dissection of the cervical and retropharyngeal lymph nodes is carried out. The lateral lobe of the thyroid is then mobilised and displaced forwards. The inferior constrictor muscle is detached from the thyroid and cricoid cartilages. The great cornu of the hyoid bone and the posterior two-thirds of the ala of the thyroid cartilage are removed without opening the mucous lining.

Should the growth be situated on the lateral wall and have invaded the tonsillar region, the cervical operation is halted at this stage, and through the widely open mouth an incision is made at least 2·5 cm. distant from the accessible margins of the neoplasm with a diathermy knife, the internal carotid artery being held aside in the neck (Raven). Returning to the neck, the pharynx is opened longitudinally and that portion of the pharyngeal wall containing the growth and a wide margin of healthy tissue is removed in continuity with its lymphatics.

When the loss of the pharyngeal wall is not great the pharynx is closed. A portion of the upper part of the posterior skin flap is anchored by stitches to the prevertebral fascia, thus providing free exit to infected matter should leakage occur. The remainder of the cervical wound is closed with drainage at its lower end.

After-treatment includes transnasal intragastric feeding and antibiotic therapy.

When it is known beforehand that the defect in the pharyngeal wall is likely to be a large one, the operation is performed through a rectangular skin flap, and after partial pharyngectomy has been performed, the first stage of the plastic procedure is carried out as shown in fig. 736. In the early post-operative period, the patient is fed by a tube passed into the stomach through the fistula.

In both the procedures just described the resulting cervical fistula will require closure; this is undertaken after a short convalescence.

Growths situated in the vallecula, unless very advanced, must be treated by pharyngolaryngectomy with, if necessary, excision of a portion of the back of the tongue.

NEOPLASMS OF THE LARYNGOPHARYNX

In accordance with their site of origin, it is customary to subdivide malignant tumours of this part of the pharynx into four groups :

1. **Epilaryngeal** (20 per cent.).—Nearly always the patient is a man

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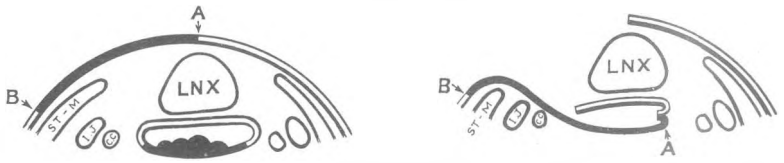

FIG. 736.—Trotter's method of reconstructing the pharynx from a flap of the skin (A–B) of the neck after excision of an extensive pharyngeal carcinoma.

between fifty and sixty years of age. The lesion is situated on an aryepiglottic fold, extending to the epiglottis or the corresponding arytenoid cartilage, and is either of the ulcerative or the papillary type. It may be symptomless in the early stages. The earliest symptom is a slightly sore throat. A muffled voice and increasing dysphagia occur later. Real hoarseness indicates involvement of the interior of the larynx, i.e. the vocal cord. Later there are attacks of dyspnœa associated with blood-stained sputum. The diagnosis is made by indirect laryngoscopy.

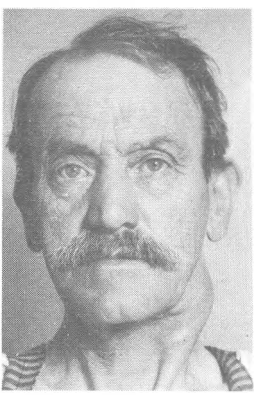

FIG. 737.—Malignant lymph nodes of the neck secondary to laryngo-pharyngeal carcinoma in the 'silent' area. The patient presented himself on account of the lump in the neck.

2. Sinus Piriformis[1] (40 per cent.).—Again, this group occurs chiefly in men about fifty years of age, and the lesion is notoriously silent. Often its first intimation is an enlarged lymph node behind the angle of the jaw. Frequently this is not heeded in its early stages (fig. 737). Exceptionally, the patient presents himself at an earlier stage, because of slight difficulty in swallowing saliva, as opposed to food. Pain is absent until the growth has involved the superior laryngeal nerve. The pain is referred to the ear. Carcinoma of the piriform fossa is nearly always of the ulcerative type. The growth may be seen with a laryngeal mirror, but sometimes its extent cannot be determined without direct laryngoscopy.

3. Lateral Wall (12 per cent.).—Once again men are attacked much more often than women. Contrary to the sinus piriformis, the growth is often papillary.

A lateral radiograph of the neck, with air inflation of the pharynx, is often more informative than a barium swallow in the demonstration of a neoplasm in this and nearby situations.

4. Post-cricoid (28 per cent.) occurs on the anterior wall of the hypopharynx at the level of the cricoid cartilage. The patient is nearly always a woman of over forty years of age, who gives a history of increasing dysphagia.

The higher incidence in women is possibly due to their ability to swallow hotter fluids and food than men can tolerate. Many of these neoplasms are secondary to the Plummer-Vinson syndrome (p. 802).[2] Indirect laryngoscopy seldom reveals the growth, which lies hidden beneath a pool of mucus. Radio-

[1] Piriformis—Latin, *pirum* = a pear.
[2] Also widely known as the Paterson-Brown Kelly syndrome.

graphic examination after a barium swallow is often helpful in determining the site of the lesion. Direct pharyngoscopy is the most informative, and allows a portion of the growth to be removed for biopsy.

Treatment of Carcinoma of the Laryngopharynx.—The condition is notoriously difficult to treat successfully. Surgery, often the treatment of choice, involves the major operation of laryngo-pharyngectomy, combined with block dissection of the neck (p. 610), the establishment of a permanent tracheostomy, and the plastic reconstruction of the pharynx. About 20 per cent of patients survive five years.

External irradiation is the alternative method of treatment and is advised for those patients who are considered unsuitable for such a major procedure, either because of their poor general condition, or because it is thought that they will not be able to adjust themselves to a life without a larynx.

Types of Operation.—Operations which combine excision and reconstruction of the gullet in one stage are preferable to staged procedures. The following is an example of a one-stage operation, in which the colon is used to replace the pharynx :

Two teams are used. The first team performs the pharyngo-laryngectomy, the block dissection of the lymph nodes of the neck on the side of the tumour, and establishes the tracheostomy. The larynx and pharynx are excised from above the level of the hyoid bone down to the level of the third or fourth tracheal ring, and including the whole of the cervical œsophagus. The stump of the œsophagus is closed by inverting sutures and allowed to slide into the thorax. The thyroid gland is included in the excision. A block dissection of lymph nodes on the less involved side may be undertaken, but the sternomastoid muscle and the internal jugular vein are usually spared.

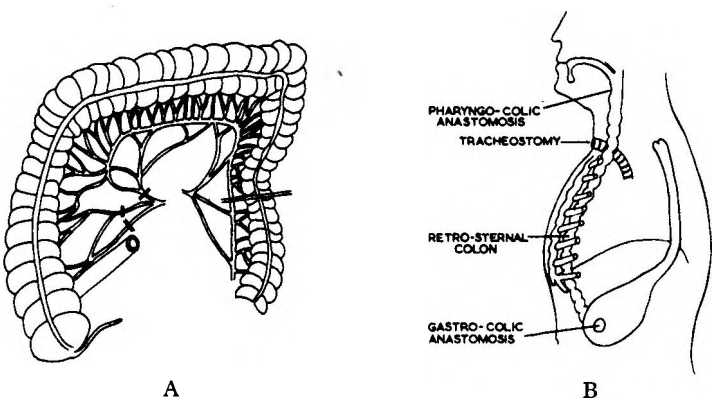

FIG. 738.—Reconstruction of the pharynx, using colon.

(*A*) Mobilisation of the colon. The middle and right colic vessels are divided. The blood supply of the colon now depends upon the left colic artery and the marginal arterial anastomoses. (*B*) The completed operation. The right and transverse colon have been passed retrosternally up into the neck. (*J. T. John, F.R.C.S., Bath.*)

The second team, working in the abdomen, mobilises the colon so that it is attached only by the mesocolon and its blood supply. The pattern of the blood supply of the colon is such that a long enough length of bowel can be obtained to join the oro-pharynx to the stomach based on a single feeding artery, usually the upper left colic artery (fig. 738A). A tunnel is made in the anterior mediastinum by stripping the pericardium, pleura, and great vessels from their sternal attachments. The colon is drawn up through the tunnel and the upper end anastomosed to the divided oro-

pharynx and the lower end to the front of the stomach (fig. 738B). Restoration of continuity of the abdominal colon is achieved by end-to-end anastomosis.

The patient is allowed to swallow fluids the following day and to eat normal foods within two weeks. Should recurrence of the neoplasm occur, radiotherapy can be given with little risk of damage to the artificial gullet and hence little interference with the patient's swallowing.

Alternatives. (i)—The colon may be drawn up through a presternal, subcutaneous route (fig. 739). (ii) Another method of reconstruction is to transect the œsophagus at the cardia closing its distal end, and mobilising the stomach to elevate it through the posterior mediastinum: then it is sutured to the pharyngeal remnant high in the neck.

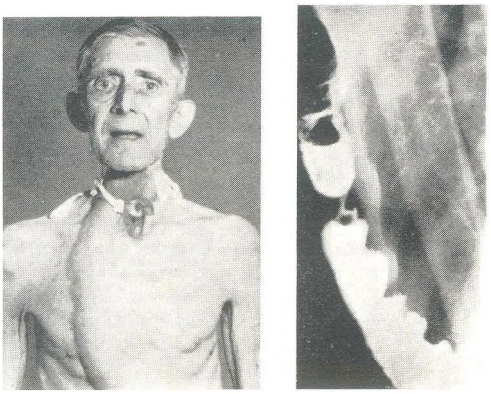

FIG. 739.—Reconstruction of the pharynx with pre-sternal colon, showing patient (*left*) and his barium swallow (*right*). (*P. V. Reading, F.R.C.S., London.*)

FIG. 740.—Perfusion with cytotoxic drugs. The tracer dye (see text) shows that the intra-arterial catheter is correctly placed for perfusion of carcinoma of the ethmoid. (*Professor D. F. N. Harrison, F.R.C.S., London.*)

CHEMOTHERAPY IN HEAD AND NECK CANCERS

Cytotoxic drugs have a limited value as an adjunct to surgery and radiotherapy in patients who have not responded, or who have recurrent growths. Before deciding to give chemotherapy, one must balance the prognosis of the disease against the expected relief and possible toxic side-effects and then consider the types of agent required and the most suitable method of administration.

The toxic side-effects of cytotoxic drugs include: bone marrow depression, alopecia, cutaneous vesiculation, and œdema. They may be lessened by giving a systemic antidote at the same time as the infusion (e.g. protective doses of folinic acid may be given intramuscularly with methotrexate).

The agents include (i) *Antimitotic drugs*, such as vinka-alkaloids[1] (vinblastine, vincristine), or bleomycin which interferes directly with D.N.A. (ii) Antimetabolites, such as methotrexate, and (iii) Alkalating agents such as nitrogen mustards which inhibit cell division by causing the chromosomes to adhere.

The method of administration may be as a regime similar to that described for carcinoma of the tongue (p. 581). Intra-arterial infusions have a possible place when dealing with tumours of the ear, nasopharynx, oropharynx, tonsils, maxillary sinuses and laryngopharynx because they lie mainly in the territory supplied by the external carotid artery, and they rarely metastasise systemically until very late. To do this it is necessary to manœuvre a catheter into the artery via the superior thyroid branch and to isolate the territory and to protect the brain from the infusate by suitably placed ligatures. An injection of a tracer dye helps (fig. 740) to confirm that it is possible to infuse the smallest effective dose to be infused without minimal spillover into the circulation. Hypothermia (Harrison) during infusion also helps to protect the brain and bone marrow.

[1] Derived from Periwinkle extracts.

Donald Frederick Norris Harrison, Contemporary. Professor of Laryngology and Otology, Royal National Throat, Nose and Ear Hospital, London.

CRYOSURGERY

Cryosurgery[1] is a technique in which tissues are exposed to extreme cold in order to produce irreversible cell damage. Alternate rapid freezing and thawing to a temperature of at least −20° C. causes multiple intra-cellular ice crystals to form which will be lethal to the cell. Its main use in surgery at present would seem to be, 1. destruction of benign vascular lesions of the head and neck (e.g. hæmangioma, angiofibroma), 2. palliation and relief of pain in uncontrolled or recurrent accessible cancers (e.g. carcinoma of nasopharynx), 3. a large number of ophthalmological procedures, including fixation of detached retina. It has also been used in pituitary gland ablation and prostatectomy.

Technique.—Modern equipment consists of a hollow probe refrigerated by liquid nitrogen or pressurised nitrous oxide. Incorporated in the probe is a rewarming device, thus tissue can be rapidly frozen and thawed as required. The probe is applied to the tissue and freezing started, and when a ' tissue ice-ball ' has formed in the area to be treated, it is then immediately allowed to thaw. The process must be repeated 2 or 3 times to achieve maximum destruction.

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[1] The idea of ' freezing surgery ' is not new; a Dr. James Arnott in 1851 at the Middlesex Hospital described the beneficial effects of a salt-ice mixture at −20°C. in the treatment of various superficial cancers.

James Moncrieff Arnott, 1794–1885. Surgeon, Middlesex Hospital, London.

CHAPTER 39

THE LARYNX

FOREIGN BODY IN THE LARYNX

IMPACTION of foreign bodies is more likely to occur in children than in adults. Cardiac arrest and death may occur.

Treatment.—If the foreign body cannot be dislodged by hooking it out with a finger, or by inverting a child and slapping its back, immediate tracheostomy is necessary in urgent cases with obstructive symptoms. In less urgent cases radiography is useful when the foreign body is radio-opaque. By the aid of direct laryngoscopy the object can be seized and removed.

ACUTE ŒDEMA OF THE GLOTTIS

Pathology.—Strictly speaking, the œdema is not of the glottis (the chink between the vocal cords) but of the aryepiglottic folds and the epiglottis, but this ancient and inaccurate term is difficult to displace.

Ætiology.—1. Extension of acute inflammation, especially acute streptococcal laryngitis or tonsillitis, diphtheria, acute parenchymatous glossitis (p. 571), and Ludwig's angina (p. 604).

2. Angioneurotic œdema.
3. Irradiation either with or without perichondritis.
4. Trauma (e.g. car accidents).
5. Indirect irritants such as corrosives, scalds or noxious gases.
6. Extension of an adjacent carcinoma.
7. Local dropsy (renal or heart failure).

The patient's voice is reduced to a hoarse whisper and some dysphagia may be present. Increasing dyspnœa occurs and frequently the œdema is sufficient to cause urgent stridor. If laryngoscopic examination is possible, the entrance to the larynx can be seen resembling the appearance of the cervix uteri.

Treatment.—Inhalation of steam and spraying with a dilute solution of adrenaline afford relief in early and mild cases. Systemic antihistamines (e.g. Phenergan) or cortisone are valuable.[1] When dyspnœa is urgent, intubation (p. 704) or tracheostomy must be performed forthwith.

TRACHEOSTOMY

The indications are as follows:

(*a*) **To relieve obstruction of the upper air passages.**

1. Impaction of a foreign body.

2. Acute infections such as: acute laryngo-tracheo-bronchitis of children, acute epiglossitis of influenzal or virus origin. Laryngeal diphtheria.

3. Œdema of the glottis.

4. Bilateral abductor paralysis of the vocal cords following injury to the recurrent laryngeal nerves during thyroidectomy.

[1] In cases of laryngeal obstruction, never give morphine. A patient under the influence of morphine stops fighting for breath, seems peaceful, and not infrequently the nurse returns to find him dead (Chevalier Jackson).

Chevalier L. Jackson, 1900–1961. Professor of Laryngology and Broncho-Œsophagoscopy, Temple University, Philadelphia, Pennsylvania, U.S.A.

5. Tumours, particularly carcinoma of the larynx.
6. Chronic stenosis following tuberculosis or scalding.
7. Congenital webs or atresias.
8. Cut throat.

(*b*) **To improve respiratory function,** by reducing the anatomical dead space, and also enabling effective aspiration of bronchial secretions to be done.

1. Fulminating bronchopneumonia.
2. Chronic bronchitis with severe emphysema.
3. Chest injuries, particularly 'flail' chest.

(*c*) **Respiratory Paralysis.**—It allows assisted or positive pressure respiration to be performed. Also secretions or inhaled foreign material (e.g. vomitus) can be aspirated.

1. Unconsciousness associated with head or facio-maxillary injuries.
2. Coma from other causes persisting for more than a few hours where there is difficulty in maintaining a free airway, e.g. barbiturate poisoning.
3. Bulbar type of poliomyelitis.
4. Tetanus. Many of these patients are, of necessity, heavily sedated, have trismus, and are in mortal danger because of inability to expectorate.

(*d*) **As a preliminary to certain operations, particularly extirpation of the larynx.**

In cases of dire emergency the operation has been performed successfully with nothing available except a penknife. If at all possible, intubation even with a small intratracheal tube, should be attempted. If successful, a general anæsthetic can be administered and a hurried procedure transformed into a deliberate, calm operation. Intubation brings the abnormally low intrapleural tension to normal and prevents the occurrence of surgical emphysema and possibly pneumothorax, especially in children (see Mediastinal emphysema, p. 706). Insertion of an intratracheal tube also permits aspiration of secretions which have accumulated below the laryngeal obstruction. Intubation will also prevent spasm of the glottis.

FIG. 741.—The position for tracheostomy.

Operation.—If a skilled anæsthetist is unavailable, local anæsthesia is employed, but in desperate cases none is required. The patient may be pinned in a blanket so that a sudden movement of the arms may not embarrass the surgeon. When preparations are complete, a rolled towel or a small sandbag is inserted beneath the shoulders, and an assistant keeps the head extended strictly in the midline (fig. 741).

The surgeon, standing at the right side of the patient, places his left index finger on the upper border of the cricoid cartilage, with the thumb and the second finger on either side of the trachea, and makes an incision vertically downwards for 2·5 to 3·75 cm., dividing skin, fascia, platysma, pretracheal fascia, and passing between the infrahyoid muscles. (When circumstances permit, a horizontal skin incision midway between the cricoid and the suprasternal notch should be used, as subsequent healing is far more satisfactory.) If seen, the isthmus of the thyroid gland is divided between hæmostats. In an emergency, hæmorrhage is ignored. A cricoid hook (fig. 742) is then inserted under the cricoid cartilage and grasped in the left hand. The hook steadies the trachea and brings it to the surface of the wound. The trachea is incised with a scalpel, the second, third, and often the fourth rings being divided: the lower the tracheostomy, the less will be the liability to laryngeal stenosis. A tracheal dilator is inserted through the tracheostoma, the cricoid hook removed, and the edges

of the tracheal wound are separated gently. In the case of diphtheria the surgeon places a swab over the wound so that the violent expiratory efforts which follow do not spray membrane, infected mucus, and blood over himself and his assistants. When respiratory efforts have become less violent, a tracheostomy tube on a pilot (fig. 742) is inserted into the trachea, the dilator is removed, and the surgeon keeps his finger on the tube while the assistant ties the attached tapes around the patient's neck. The inner tube[1] is then fixed in position, and one or two nylon or silk stitches are introduced if necessary.

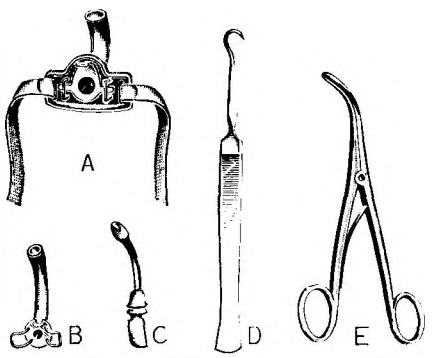


FIG. 742.—Instruments for tracheostomy (excluding scalpel and hæmostats).

A. Outer tube with tapes attached. B. Inner tube[1]. C. Pilot. D. Cricoid hook. E. Tracheal dilator.

In the case of a less urgent tracheostomy all bleeding is stopped before the trachea is opened. The injection of a few drops of 2 per cent. Xylocaine before the trachea is incised prevents the bout of coughing that follows the insertion of the tube. When the operation is performed on an adolescent or an adult, the isthmus of the thyroid gland is divided. The tracheostomy opening should be circular in shape (not just a vertical slit), by excising the edges of the incision with a scalpel strong enough to cut cartilage. A circular stoma facilitates the introduction and later changing of the tracheostomy tube, and heals well after eventual removal of the tube.

If a patient is unable to breath unaided, an *inflatable rubber or polyethylene cuff-tube* is introduced through the tracheostomy opening in order to seal off the air passage. The airway must be kept clear by frequent aspiration, assisted by postural drainage.

After-treatment.—Beside the bed is placed a trolley containing a tracheal dilator, duplicate cannulæ and introducer, retractors, and dressings. Oxygen is at hand. For the first few days a special nurse must be in constant attendance. A mechanical humidifier is essential in order to render the secretion less viscid. A sucker with a catheter attached should be at hand to keep the tracheo-bronchial tree free from secretions. The catheter must be kept sterile on a special tray covered by a sterile towel. The introduction of the catheter must be carried out under aseptic precautions by all concerned (nurses and physiotheraipsts). Unless these precautions are observed, secondary broncho-pneumonial infection is inevitable. When mucus is very tenacious, and consequently difficult to aspirate, normal saline or a detergent such as Alevaire (Bayer Products Ltd.) is administered through the tracheostomy by a fine nebuliser.

The inner tube is removed and washed in sodium bicarbonate solution every four hours, or more often if necessary. Before removal the patient should be able to sleep with the tube occluded.

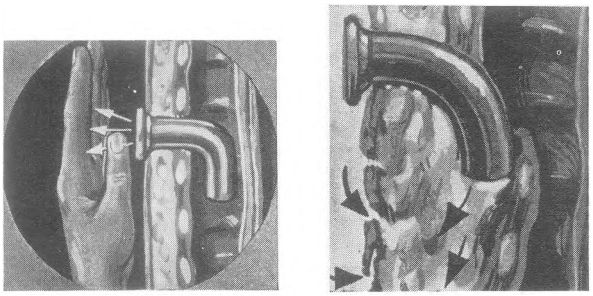
Complications:

1. *Crusting in the trachea* or main bronchi can seriously embarrass the airway, it can be cleaned by the aid of a bronchoscope passed through the tracheostoma.

[1] The inner tube is longer than the outer tube, so that the latter cannot remain obstructed when the inner tube is removed for cleaning. To carry out suction effectively, the inner tube must be of such a diameter that it will not be occluded by the passage of a No. 3 or 4 rubber catheter.

2. *Surgical emphysema* in the neck is a complication and may occur if the skin is too tightly sutured around the tube, or if the tube slips into the tissues of the neck (fig. 743).

3. *Mediastinal Emphysema.*—The cause is an abnormally low intrapleural tension—air is sucked into the tissue planes during the operation before the trachea is opened,



(*a*) (*b*)

FIG. 743.—(*a*) Tracheostomy tube in the correct position. Testing for a free airway. (*b*) The tube has slipped, or has been incorrectly positioned, so causing surgical emphysema.

causing subsequent dyspnœa and cyanosis. In severe examples the air in the mediastinum causes the mediastinal pleura to rupture, and a pneumothorax results. The diagnosis can be confirmed by radiography. Apart from oxygen therapy in high concentration, there is no specific treatment. The extravasated air is slowly absorbed.

4. *Tracheal Stenosis* is described on p. 728.

Endotracheal catheterisation is a substitute for tracheostomy (mainly cases in group (c), p. 704), but it is usually less desirable, for two reasons:

(*a*) Repeated bronchial tree toilet is performed more easily by a nurse through a tracheostomy. (*b*) Intubation granuloma of the vocal cords (p. 708) and subsequent stricture may occur.

LARYNGEAL PARALYSIS

The muscles of the larynx are innervated by the recurrent laryngeal nerves, with the exception of the crico-thyroid muscle which is supplied by the superior laryngeal nerve. Lesions of the recurrent laryngeal nerve cause the vocal cord on the affected side to lie in the paramedian position; this is due to the unopposed tensing and adducting action of the crico-thyroid. Lesions of the vagus nerve above the origin of the superior laryngeal nerve will cause complete vocal cord paralysis on the affected side. The cord will be flaccid and lie midway between abduction and adduction, which is the state and position of the vocal cords soon after death.

Ætiology.—The lesion may be central, cervical, or mediastinal. Of over-riding importance is the relation of a goitre, and especially of thyroidectomy, to recurrent laryngeal paralysis. Routine laryngoscopic examination must be made before thyroidectomy, as 3 to 5 per cent. of patients are found to have paresis or paralysis of one vocal cord, possibly due to neuritis following exanthemata during childhood, although no symptoms point to such a lesion. Pre-operative laryngoscopy is especially necessary when operating on a case of recurrent goitre. If pre-operative paralysis of a vocal cord is found, this fact *must* be recorded in the patient's notes in order to protect the surgeon from possible litigation. Pre-operative paralysis of a vocal cord with symptoms, e.g. a recent husky voice, is highly suggestive that the goitre is carcino-

matous. Other causes of recurrent laryngeal paralysis are a central lesion (e.g. tabes), carcinoma of the upper œsophagus, carcinoma of the bronchus, malignant disease of the mediastinal lymph nodes, aneurysm of the arch of the aorta (always left-sided), and peripheral neuritis. Thirty per cent. of cases are idiopathic.

Clinical Features.—Unilateral recurrent laryngeal palsy of sudden onset produces a whispering voice and occasionally some slight difficulty in

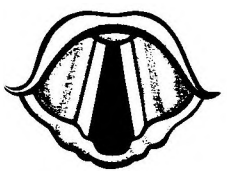


FIG. 744.—Normal larynx on inspiration. Indirect laryngoscopy.

FIG. 745.—Left recurrent nerve laryngeal palsy.

FIG. 746.—Bilateral recurrent nerve laryngeal palsy.

swallowing fluids, due to paralysis of the crico-pharyngeus on the affected side. These symptoms are short-lived and the voice may return to normal within a few weeks as the muscles in the opposite cord compensate and move it across the midline to meet the paralysed cord. Owing to this efficient compensation, in slowly progressive lesions, the patient may only experience slight weakness of the voice towards the end of the day.

Bilateral recurrent laryngeal palsy is an occasional and very serious complication of thyroidectomy. Acute dyspnœa occurs due to the paramedian position of the cords which tend to get sucked together on inspiration. Unless tracheostomy or intubation is carried out forthwith, death from asphyxia is probable.

Unilateral complete laryngeal paralysis causes a hoarse voice in which compensation does not readily occur owing to the flaccid state of the vocal cord and its lateral position. The healthy vocal cord has difficulty in meeting it.

FIG. 747.—Bilateral flaccid paralysis of the vocal cords. The cadaveric position.

Bilateral complete laryngeal palsy is an uncommon condition which occurs in lesions of the brain stem. It is usually associated with other cranial nerve lesions.

Treatment.—Tracheostomy should be performed in all cases of bilateral lesions, even when the paralysis is flaccid, for it is far better to provide the patient with a free airway than to permit him to suffer from chronic dyspnœa and its attendant evils, chief of which is the risk of asphyxia. The use of a tracheostomy tube with a recessed valve allows the patient to speak: the delicate valve opens on inspiration and closes on expiration. The expired air passes through the vocal cords so that the patient has an audible voice. With an ordinary tracheostomy tube the inspired and expired air by-passes the larynx and the patient's voice amounts only to a whisper. In cases following

thyroidectomy where the patient is otherwise in good health, the next step is to wait up to a year in the hope that one or both of the nerves will recover, otherwise extralaryngeal arytenoidectomy may be considered. It gives permanent relief of stridor at the expense of a good voice.

SIMPLE SWELLINGS OF THE LARYNX

1. **Vocal Cord Polypus.**—This is the most common of the simple swellings in the larynx and must be distinguished from true benign neoplasms. It originates in the sub-epithelial space within the vocal cord. Initial congestion is followed by localised areas of œdema and hyalinisation ; a soft, pearly grey often pedunculated mass is formed. It is easily removed endoscopically with cupped forceps.

2. **Intubation granuloma** may arise as a rare complication following the use of endotracheal anæsthetic tubes.

3. **Laryngocele.**[1]—A laryngocele is a unilateral (occasionally bilateral) narrow-necked, air-containing diverticulum resulting from a herniation of laryngeal mucosa. It originates in the laryngeal sacculus, situated in the anterior third of the laryngeal ventricle, and ascending between the false cords and the ala of the thyroid cartilage, and it herniates through the thyrohyoid membrane, and when distended forms a visible, often resonant, swelling in the neck.

The symptoms, due to a recrudescence of infection, come in attacks when the swelling, which often appears when the patient blows his nose, does not abate completely for hours or days; the explanation being that the neck of the sac becomes obstructed by mucopus. The attack often terminates with a gurgling noise and discharge of mucus into the pharynx. The sac should be excised and the neck, which is crushed, ligated, and divided, is invaginated like the stump of a vermiform appendix.

NEOPLASMS OF THE LARYNX

Benign.—Papilloma is the commonest benign tumour of the larynx.

In an adult a papilloma is usually single, and its pedicle is attached to one of the true or false vocal cords. The symptoms to which it gives rise are similar to those of carcinoma of the larynx, from which it must be distinguished. The diagnosis is made by laryngoscopic examination. Rarely, a papilloma becomes malignant; therefore the papilloma should be removed and submitted to microscopical examination.

In a child the growth is relatively common: it is usually more vascular and softer than a papilloma appearing during adult life. Moreover, implantation growths soon appear in the vicinity and may obstruct the glottis. There is a marked tendency to recurrence after removal, and the papillomas may spread to almost any site in the larynx, pharynx, or trachea.

Treatment.—Chevalier Jackson's warning was not to be too radical in the treatment of multiple papillomas for fear of damaging the vocal cords. 'Laryngeal papilloma,' he said, 'is a self-limiting disease and disappears spontaneously in early adult life provided the patient can be carried through until that time.'

Endoscopic removal with cupped forceps is the usual method of treatment.

[1] Cervical air-pouches are present in many mammals, and can be inflated voluntarily; they are exceptionally well developed in South American monkeys that utilise them for howling (howling pouches). The condition, therefore, can be looked upon as partly atavistic but mainly acquired, for it occurs in professional trumpet-players, glass-blowers, and in persons with chronic cough.

Micro-surgery of the Larynx.—This is a technique whereby the vocal cords can be inspected under magnification and benign lesions more carefully and accurately removed (Kleinsasser). The operating microscope is used with a special laryngoscope which is inserted and fixed to a clamp for stability. Healing of the cords and the post-operative voice are generally much better than after ordinary laryngoscopy.

Angiofibroma is always single, and is distinguished from a papilloma by its smooth contour (fig. 748). Except that occasionally it gives rise to hæmoptysis, it resembles a papilloma in symptomatology. In appropriate cases the condition must be distinguished from *singer's nodules*, which are nearly always bilateral. The latter condition, which produces a pearly-white nodule on the free edge of the vocal cord, is not a neoplasm, but an epithelial hypertrophy, and should, if possible, always be treated by prolonged rest (which is sometimes successful) before resorting to operation. On the other hand, an angiofibroma should be removed endoscopically with cupped forceps. Great precision is necessary, because if normal tissue is removed the speaking voice will be impaired and the singing voice ruined.

FIG. 748.—Angiofibroma of the left vocal cord. It seldom becomes much larger than depicted here.

Malignant.—Carcinoma is more common than an innocent tumour of the larynx. It usually occurs between forty and sixty years of age, and men are ten times more often attacked than women.

There are three varieties of laryngeal carcinoma:

1. *Glottic* (70 per cent. of growths), arising from the true vocal cord, which is relatively common and the most favourable type.

2. *Subglottic* (10 per cent.), below the vocal cords, with a worse prognosis.

3. *Supraglottic* (20 per cent.), originating from the ventricular bands (false vocal cords), laryngeal ventricles or the root of the epiglottis. This group has the worst prognosis.

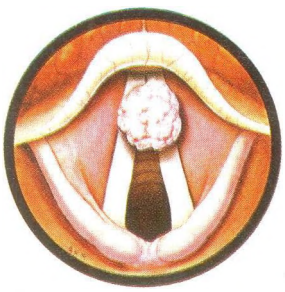


FIG. 749.—Carcinoma of a true vocal cord.

1. Carcinoma of a vocal cord (glottic) usually arises from the anterior half of one of the true vocal cords. Most frequently it is of the papillary variety (fig. 749), occasionally it is flattened, rarely it is ulcerative. Due to the paucity or absence of lymphatic vessels of the vocal cords, this type of carcinoma of the larynx remains locally malignant for a long period.

The first symptom is huskiness of the voice. The huskiness is progressive, and the patient can speak only in a low whisper, which finally gives place to aphonia. About this time the growth breaks through its confines, and metastases occur in the cervical lymph nodes and elsewhere.

The diagnosis is made by laryngoscopy examination and biopsy, and every patient with hoarseness persisting for more than three weeks should be submitted to this form of examination. According to the length of time the growth has been present, *four stages* of the disease are recognised:

i. The growth is confined to a *still mobile* vocal cord.

ii. Infiltration impairing mobility of the cord. Extension to other cord.

Oskar Kleinsasser, Contemporary. Associate Professor, Ear, Nose and Throat Clinic, Cologne, Germany.

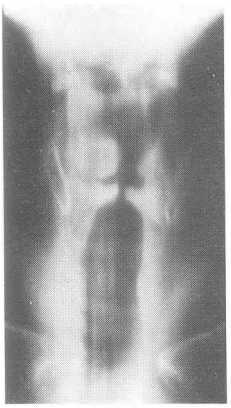


FIG. 750.—Tomogram of the larynx showing tumour of the right false vocal cord encroaching on the laryngeal ventricle. The vocal cords are not affected.

iii. Fixation of the cord. The growth has entered adjoining part of larynx. Isolated involved lymph nodes.

iv. Extension to the pharynx or skin. Lymph node metastases with fixation.

2. **Subglottic carcinoma** is a less common variety that occurs beneath the vocal cords. In this site the neoplasm grows steadily and silently, until dyspnœa develops. The paratracheal and lower deep cervical nodes, and even the thyroid gland may be involved.

3. **Supraglottic Carcinoma.**—The inital symptom is often a sense of discomfort in the larynx. Pain and hoarseness come relatively late. In 60 per cent. of cases there is cervical lymph node involvement at the time of presentation.

Tomograms are very helpful to determine the extent of the growth (fig. 750).

Treatment. — Supraglottic tumours metastasise early into the cervical lymph nodes, whereas tumours of the true vocal cords remain locally malignant for many months. For early (stage 1 and 2) carcinoma of the true vocal cord, the results of irradiation are as good as those of surgery and the voice is better. Therefore, where adequate facilities exist, irradiation (super voltage X-ray therapy, or telecobalt) is preferable to surgery. Although in the stage 1 growths there is an 80 per cent. five years' survival rate, every effort should be made to have a careful follow-up. If recurrences occur, or where the cervical lymph nodes are involved by metastatic deposits of squamous-celled carcinoma, laryngectomy, with block dissection of the lymph nodes in continuity, gives results superior to irradiation.

Total laryngectomy is performed for carcinoma of the vocal cord which is already *fixed* by infiltration, when radiotherapy fails to destroy the growth or where the growth recurs after radiotherapy.

Anæsthesia.—General anæsthesia is given through an endotracheal tube inserted via the nose. This is changed by the surgeon during the later stages of the operation after delivery of the larynx (fig. 752B).

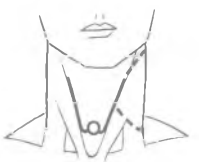


FIG. 751.

Incision.—A variety of incisions is used. A satisfactory exposure is shown in fig. 751. This may be modified if a block dissection is necessary.

Dissection.—The laryngeal cartilages and trachea are exposed by division of the thyroid isthmus. The strap muscles are divided and the inferior constrictor is carefully dissected off the thyroid cartilage. The hyoid is freed. The pharynx is opened by incisions above the hyoid bone (fig. 752A). After changing the anæsthetic tube, the hyoid is drawn forward and the mucous membrane incised down each pyriform fossa, to meet along the back of the arytenoids (fig. 752B). A nasal feeding tube is passed into the œsophagus and the defect in the pharynx is closed by interrupted sutures, and reinforced by suturing the inferior constrictors together anteriorly (fig. 752C).

The trachea is divided between the second and third rings. The tracheostomy is fashioned by suturing the trachea to the cut edges of the wound in the suprasternal notch.

For supraglottic carcinoma, subglottic tumours, and in cases where cervical lymph nodes are obviously the sites of secondary deposits, block dissection of the lymph nodes

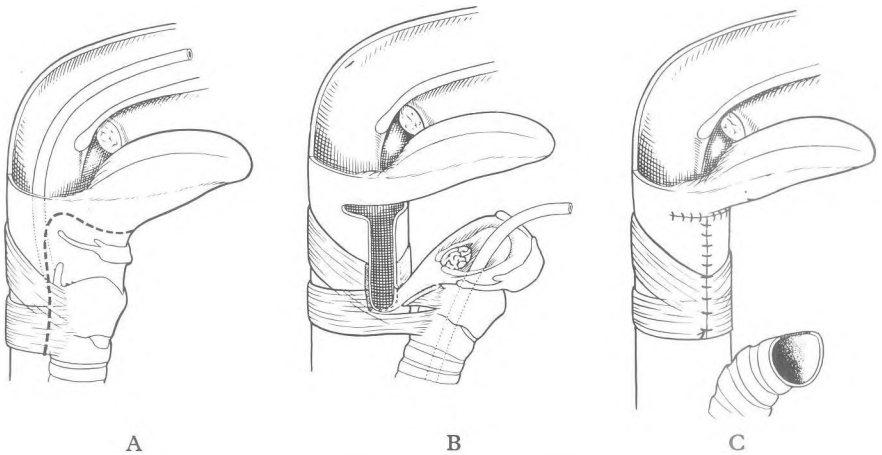


FIG. 752.—Total laryngectomy (see text).

must be combined with the laryngectomy. If the carcinoma extends into a lobe of the thyroid gland, this lobe is also removed.

Speech after Laryngectomy.—Many younger patients learn œsophageal speech, belching swallowed air for this purpose. Others require a mechanical larynx which can be fitted to the tracheostomy tube. Another type of electrically operated *oral vibrator* attached to the patient's upper denture or an artificial palate has been invented recently.

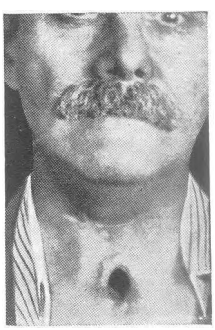


FIG. 753. Patient after complete laryngectomy. A transverse skin incision was used in this case. (*Jackson and Babcock.*)

Laryngo-fissure and Excision of the Growth.—The vocal cord must be still mobile. After preliminary tracheostomy, the thyroid cartilage is bisected and the whole vocal cord is removed. The tracheostomy tube is removed after a few days. As the vocal cord becomes replaced by fibrous tissue, the patient tends to have a gruff voice.

CHAPTER 40

THE THORAX

ANATOMY OF THE BRONCHIAL TREE

A THOROUGH knowledge of the anatomy of the bronchial tree is essential, not only for the diagnosis of many chest conditions, but for the planning and execution of the majority of pulmonary operations.

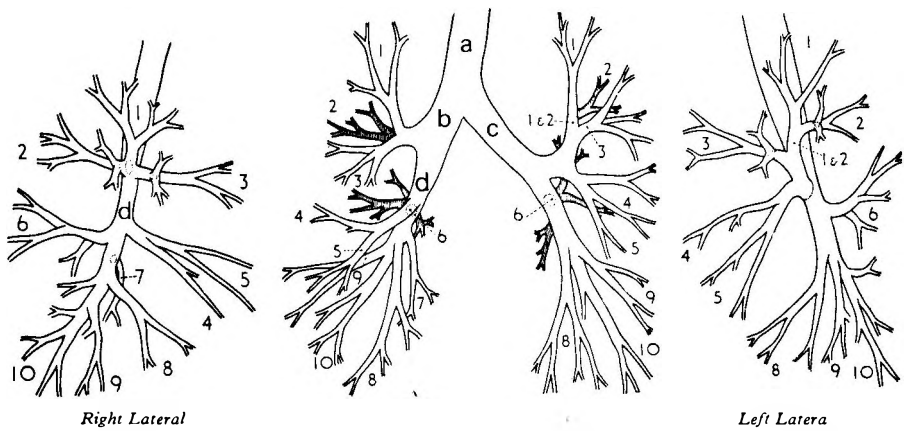


FIG. 754.—The bronchial tree with internationally adopted nomenclature.

RIGHT LUNG		LEFT LUNG	
1. Apical	Upper Lobe.	1. Apical	Upper Lobe.
2. Posterior		2. Posterior	
3. Anterior		3. Anterior	
4. Lateral	Middle Lobe.	4. Superior lingular	
5. Medial		5. Inferior lingular	
6. Apical	Lower Lobe.	6. Apical	Lower Lobe.
7. Medial (Cardiac)		8. Anterior basal	
8. Anterior basal		9. Lateral basal	
9. Lateral basal		10. Posterior basal	
10. Posterior basal			

(a) Trachea, (b) R. main bronchus, (c) L. main bronchus, (d) Intermediate bronchus.

Fig. 754 illustrates the common pattern of the bronchial tree. Each bronchus is accompanied by a branch of the pulmonary artery and together they form a broncho-vascular pedicle supplying conical segments of lung tissue. The pulmonary veins lie between the segments with tributaries issuing from adjacent segments. Each segment is largely an independent unit which can be removed (segmental resection) without interfering with adjacent segments. The veins indicate the true position of the intersegmental plane, which must be followed accurately during the operation. Two or more bronchial arteries on each side enter the lung in the areolar tissue surrounding the bronchi. They arise from the descending thoracic aorta and supply the broncho-pulmonary tissues with arterialised blood. They develop links with the smaller pulmonary vessels which, in certain diseases, may enlarge considerably.

The trachea and main bronchi with their lobar and first segmental divisions can all be examined visually by bronchoscopy. This investigation will demonstrate abnormalities in size (stenosis, obstruction or dilatation), movement (immobility or spasm), shape or position. The examination may also reveal the presence of a tumour or an intrabronchial foreign body. Material for biopsy and secretions for cytological or bacteriological examination can be obtained. Only the proximal part of the bronchial tree can be examined with the rigid bronchoscope even with the aid of a telescope. The flexible or fibreoptic bronchoscope can be introduced much farther into the bronchial tree and permits examination and biopsy of peripheral lesions. Introduced under fluoroscopic control its accuracy is further enhanced.

A more detailed anatomical study can be made by bronchography, which consists of the introduction of radio-opaque material (iodised oil) into the bronchial tree. An attempt is made to fill all the branches of the bronchial tree on one side, and radiographs are taken in two or more planes. Bronchography provides information about the size and distribution of the bronchi, and will also demonstrate bronchial occlusion or narrowing.

LUNG FUNCTION

The pulmonary alveoli enable the circulatory blood and inspired air to virtually come into contact across the alveolar wall membrane. This enables the blood to take up oxygen from the air and discharge carbon dioxide into it, thus maintaining the normal levels of these gases in the lung. Any disturbance of this mechanism may, if severe enough, lead to changes in the blood gases.

Blood gas and pH determinations measure overall lung function. Measurement of the *mechanics of ventilation* (chest expansion, vital capacity, forced vital capacity) determine the patient's ability to ventilate. The movements of air in the tracheo-bronchial tree and of blood in the pulmonary vasculature can be determined by lung scanning with either inhaled (ventilation) or injected (perfusion) radioactive xenon. In this way the relationship between ventilation and perfusion (*ventilation/perfusion ratio*) can be worked out. An outline of the pulmonary vascularity can be obtained with radio-opaque diatrizoate injected into the right ventricle (*pulmonary angiography*). Both lung scanning and pulmonary angiography are useful in the diagnosis of pulmonary embolism.

Developmental Anomalies

The trachea develops as a bud from the primitive fore-gut which soon divides into two main branches—the right and left main bronchus. Each bronchus continues to develop by branching so that all generations are present by the sixteenth week of intrauterine life. The fully developed segment has approximately 20 to 25 generations of branches. Failure of a main bronchus to develop results in *unilateral agenesis*, whilst failure of a lobar bronchus to develop produces *lobar agenesis*. Subsequent aberrations of development result in variations of the segmental pattern. Each developing bronchial bud becomes closely associated with the future pulmonary artery which covers its surface. Failure to link up with the pulmonary artery system occurs occasionally. In this case, ectopic bronchial buds develop anomalously, producing a mass of bronchial elements which often lose their connection with the parent bronchi.

Vascular supply is derived direct from the descending aorta. In adult life the condition presents as a bizarre cystic mass, usually lying in the posterior aspect of the lower lobe with large systemic arteries the size of the radial artery or even larger entering the mass through the pulmonary ligament—*intralobar sequestration.* Infection of the cystic spaces is common and is the usual cause of symptoms. Removal of the lower lobe with the offending mass is recommended.

INJURIES TO THE CHEST

Apart from road accidents and stab-wounds chest injuries are not common in civil practice ; in war, however, they constitute nearly 10 per cent. of all wounds, whilst of those killed in battle, 25 per cent. have chest injuries. They are often associated with injuries elsewhere, particularly the head and abdomen. Two main varieties are encountered—crush injuries and wounds —although in any particular patient both may be present.

Crush Injuries

These are common both in war and in civilian practice. The injury may be produced by a localised blow or a more extensive crushing force. Road accidents frequently result in severe crush injuries. Similar injuries can also be produced by concussion waves from explosions conveyed either through air or water (blast injuries).

The injuries may produce contusion of the chest wall, rib fractures, a stove-in chest, a flail chest, lung contusion or laceration, aortic rupture or myocardial damage.

The effects of a crush injury depend on its severity and the extent of involvement of chest wall, pleura and lung. The more severe forms interfere seriously with ventilation and coughing either from pain or from instability or deformity of the chest wall. The presence of blood or secretions in the bronchial tree makes ventilation still less effective. Respiration may, in addition, be depressed further by an associated head injury.

The serious interference with ventilation results in rapid and shallow respiration with cyanosis, tachycardia and hypotension. Respiration may be noisy from tracheo-bronchial secretions. Milder degrees of hypoxia produce cerebral confusion and restlessness, and this is followed by unconsciousness in severe cases.

Management in the early stages depends on a careful assessment of the damage. The chest wall is examined for paradoxical movement (multiple fractured ribs), depressions (stove-in chest) or sucking wounds. The posterior aspect of the chest should be examined as the patient is gently rolled on one side. The position of the mediastinum (trachea and apex beat) provides useful information concerning the presence of a pneumothorax, a hæmothorax or massive collapse of the lung. An early chest X-ray is imperative for accurate diagnosis of the intrathoracic damage.

Early Treatment is directed at improving ventilation by the following means where indicated :

(1) Relief of pain with repeated small doses of morphine or other analgesics, or intercostal nerve block.

(2) Stabilisation of the chest wall : (*a*) strapping can be used in mild cases and temporarily in emergencies ; (*b*) internal fixation of fractured ribs by wire or nails for more severe cases ; (*c*) tracheostomy is often effective by reducing the effort of respiration.

(3) Removal of pleural blood or air should they be large enough to compress the lung. Simple aspiration may be sufficient, but rapid reaccumulation may demand an indwelling tube or thoracotomy.

(4) Removal of tracheo-bronchial secretions may be effected by natural means after relief of pain. Bronchoscopic aspiration may be used in an emergency but if secretions persist a tracheostomy is advisable.

(5) *Tracheostomy* is the most important measure in all serious cases (see also p. 703). It permits effective and repeated aspiration of the tracheo-bronchial tree. By eliminating the dead space of the oropharynx it reduces the ventilatory demands and the work of respiration. It reduces or eliminates paradoxical chest wall movements.

Tracheostomy alone will often transform the condition of a seriously ill patient. If ventilation is seriously depressed, artificial respiration can be applied through the tracheostomy tube by mechanical ventilators. Repeated blood gas estimations are desirable to control artificial ventilation.

Tracheostomy and artificial respiration are of particular value in serious multiple injuries, especially those involving the head, in addition to the chest.

(6) Oxygen is usually necessary and is best administered by a mask.

(7) Blood transfusion, fluid replacement and vasopressor agents may be required to maintain the circulation.

Fractured Ribs.—Isolated rib fractures due to direct violence are a common complication of many varieties of chest trauma. The condition is painful but rarely disturbing. Localised tenderness and crepitus are often elicited, but the fractures may be difficult to demonstrate radiologically. Local support and analgesics are often all that is required for treatment. Intercostal nerve block may be required for persistent pain.

Multiple rib fractures (indirect or burst) are produced by crushing. The ribs fracture at the site of maximum curvature and stress, at the anterior and posterior angles. The stability of the chest wall is often greatly disturbed and in addition to pain, paradoxical movement occurs with difficulty in breathing and coughing. Ventilation is considerably hampered with the development of hypoxia and carbon dioxide retention; these changes are further aggravated by retained secretions.

Tracheostomy is advised in severe injuries; this will often correct paradoxical respiration and provide a ready route for tracheo-bronchial suction.

Stove-in Chest.—This severe injury is produced by an extensive but localised crushing force which produces multiple rib fractures and a permanent indentation of the chest wall. The affected hemithorax may be considerably reduced in size and the function of the underlying lung markedly reduced. Some paradoxical movement is usually present. The compression of the underlying lung and its relative immobility results in the accumulation of broncho-pulmonary secretions.

The minor degrees of stove-in chest may require little specific treatment additional to that already outlined for rib fracture, but the more severe forms require surgical correction. Reduction of the deformity is sometimes achieved by positive pressure ventilation, but where this proves impossible surgical elevation of the depressed ribs is required. This can sometimes be achieved by traction using percutaneous towel clips. Occasionally the depressed ribs will have to be exposed for elevation and fixation.

Crushed chests associated with a fractured clavicle are particularly serious as the deformity will increase unless the clavicle is controlled with a plate or pin.

Flail Chest.—This type of injury is seen particularly after automobile accidents of the more severe type, and is often accompanied by other serious injuries.

Multiple rib fractures result in a flaccid unstable chest wall exhibiting paradoxical movement in which the flail area is sucked inwards during inspiration and pushed out in expiration. With quiet respiration paradoxical movement may not be apparent, but if the respirations are difficult or if the patient should cough the paradox becomes more obvious. The paradoxical movement produces imperfect ventilation with hypoxia whilst defective coughing results in the retention of secretions. Any attempt on the part of the patient to overcome these defects by over-breathing or coughing only aggravates the condition.

Endotracheal intubation with positive pressure ventilation is indicated for the more severe injuries and will often control the situation. Ventilation may be required for several days until the chest wall stabilises. If ventilation is required for more than ten days a tracheostomy will be needed owing to the risks of tracheal stenosis if an endotracheal tube is used for longer periods. Alternatively the fractured ribs may be secured with the use of stainless steel wire, intramedullary nails or Kirschner wires. This should enable one to shorten the period of artificial ventilation or eliminate it altogether.

Traumatic Pneumothorax.—Air in the pleural cavity appears commonly after many forms of trauma. In the majority it is associated with blood as well (*hæmo-pneumothorax*). Air may reach the pleural cavity through a wound in the chest wall (*sucking wound*). More commonly air leaks from damaged lung; if the leak is valvular a considerable quantity of air will accumulate producing total collapse of the lung and displacement of the mediastinum towards the opposite side (*tension pneumothorax*).

Sucking wounds should be sealed with an occlusive pad until definitive surgical repair can be arranged. A tension pneumothorax can be relieved in an emergency by plunging an unmounted aspirating needle into the chest which allows the air under tension to escape. Subsequently, a small soft catheter should be introduced through a cannula and connected to a water seal or suction motor.

Traumatic Hæmothorax (see also p. 722).—Blood collects in the pleural cavity in the vast majority of chest injuries. The chest wall, lung or

Martin Kirschner, 1879–1942. Professor of Surgery, University of Heidelberg.

heart and great vessels (p. 786) may be the source of the bleeding. Bleeding is usually limited and should be dealt with by repeated daily aspirations after an initial interval of twenty-four hours to allow the bleeding vessel to seal. If the accumulation is large, earlier aspiration is necessary but, if progressive, a thoracotomy will be required to control the bleeding.

Pulmonary Contusion and Laceration.—The lung is injured in the majority of cases of moderate and severe trauma. Contusion results in areas of consolidation which will usually resolve spontaneously. Laceration permits the leakage of blood and air into the pleural cavity. Minor lacerations heal spontaneously, but more severe degrees produce persistent collapse of the lung and leakage of air and should be explored by thoracotomy. Secondary infection of the damaged lung is an important complication which can often be prevented by chemotherapy, bronchial aspirations and physiotherapy.

Fracture of Bronchus.—Severe crushing or deceleration injuries occasionally result in a transverse fracture of a bronchus which most commonly occurs at the origin of the more mobile left main bronchus. Total atelectasis and mediastinal surgical emphysema result. The condition can be confirmed at bronchoscopy. Early repair should result in a functioning lung, but delay will lead to irreversible infection and fibrosis.

Wounds of the Chest

These may be simple, or complicated by serious underlying visceral damage. In either case the wound may be penetrating or perforating (through and through wound). In all cases, however simple, it is necessary to exclude serious damage to either thoracic or abdominal viscera.

Simple wounds from stabs or bullets may give rise to little trouble—the external wound is small and usually clean. A hæmothorax is usual and will require aspirations until the pleural cavity is dry.

Complicated wounds are common in injuries with large missiles such as shell and bomb fragments. The chest wall damage may be considerable and fragments of clothing, etc., may be carried into the chest. Lung contusion and laceration is common and the mediastinal structures, the diaphragm and abdominal viscera may be involved.

Operative treatment will be required in the majority of cases, and should be carried out as soon as practicable after resuscitation. The only indication for emergency thoracotomy is uncontrollable internal or external bleeding.

Endotracheal anæsthesia is essential in order to ensure adequate ventilation and permit the removal of bronchial secretions during the operation. The chest wound is excised, rib fragments removed, and intercostal vessels ligated, if necessary. If the wound is conveniently situated, the pleural cavity can be explored through it, but if not, a separate thoracotomy is required. All blood, clot, and debris should be removed from the pleural cavity in addition to any retained foreign bodies. Lung lacerations should be sutured if small, or a formal resection carried out if more extensive. The pleural cavity should be drained through a separate stab incision. After operation it is essential that the lung should expand rapidly and completely; this is encouraged by suction drainage, breathing exercises and early ambulation.

Thoraco-abdominal Wounds.—These are always potentially serious owing to the involvement of both the abdomen and the chest; their import-

ance depends on whether or not one of the hollow abdominal viscera is damaged. The predicted course of the missile should be considered in order to anticipate the visceral damage. Apart from serious involvement of the chest, as outlined above, the main indications for exploration are signs of persistent bleeding or evidence of involvement of a hollow viscus. The thoracic approach has the advantage of permitting correction of the thoracic injuries, whilst giving adequate exposure of the upper abdomen through the diaphragm.

Shock Lung

Post-traumatic respiratory failure or shock lung may develop in 50% of patients after major trauma (Blaisdell). There is severe depression of gas exchange in the lungs due to pulmonary consolidation. Lung compliance is markedly decreased (stiff lung). Urgent treatment is required if a fatal outcome is to be avoided. Endotracheal intubation and positive pressure ventilation is instituted and large doses of steroids administered. Additionally, antibiotics and intravenous infusion are administered where indicated.

The condition is caused by micro-thrombo-embolism of small lung vessels following extensive intravascular coagulation produced by trauma. Similar changes are seen in septicæmia, after massive infusions of blood or clear fluids and following heart-lung bypass (pump lung).

DISEASES OF THE CHEST WALL

Kypho-scoliosis.—Deformities secondary to pleural and pulmonary disease are frequent unless preventive measures are taken; they can cause a considerable loss of pulmonary function, and may lead to repeated respiratory infections and cardiovascular disturbances. Vigorous breathing exercises can do much to prevent serious deformity provided they are initiated sufficiently early, but even in established cases some improvement can be expected. Primary kypho-scoliosis is considered on p. 285.

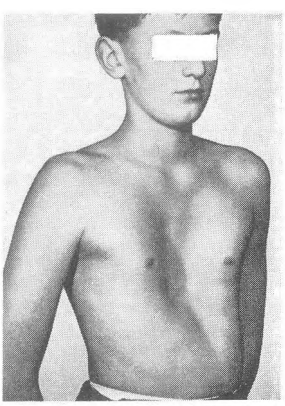


FIG. 755.—Funnel chest deformity.

Tietze's disease is a not uncommon painful and non-suppurative swelling of the second or third costal cartilages. X-ray is negative. Reassurance and not exploration is required. Local heat will often relieve pain but injections of hydrocortisone into the perichondrium may be required to encourage resolution.

Funnel Chest (Pectus Excavatum).—This deformity consists of a depression of the body of the sternum and the xiphoid process, combined with an inward curving of the costal cartilages and adjacent ribs. The deformity is usually minimal at birth, but becomes progressively more obvious during childhood. It predisposes to repeated respiratory infections and to cardiovascular disturbances, whilst the cosmetic appearances are frequently embarrassing (fig. 755).

Correction of the deformity is carried out through a midline or transverse incision: the sternum and costal cartilages are exposed and the deformed costal cartilages are resected subperichondrially. The attachments of the diaphragm to the xiphoid process are detached and the sternum separated from the mediastinal tissues. A wedge osteotomy of the anterior table of the sternum is performed at the upper limit of the deformity, and the mobilised sternal body can then be easily elevated. The sternum should be maintained in an elevated position by a stainless steel strut which is placed beneath the sternum with the ends resting on the ribs laterally. The strut is

F. William Blaisdell, Contemporary. Surgeon, San Francisco General Hospital.
Alexander Tietze, 1864–1927. Surgeon, Breslau, Germany (now Wroclaw, Poland).

not removed for at least a year. The use of internal splinting has greatly improved the long-term cosmetic results.

Pigeon chest (Pectus carinatum) and other deformities may require surgery for cosmetic reasons. The deformed ribs or cartilages should be resected.

Cold Abscess

The majority of cold abscesses of the chest wall are secondary to tuberculous intercostal lymphadenitis. A minority are associated with Pott's disease of the spine and tuberculosis of the ribs or sternum. The intercostal lymph nodes are situated posteriorly near the neck of the rib, or anteriorly in association with the internal mammary vessels. Tuberculous pus, forming in these sites, may track a considerable distance in the intercostal space before becoming subcutaneous. It usually reaches the superficial tissues by following the lateral or anterior branches of the intercostal vessels, and thus most commonly presents in the anterior axillary line or the parasternal region. The abscess has all the characteristics of a tuberculous cold abscess. Involvement of the skin and rupture is common in neglected cases, resulting in a persistent discharging sinus surrounded by typical tuberculous granulation tissue. A cold abscess must be distinguished from a lipoma or an empyema necessitatis; in the latter, the swelling is often tender and may exhibit an impulse on coughing, and is associated with the physical and radiological signs of an empyema.

The abscess should be treated by repeated aspirations through healthy skin with the instillation of streptomycin. If the abscess fails to respond to repeated aspirations, surgery will be required. The extent of the ramifications of the abscess should first be determined by the injection of iodised oil into the abscess. Ideally, the abscess and its ramifications should be completely excised, together with any secondary involved bone; but if this is not practicable, the abscess should be evacuated, granulation tissue removed and the wound closed. Chronically discharging sinuses can sometimes be cured by regular instillations of streptomycin; alternatively, excision of the track is advisable. All patients should have a long course of chemotherapy.

Empyema Necessitatis

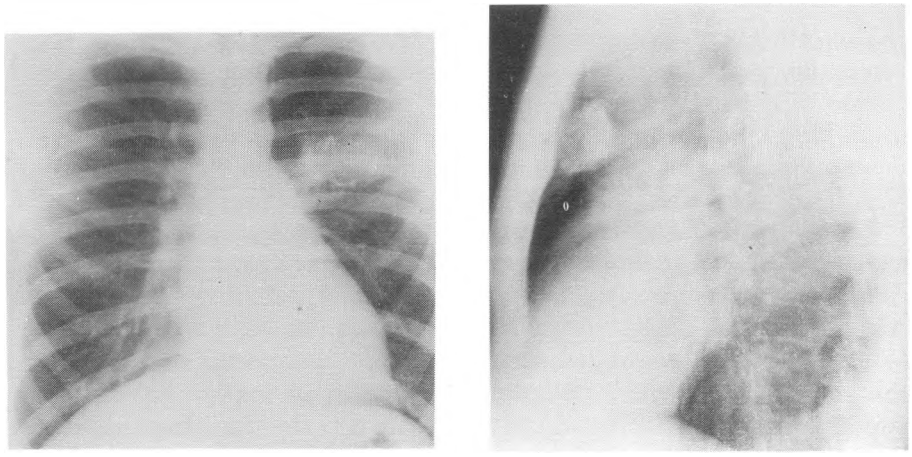
This is caused by an empyema perforating the chest wall and presenting with a subcutaneous collection of pus communicating, often by a tortuous channel, with the main pleural collection. The condition is seen either with a neglected or an undiagnosed empyema, or following aspirations of thin, highly infective pus from an empyema. In the latter case, the superficial tissues are infected by the seepage of pus through a needle track. The site of the subcutaneous abscess does not always correspond to the site of pleural perforation, as the abscess may track along the intercostal spaces before becoming superficial. The signs are those of a diffuse, fluctuant, tender swelling which may exhibit an impulse on coughing, and is associated with the clinical and radiological signs of an empyema. Treatment is primarily aimed at the empyema, which should be aspirated and drained. The superficial abscess may disappear as a result of aspirating the empyema, or may itself require separate aspirations or drainage.

Tumours of the Ribs

Rib tumours may be benign or malignant, and the latter either primary or secondary.

Percivall Pott, 1714–1788. Surgeon, St. Bartholomew's Hospital, London.

Benign tumours are frequently discovered by routine radiography. Many are symptomless and produce no external evidence of their presence. A few produce pain or an external swelling. The commonest is the chondroma (fig. 756(*a*) and (*b*)), whilst osteochondroma, multiple exostoses, fibrous dysplasia,

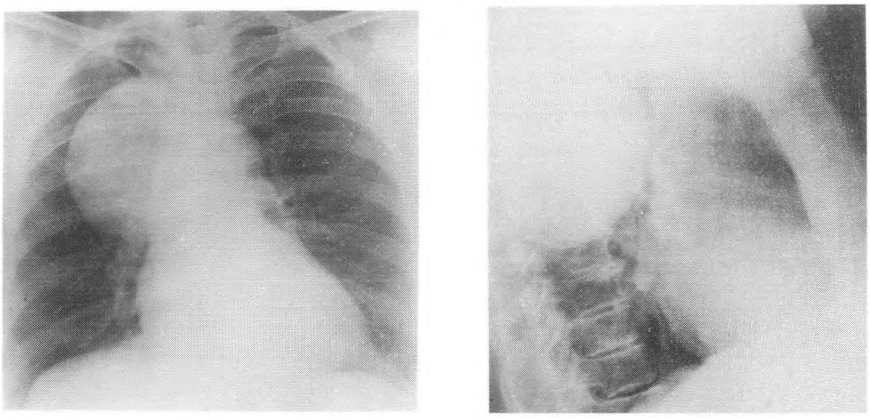


(*a*) (*b*)

FIG. 756.—(*a*) Calcifying chondroma of anterior portion of Left III rib. (*b*) Lateral view.

lipoid granulomas and multiple myelomas are less common. Benign tumours usually produce expansion of the rib and are less dense radiologically than normal rib, whilst pathological fracture is rare. Malignant change can occur, especially in chondromas, and removal is advisable.

Primary malignant tumours are rare, the commonest being chondrosarcoma; they should be widely excised. Any sizeable deficiency of the thoracic cage which might result in paradoxical movement should be repaired with marlex mesh, tantalum gauze etc. *Secondary deposits* in rib are particularly common in carcinoma of the lung and with breast cancer. The de-



(*a*) (*b*)

FIG. 757.—(*a*) Large neurofibroma of chest. (*b*) The lateral film shows the tumour lying posteriorly.

posits are almost always painful and tender. They result in destruction of bone, and pathological fracture is common. X-ray therapy will usually relieve the pain.

Neurogenic Tumours (see also p. 761).

These are of two main types—neurofibroma (fig. 757(*a*) and (*b*)) arising from the intercostal nerves and ganglioneuroma from the sympathetic chain. The former appear close to the neck of the rib, whilst the latter lie slightly more medially applied to the vertebral bodies. The majority are found on routine radiography. They are usually symptomless and single. Their growth is slow and the adjacent ribs and vertebral bodies are often distorted and eroded. A small percentage extend through the intervertebral foramen into the vertebral canal, where compression of the spinal cord may result in paraplegia (dumb-bell tumour). Removal of the tumour is safe and simple and is recommended owing to the risks of malignant changes. Dumb-bell tumours, and particularly those with spinal-cord involvement, require urgent treatment. A small intraspinal extension can easily be removed from the chest, but extensive intraspinal tumours will require laminectomy for their removal; the latter can usually be carried out at the same time as the chest operation, but if very extensive, separate operations are advisable.

DISEASES OF THE PLEURA

Pneumothorax —A pneumothorax is produced by the presence of air between the layers of the pleura. The air may be present alone or associated with serous fluid (*hydro-pneumothorax*), pus (*pyo-pneumothorax*), or blood (*hæmo-pneumothorax*). Air may be deliberately introduced into the pleura, in the treatment of pulmonary tuberculosis (*artificial pneumothorax*), or be associated with trauma (*traumatic pneumothorax*), or may appear without any obvious exciting cause (*spontaneous pneumothorax*). The physical signs of a pneumothorax are a hyper-resonant percussion note with absent breath-sounds. Radiographs reveal translucency on the affected side with absence of lung markings; the edge of the collapsed lung is usually visible.

Spontaneous Pneumothorax.—Causes :

(*a*) Tuberculous. (*b*) Non-tuberculous:— (1) *Emphysematous bullæ.* (2) *Solitary lung cysts.* (3) *Honeycomb or cystic lung.* (4) *Idiopathic.*

Tuberculous spontaneous pneumothorax is usually associated with obvious clinical or radiological signs of tuberculosis. The condition is produced by the rupture of a small subpleural tubercle or cavity, and is usually associated with infection or irritation of the pleura resulting in the appearance of fluid in the pleural cavity and a febrile reaction. Treatment depends on the underlying pulmonary lesion.

A non-tuberculous spontaneous pneumothorax by contrast is rarely associated with fluid formation or fever. In a minority an obvious underlying pulmonary lesion, such as an emphysematous bulla, a lung cyst, or a honeycomb lung, can be demonstrated. Absorption may occur spontaneously and rapidly, or may be considerably delayed (*chronic spontaneous pneumothorax*). Attacks may be single or repeated (*recurrent spontaneous pneumothorax*). With a first attack, in the absence of an underlying cause, air can be allowed to absorb spontaneously, or expansion can be encouraged by occasional aspirations. Should tension occur, however, air should be aspirated, but if reaccumulation is rapid, a fine catheter should be introduced into the pleural

cavity and attached to an under-water seal or suction motor. A soft catheter is preferable to a rigid needle or cannula as it will not lacerate the lung as it expands.

Failure of absorption (*chronic spontaneous pneumothorax*), or repeated attacks (*recurrent spontaneous pneumothorax*), demand full investigations to determine the cause. The most useful investigations are tomography and thoracoscopy (inspection of the lung.) These investigations may reveal:

(*a*) A localised lesion, such as a cyst.
(*b*) Generalised lesions, such as emphysema or honeycomb lung.
(*c*) Nothing abnormal.

A localised condition is best treated by thoracotomy and excision, but the latter two groups should be treated by artificial obliteration of the space (pleurodesis). This is achieved by the use of chemical or other irritants (5 per cent. silver nitrate, iodised talc, 0 5 per cent. camphor in oil) introduced either through a needle or applied to the lung surface at thoracoscopy. These irritants set up a diffuse pleuritis which will result in the fusion of the visceral to the parietal pleura if the two surfaces can be maintained in apposition. It is thus important to ensure that the lung expands rapidly and completely after pleurodesis by using catheter drainage with suction. Thoracotomy is preferred by some for all cases. The lung leak is oversewn, cysts if present are excised and the parietal pleura at the apex is removed or abraded in order to ensure fusion of the lung to the chest wall and prevent recurrence.

Hæmothorax.—Blood in the pleural cavity may occur in a variety of conditions and is often associated with air as well. The respiratory and cardiac movements defibrinate the blood as it reaches the pleural cavity so that the collection remains fluid. Massive clotting only occasionally occurs. Blood is a pleural irritant and its presence produces pain and shock in the early stages and later excites the formation of a considerable effusion. It is also an excellent culture medium and infection is relatively common.

Causes.—(1) Trauma (fig. 758).

(2) Post-operatively, following pulmonary, cardiac or œsophageal operations, thoracoscopy for division of adhesions, cervical sympathectomy and after refills of an artificial pneumothorax.

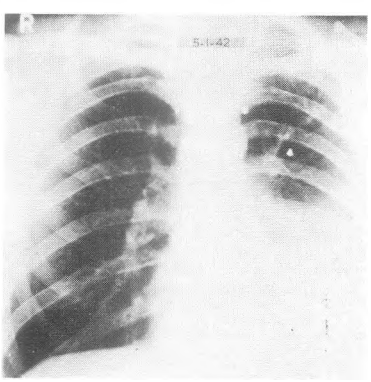


FIG. 758.—A small left hæmothorax with two retained metallic foreign bodies.

(3) Associated with new-growths of lung, mediastinum or pleura.

(4) Leaking aneurysms.

(5) Spontaneous.

The signs are those of a collection of fluid in the pleura, and the diagnosis is confirmed by exploring with a needle.

Treatment.—The initial treatment should be aimed at relieving pain, shock and blood loss. If signs of persistent bleeding are present, thoracotomy is advised. In all other cases the aim should be to remove the blood by aspiration as early and as completely as possible. Aspirations can be safely started twenty-four hours after the onset and should be repeated daily until no more fluid is obtained and the X-rays appear clear. Accidental or deliberate introduction of air must be

avoided at all costs as it will lead to further collapse of the lung. Early and vigorous breathing exercises are advised.

A clotted hæmothorax is diagnosed when the aspirating needle fails to remove a collection of blood. Liquefaction of the clot is often possible with streptokinase or Trypsin ferments (Trypur). An initial dose of 200,000 units of streptokinase or 1 ml. of Trypur is injected into the clot and aspirations performed after twenty-four hours. Further doses can be given if the initial injection is not effective. In many instances however, the result is unsatisfactory, and thoracotomy with evacuation of clot and decortication of the lung is required.

An infected hæmothorax should be treated initially with repeated aspirations and the instillation of suitable antibiotics. Early drainage or decortication will probably be required, owing to the difficulty of sterilising and aspirating the hæmothorax if clotting has occurred.

Pleural Effusion.—The pleural cavity is constantly bathed in fluid which exudes from the visceral pleura and is absorbed by the parietal pleura. Any disturbance of the finely adjusted balance may result in the accumulation of fluid in the pleural cavity. Disturbances in osmotic or hydrostatic pressures produce a transudate (protein less than 3 g/100 ml.); inflammatory lesions produce an exudate (protein more than 3 g/100 ml.) and neoplasms produce variable changes and the fluid is often blood stained.

Pleural fluid when first formed gravitates to the most dependent part of the chest (usually posterior costo-phrenic angle), but later adhesions may lead to loculation.

Pleural fluid produces stony dullness with absent breath sounds and vocal fremitus with displacement of the mediastinum to the opposite side. Diagnosis can only be confirmed with the aspirating needle.

In all cases the ætiology must be determined by (*a*) bacteriological, biochemical and cytological analysis of fluid, (*b*) needle biopsy of pleura (Abrams), (fig. 759), (*c*) thoracoscopic examination and biopsy of pleura, or (*d*) open thoracotomy.

In addition to these local measures a careful assessment of the lung and the

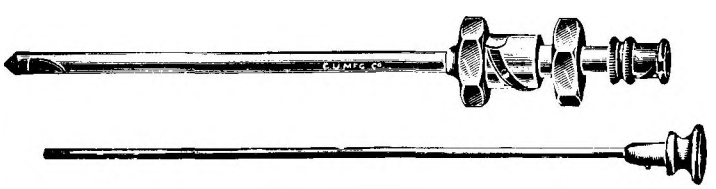


FIG. 759.—Abrams' Pleural Biopsy Punch.

heart is required and a search should be made for distant neoplasms (ovarian carcinoma, ovarian fibroma—Meigs' syndrome).

Pleural Tumours.—These may be benign or malignant and primary or secondary. A benign fibroma is rare, presents as a rounded shadow in the chest X-ray in an otherwise symptom-free patient. Finger clubbing is common. Malignant mesothelioma may affect the pleura diffusely, is often accompanied by pains and associated with a pleural effusion. These lesions are often associated with the inhalation of asbestos dust. Secondary pleural tumours are most often bronchial in origin, but the breast and ovary are also implicated. Secondary lesions are often associated with large, recurring blood-stained effusions.

Leon David Abrams, Contemporary. Thoracic Surgeon, United Birmingham Hospitals, England.
Joe Vincent Meigs, 1892–1963. Emeritus Professor of Clinical Gynæcology, Harvard University, Boston, U.S.A.

Chylothorax.—Chyle may leak into the pleural cavity from the thoracic duct or its tributaries as a result of trauma (stab wounds, crush injuries, surgical injury) or obstruction by tumour. It is commonly mistaken for pus.

Chyle contains fat and the diagnosis depends on its demonstration by naked eye or microscope using Sudan III to stain the fat globules. Confirmation of the presence of chyle can be obtained by cholesterol and triglyceride estimations of the suspected fluid compared with fasting serum. Lipoprotein electrophoresis will reveal the presence of chylomicron which is present in chyle.

Pseudochylous effusions have a milky appearance not due to fat (cholesterol and calcium phosphate crystals, filarial parasites).

Chyliform effusions contain fat, but this has been derived from the breakdown of cells in encysted effusions. Distinction between chylous effusions and pseudo-chylous or chyliform effusions is possible using lipophilic dyes. A suitable dye is mixed with butter or oil and taken by mouth. Twelve to twenty-four hours later aspiration of the suspected fluid will be appropriately coloured if it be chylous. This technique can be used to aid identification of a leaking duct at thoracotomy.

Management will depend on the cause. The majority of surgically produced chylous effusions and some traumatic ones resolve with conservative measures (low fat diet and chest aspiration). If accumulation continues after a trial period of two weeks, thoracotomy is indicated to locate and suture or ligate the severed duct.

Empyema.—An empyema is a pleural abscess and consists of a collection of pus in the pleural cavity. The term is often wrongly used in a much wider sense to include all phases of pleural infection from an infected turbid effusion to a mature abscess containing thick pus. In the management of an empyema, it is just as important to consider the degree of localisation and pus formation as it is in other forms of septic cellulitis.

Ætiology.—An empyema is never primary. The majority are secondary to pulmonary infection, particularly pneumococcal pneumonia and bronchopneumonia, but any infective process, such as tuberculosis, lung abscess and bronchiectasis may be complicated by an empyema. Any inflammatory condition in the vicinity of the pleura may give rise to an empyema, namely:

(*a*) Chest wall (wounds, osteomyelitis of rib).
(*b*) Lung (pneumonia, abscess, bronchiectasis, tuberculosis, new growth).
(*c*) Post-operatively (thoracotomy).
(*d*) From the œsophagus (perforations, carcinoma).
(*e*) From below the diaphragm (subphrenic abscess).

Pathology.—In the common post-pneumonic empyema actual pleural infection is preceded by the development of a serous effusion. The pleura is subsequently invaded by organisms from the lung with associated inflammatory changes and further exudation of fluid. Fibrin is deposited on the surfaces of the pleura, whilst intrapleural clotting is common in certain types which form a protein-rich exudate (pneumococcal empyema). The

natural defences of the body are aimed at encircling the septic area by a barrier of fibrous tissue : this is initially achieved by the fusion of the lung to the chest wall at the periphery of the collection of fluid. Subsequently the fibrin deposits on the pleura are invaded by blood-vessels from the adjacent lung or chest wall with the formation of granulation tissue, and later of fibrous tissue. This process is progressive with an ever-increasing thickness of the wall of the empyema. Left to her own devices, nature will try to obliterate the empyema by converting it into a plaque of fibrous tissue. As the empyema becomes walled off by adhesions, the fluid thickens so that the presence of thick pus is a good indication that the empyema is localised and is unlikely to spread further. A mature empyema consists of visceral and parietal layers of fibrous tissue on the lung- and chest-wall surfaces respectively, with pus and debris between them. There is usually a good plane of cleavage between the wall of the empyema and the visceral pleura which is important in the operation of decortication. Secondary changes in the surrounding structures appear as the fibrous tissue contracts. The ribs are drawn together and lose their mobility. The diaphragm is elevated and fixed, and the mediastinum drawn towards the affected side. The lung is encased in a rigid covering of fibrous tissue and is immobile and functionless. The final picture of a neglected chronic empyema is a rigid contracted chest with relatively functionless lung underneath (frozen chest).

Clinical Features.—Empyemas, for convenience, can be divided into three groups : (1) Acute. (2) Subacute. (3) Chronic.

There are many potential causative organisms but many empyemas are sterile on the first aspiration owing to previous chemotherapy. The most frequently isolated organisms are therefore those resistant to the commoner antibiotics (*E. Coli, Ps. pyocyaneus*, resistant staphylococci).

Acute Empyema.—The acute fulminating toxic empyema is now rare except when it follows perforation of the œsophagus or rupture of a lung abscess. There is profound toxæmia and shock with pleural pain and rapid, shallow respiration. The signs are those of pleural fluid, which should be confirmed by needle exploration. Early thoracotomy is required if the condition follows rupture or perforation of the œsophagus. Other cases should be treated by repeated aspirations with systemic and intrapleural antibiotics in an attempt to control the infection. If these measures fail to control the toxæmia, drainage should be carried out with an intercostal catheter inserted through a cannula and connected to an under-water seal, even though the pus is thin and the empyema not walled off. Subsequent management will be that of the subacute empyema.

Subacute Empyema.—The majority of empyemas present in a less severe form largely owing to the general use and efficiency of the antibiotics administered for the primary condition. As a result, an empyema may develop insidiously and its presence be completely overlooked. It should always be considered in cases of delayed resolution, slow convalescence or persistent fever following a pneumonic illness. Likewise, it should be suspected where the physical or radiological signs of resolution are inconclusive. Clinical signs

are those of fluid with stony dullness, absent breath-sounds, diminished chest movements, and displacement of viscera.

Chronic Empyema.—Many chronic empyemas are the result of mismanagement of the acute or subacute stages, some are due to failure to diagnose the original condition, and the remainder to some underlying pathology in the lung (bronchiectasis, lung abscess, tumour), pleura (foreign bodies, actinomycosis), or chest wall (rib sequestrum). Toxic absorption from the empyema is slight but symptoms of vague ill-health, febrile bouts, anæmia etc., may occur. A chronic empyema may present in one of several ways, namely :

(1) A closed collection of pus completely walled off from its surroundings (localised empyema).

(2) An empyema which is discharging either continuously or intermittently into a bronchus (bronchopleural fistula).

(3) An empyema which is discharging either continuously or intermittently through a sinus in the chest wall.

Diagnosis.—An empyema is diagnosed by finding pus with an exploring needle. Failure to find pus may be due to :

(1) Use of too narrow a needle or the employment of an inefficient syringe.

(2) Selection of the wrong site for aspiration.

(3) The presence of clot or fibrin tags which block the aspirating needle.

The commonest cause of failure is selection of the wrong site, but this should be avoided by a careful clinical and radiological examination before-

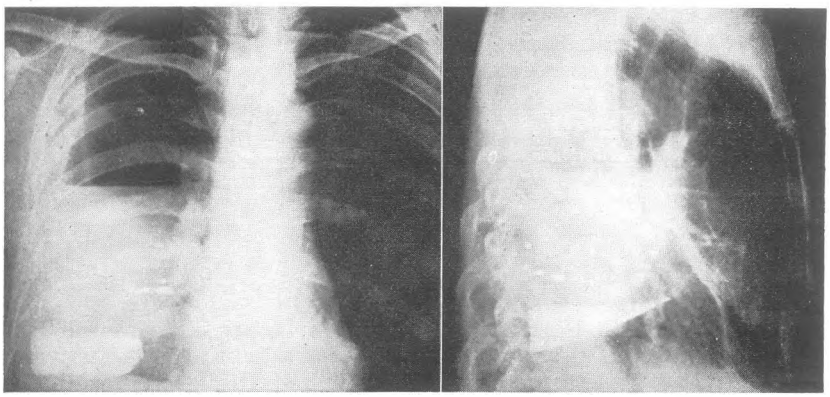


FIG. 760. — Postero-anterior and lateral radiograph of an empyema after injection of iodised oil to delineate the lower limits of the space prior to drainage.

hand. If pus is found, it is an advantage to keep two specimens—one for bacteriological studies, and the other for comparison with subsequent specimens.

Iodised oil can be injected into the empyema so that in subsequent radiographs the lowest limits of the space will be outlined (fig. 760).

Having confirmed the diagnosis, it is important to determine the under-

lying cause; if the empyema is secondary to a carcinoma, lung abscess or a subphrenic abscess, etc., the primary lesion requires appropriate treatment.

Management.—The principles of management of an empyema are basically the same for all varieties though the emphasis tends to vary in the different types. Following diagnosis and the determination of the cause, management resolves itself into controlling infection and eliminating the dead space. Every case requires vigorous breathing exercises if residual deformity and limitation of movement are to be minimised or possibly avoided.

Control of Infection.—Initially all empyemas should be treated by aspirations repeated on alternate days, with the removal of as much pus as possible and the introduction of antibiotics. Sterilisation is usual within seven to ten days. Only occasionally do aspirations fail to control infection, in which case drainage should be carried out.

Elimination of Dead Space.—(*a*) *Aspirations.*—Repeated aspirations without replacement of air will often result in full expansion of the lung and cure of the empyema. This is often the case with children and is more likely after the use of streptokinase. Aspirations should be continued until fluid is no longer obtained and the X-rays show no residual opacity.

(*b*) *Drainage.*—Simple tube drainage after rib resection is indicated when aspirations fail to produce expansion of the lung.

The operation is carried out under local anæsthesia. The site selected for drainage lies immediately above the lowest limit of the empyema posteriorly. The lower posterior site is preferred because this is the most dependent part of the empyema with the patient sitting up in bed. A vertical incision is made over the selected rib and the muscles divided in the line of incision. The periosteum of a 7·5cm. segment of rib is elevated and the segment removed. The intercostal bundle should be securely ligated. The empyema is entered through the posterior periosteal bed and the hole enlarged to permit complete evacuation of the empyema and a thorough inspection and biopsy of the pleura. Drainage should at first be closed, using an under-water seal, but when the discharge is reduced to 60 ml. daily open drainage should be instituted. Drainage must be maintained until the empyema cavity is completely obliterated, which may take as long as six to eight weeks. Premature removal of the tube is a frequent cause of chronicity. Control is effected by carrying out serial pleurograms with radio-opaque oil injected into the empyema cavity at intervals of three weeks. The tube should project 2·5 to 5 cm. into the empyema cavity and will require little alteration until the final stages, when it should be adjusted to keep it 4 cm. shorter than the empyema track.

(*c*) *Decortication* (Excision of Empyema).—This operation, as an alternative to drainage, has become popular since the last war, when experience showed that satisfactory results could be obtained. The operation aims at a complete removal of the empyema with its fibrous-tissue walls, leaving the lung and chest wall free to expand. It gives excellent functional results with minimal pleural thickening. Convalescence is rapid without the necessity for repeated dressings, and return to work is possible within a few weeks. The operation is, however, a major one requiring skilled anæsthesia and blood transfusion. It is indicated particularly for large and chronic empyemas, and for those secondary to bronchiectasis, lung abscess, or carcinoma where the underlying lung condition can be dealt with at the

same time. It is contraindicated in the frail and elderly, or where complete resolution of a pneumonic process has not yet occurred.

(*d*) *Operations on the chest wall*, such as a thoracoplasty, the deroofing procedure of Schede, Roberts' flap operation, and the use of muscle grafts have been almost entirely replaced by the operation of decortication.

Tuberculous Empyema.—May be either a simple tuberculous infection or one complicated by secondary infection or a bronchopleural fistula. The majority are complications of an artificial pneumothorax or surgical treatment. They are much less common than they were, partly due to the widespread use of antituberculous drugs, but also to the decreased popularity of artificial pneumothorax. *Treatment* is basically the same as for any empyema, namely control of infection and obliteration of the pleural space, but is complicated by the presence or not of active tuberculosis of the lung or a bronchopleural fistula. Open drainage should normally be avoided as it will inevitably result in secondary infection of the space. Control of infection is often possible with regular aspiration and the use of systemic and intrapleural streptomycin, P.A.S., or I.N.A.H. The method adopted to obliterate the empyema depends on the condition of the underlying lung. If active pulmonary disease is present, either a thoracoplasty or a pleuropneumonectomy will be required. If the lung disease is controlled and inactive, obliteration may be possible by repeated aspirations over a prolonged period, or alternatively, by excision of the empyema. The operations of decortication and pleuropneumonectomy have dramatically altered the outlook in these cases.

Interlobar Empyema.—An interlobar empyema is a rarity, but one which is wrongly diagnosed with remarkable frequency. It can only occur when the interlobar space is completely shut off from the general pleural cavity. Occasionally a collection of pus remains localised in the fissure in cases of generalised pleural infection when the main collection has been adequately dealt with by aspiration. The diagnosis is largely a radiological one. The collection of fluid is oval or fusiform in shape with the long axis lying in the plane of one of the fissures. The appearances are easily confused with those of segmental atelectasis ; if suspected, pus should be sought with an aspirating needle. Treatment consists of repeated aspirations with chemotherapy, or drainage if aspirations fail.

THE TRACHEA

Tracheal Stenosis.—Tracheal narrowing can be caused by extraluminal compression (carcinoma of thyroid, aortic aneurysms, aberrant aortic arch, enlarged lymph nodes, etc.) or by fibrous strictures of the tracheal wall (tuberculous, post-tracheostomy) or by foreign bodies or intratracheal tumours (squamous cell carcinoma and cylindroma are the commonest tumours found).

The commonest type, at the present time, is stenosis following tracheostomy (p. 703). The stenosis most commonly occurs at the site of the inflatable cuff of a tracheostomy tube. It results from an ischæmic necrosis from an over-inflated cuff. It can be prevented by meticulous attention to tracheostomy drill and frequent deflation of the tracheostomy cuff. Less frequently, obstruction can occur at the level of the stoma and is usually the result of excessive resection of tracheal cartilage.

Resection of Trachea.—Two to three centimetres of the trachea or 5 cartilagenous rings can be resected and an end to end anastomosis carried out. The trachea is fully mobilised from the larynx to the carina to obtain relaxation. Lesions in the upper half of the trachea can be approached by a cervical collar incision but lower strictures may require a sternal splitting incision. The stricture or neoplasm is excised and an end to end anastomosis carried out with interrupted everting mattress sutures. The

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patient should be nursed during the first week with the head fully flexed to avoid tension on the anastomosis. Replacement of a whole segment of the trachea by artificial prosthesis has not been entirely satisfactory. Best results have followed the use of pericardium reinforced with tantalum gauze or marlex mesh.

DISEASES OF THE BRONCHI AND LUNG

Intrabronchial Foreign Bodies

Inhalation of foreign bodies is not a rare occurrence, particularly in children. In many instances the individual assumes that the foreign body has been expelled as the initial choking may soon pass. The commonest foreign bodies are teeth, mutton and rabbit bones, peanuts, pins, screws and nuts. The changes produced depend upon the size and nature of the foreign body. Small, smooth metallic foreign bodies produce little reaction, whilst larger foreign bodies may lead to partial or complete obstruction of one of the bronchi more commonly on the right side because of its more vertical position. Organic foreign bodies, particularly peanuts, produce marked inflammatory changes in the bronchial wall.

The clinical features may be :

(1) Wheezing, irritating cough and signs of unilateral obstructive emphysema (figs. 761 and 762).

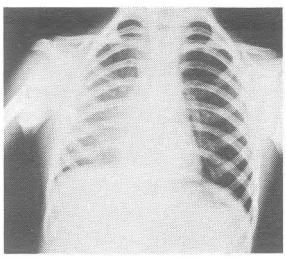


FIG. 761. — Obstructive emphysema of the left lung due to an inhaled peanut.

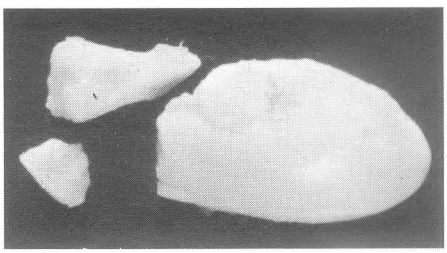


FIG. 762.—Peanut removed bronchoscopically from case illustrated in fig. 761.

(2) Symptoms due to atelectasis or pulmonary suppuration ; cough, sputum, fever, etc.

(3) The patient may be symptomless.

Radiography is an essential investigation in every suspected case. If the foreign body is radio-opaque, it should be visible either in the postero-anterior or lateral film. In many instances the foreign body is either not opaque or is obscured by the heart shadow or secondary inflammatory changes.

Treatment.—Bronchoscopy must be performed in all suspected cases. Removal is easier before secondary swelling and inflammation of the bronchial wall have occurred. Many can be removed without special equipment. Prolonged attempts at removal should not be made owing to the risks of œdema of the glottis or of damaging the bronchial tree further. Operative removal is preferable in such cases. Antibiotics should be given beforehand to minimise the inflammatory reaction. Removal is often possible by

bronchotomy, but if suppurative changes have already occurred in the lung resection will be necessary.

Bronchiectasis

The term bronchiectasis strictly describes any abnormally dilated bronchus. Bronchial dilatation may follow many inflammatory conditions of the lung, especially those associated with obstruction of the bronchus. These changes, which are often patchy and affect only small peripheral bronchi may follow pulmonary tuberculosis, a lung abscess or pulmonary suppuration. Only rarely do they give rise to symptoms. Clinical 'bronchiectasis', however, is usually applied to the variety where involvement of the bronchi is more extensive and usually follows an anatomical pattern, e.g. a segment, a lobe or a lung. It is infection in these abnormally dilated bronchi which produces cough, sputum and hæmoptyses; these together form the pattern of the clinical condition of bronchiectasis.

Ætiology.—Many cases are considered to be congenital in origin, though there is no good evidence to support such a thesis. The majority are the result of compression of the small soft bronchi in children by enlargement of the hilar lymph nodes associated with whooping-cough, measles or primary tuberculosis; the obstruction is often made complete by viscid intrabronchial secretions, Bronchial obstruction produces atelectasis. (The majority of cases of pneumonia complicating whooping-cough are really atelectases.) The subsequent course depends on whether or not secondary suppurative changes occur in the atelectatic lobe. If no infection occurs and the obstruction is relieved, the lobe may expand and return to normal. If suppuration occurs, the bronchial walls are severely damaged and distended by purulent secretions. They lose their elasticity and mobility, and when obstruction is eventually relieved they remain as inert, dilated tubes.

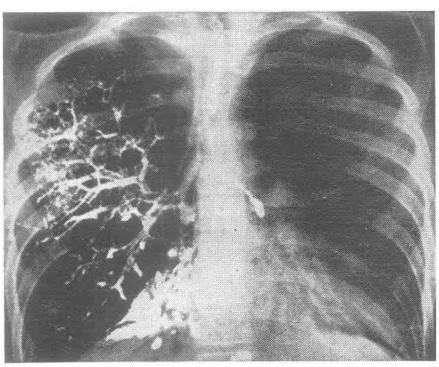


FIG. 763.—Bronchogram showing bronchiectasis and atelectasis of right middle and lower lobes.

Pathology.—The bronchi appear thickened and irregular, whilst histologically there is marked destruction of all the normal elements and extensive infiltration of the remnants with inflammatory cells. The columnar epithelium is replaced by granulation tissue or by squamous or cuboidal epithelium. The lung supplied by the affected bronchi may be fibrotic and airless with loss of the alveolar pattern and infiltrated with inflammatory cells (bronchiectasis with an atelectatic lobe) (fig. 763). In other cases the abnormal bronchi are surrounded by aerated lung tissue but the latter is usually emphysematous and relatively functionless.

Clinical Features.—The majority of cases date their symptoms from an attack of whooping-cough, measles or pneumonia in childhood. The symptoms may persist from that time or the attack may be followed by a latent

period of some duration. They may subsequently be intermittent and are often regarded as attacks of bronchitis. Frequently, especially in severe cases, they are persistent throughout the year, but with exacerbations from time to time. Characteristically, the patients have a loose cough productive of purulent sputum; the latter may be offensive and is often blood-stained, whilst frank hæmoptyses are not infrequent. Acute exacerbations with fever and increase of cough and sputum are frequent during the winter months. In children, both physical and mental retardation may occur. Finger clubbing is frequent and persistent râles and rhonchi are audible over the affected areas. Dullness and bronchial breathing with mediastinal shift occurs with an atelectatic lobe.

Diagnosis.—The diagnosis is usually possible on clinical grounds from a prolonged history of cough and sputum associated with finger clubbing and physical signs over a localised area of the lung. Bronchography is essential, not only to demonstrate the presence of bronchiectasis but also to determine its extent (fig. 763). Bronchoscopy is advisable in order to exclude the presence of a foreign body or tumour.

Treatment.—(*a*) *Prophylactic Treatment.*—Much can be done to prevent the development of bronchiectasis by treating the respiratory complication of measles and whooping cough with antibiotics and breathing exercises, and carrying out bronchoscopy at an early date should atelectasis occur.

(*b*) *Conservative Treatment.*—Considerable reduction of infection can be achieved by postural drainage, physiotherapy and chemotherapy. Such medical treatment, however, cannot cure the condition as the essential abnormality still remains. Relapse is frequent even though treatment is maintained for long periods. Postural drainage is carried out in a position designed to place the affected lobe above the draining bronchi so that pus will gravitate from the lobe into the bronchus from whence it can be expectorated. Periods of half to one hour two or three times daily are advisable. Chemotherapy is reserved for acute attacks or as a preparation for surgery.

(*c*) *Surgical Treatment.*—Extirpation of the diseased area is the only available method of curing the condition, but is not applicable to every case. It is essential in all cases to carry out complete bronchograms on both sides. Resection is only practicable if less than half of the total lung tissue is involved. Ideally, all abnormal areas should be removed (radical treatment) but in certain circumstances it is justifiable to remove a grossly diseased lobe and leave behind less severely damaged tissue (palliative resection). Children will tolerate the removal of more lung tissue than adults, and bilateral resections are rarely advisable after the age of thirty-five years. Removal should be as conservative as possible, particularly in the more extensive cases. Prior to operation, the patient should have intensive treatment with postural drainage and chemotherapy in order to reduce sputum to a minimum, whilst after operation, rapid and complete expansion of the remaining lung is encouraged by breathing exercises, posture and suction drainage to reduce the risks of empyema and bronchopleural fistulæ. The results of surgical treatment are best in cases with limited bronchiectasis in an ate-

lectatic lobe. Disappointing results are encountered in patients with generalised bronchitis or asthma in addition to bronchiectasis.

Lobectomy.—The operation is carried out through a postero-lateral thoracotomy. The passage of pus from the bronchiectatic lobe into normal lung is prevented by intrabronchial balloons or by posture (face down position). The hilum is approached either from its posterior or interlobar aspect. The relevant bronchus, artery and vein are isolated by blunt dissection using long curved artery forceps. The bronchus is divided between clamps and the vessels are securely ligated. The bronchus is closed by interrupted sutures of silk, thread, nylon or steel wire. The lobe is then freed from its attachments to the parieties and mediastinum. Apical and basal pleural drainage tubes are inserted for the removal of air and blood respectively and to ensure the rapid and full expansion of the remaining lung.

TUMOURS OF THE BRONCHI AND LUNG

With one or two rare exceptions, all pulmonary tumours arise from some part of the bronchial tree.[1]

Classification

Benign.—Carcinoid adenoma, Cylindroma (adenoid cystic carcinoma)
Hamartoma (fibroma, chondroma, lipoma, angioma)

Malignant

Primary.—Carcinoma (common).[2]
Alveolar-cell carcinoma (rare).
Sarcoma (rare).

Secondary.—Sarcoma.
Teratoma of testis.
Carcinoma (bowel, breast, thyroid, kidney).

Symptomatology.—The symptoms produced by a tumour may be due to:

(1) The presence of an irritating lesion in the bronchial tree producing cough, sputum, wheezing and hæmoptysis.

(2) The occurrence of partial or complete bronchial obstruction giving rise to obstructive emphysema or atelectasis respectively.

(3) Secondary inflammatory changes in the lung producing consolidation, suppuration or abscess formation.

(4) Symptoms due specifically to the presence of a primary tumour or of its secondary deposits—malaise, anorexia and loss of weight.

(5) Pressure on the œsophagus (dysphagia), trachea (stridor), recurrent laryngeal nerve (hoarseness) or superior vena cava.

(6) Systemic disturbances due possibly to active secretion by the tumour cells—(carcinoid syndrome,[3] Cushing's syndrome, osteo-arthropathy, neuropathy).

Benign Tumours

Representing about 2 per cent. of all pulmonary tumours, they are slightly commoner in women and more frequent in the younger individual.

Pathology.—The tumours fall into two main anatomical groups, those arising from the larger bronchi and those situated in the periphery of the lung. The former are usually visible through the bronchoscope. The intra-bronchial portion of the tumour, however, may only represent a portion of the whole as there is often a large

[1] Mesothelioma is another type of tumour but it originates from the pleural membrane. It is believed to occur more commonly in those who work with blue asbestos fibre.

[2] Kenneth Robinson, former Minister of Health, stated that over the period 1950–1965 deaths from lung cancer had increased by 73 per cent.

[3] Carcinoid tumours of bronchus are second in frequency only to those of the appendix; only a small minority produce systemic disturbances.

Harvey Cushing, 1869–1939. Professor of Surgery, Harvard University, Boston, U.S.A. (1912–1932) (See footnote p. 75.)

extra-bronchial portion (iceberg tumour). Peripheral tumours are not visible through a bronchoscope. Three main varieties are found; carcinoid adenoma, the commonest and most frequently found in the lobar or segmental bronchi; the cylindromatous adenoma (adenoid cystic carcinoma) is the least common and is largely confined to the trachea and main bronchi where it is slowly invasive. This latter tumour is similar, in many respect, to salivary gland tumours (p. 593). The hamartoma most frequently occurs in the lung but is occasionally found in a large bronchus. Histologically the cells of the carcinoid tumour are regular, well formed and consistent in appearance and tend to be arranged in solid acini. Mitoses are infrequent. In the cylindroma the cells are similar but arranged in columns or cylinders. The hamartoma is a composite tumour composed of two or more tissue elements. They represent an abnormal mixing or development of the normal components of the organ in which they occur. Thus, in the lung, cartilage, fat, glandular or vascular tissue and respiratory epithelium may be found. The hamartomas are usually described according to the preponderant tissue, e.g. chondromatous, hæmangiomatous hamartoma, etc. (p. 58).

Clinical Features.—(1) Many are symptomless and found on routine radiography; this particularly applies to the peripheral types.

(2) Recurrent hæmoptysis. The carcinoid adenoma is one of the causes of repeated large hæmoptyses occuring over a period of years. There is frequently complete freedom from symptoms between attacks, though occasionally wheeziness and an irritating cough are present.

(3) Symptoms due to bronchial obstruction and lung infection. Simple uninfected atelectasis or pulmonary suppuration with abscess formation and bronchiectasis both occur commonly. In some cases of a lung abscess or an empyema, the underlying tumour is completely unsuspected until bronchoscopy is performed.

Diagnosis.—The diagnosis is often suggested by the history, but can only be confirmed by bronchoscopy. The adenoma appears as a round, smooth or slightly lobulated raspberry-like tumour which is neither ulcerated nor necrotic. Tumours containing cartilage appear pale and hard and biopsy may be difficult. Bronchography is necessary to determine the presence and extent of any secondary lung changes which will influence subsequent treatment.

Treatment.—Surgical resection is the treatment of choice. This can be planned conservatively as the risk of recurrence is negligible. Treatment by radon implantation, curettage or irradiation is ineffective and cannot be recommended. One or other of the following methods may be applicable to the individual case:

(1) *Bronchoscopic Removal.*—This is only practicable for the rare, strictly intra-bronchial and pedunculated tumours. Bronchoscopy is advisable at regular intervals after removal in order to detect a recurrence.

(2) *Bronchotomy.*—Local removal of the tumour with part of the bronchial wall is practicable for tumours of localised extent without extra-bronchial extension and without secondary lung damage.

(3) *Lung Resection.*—A conservative resection, either segmental, lobar, or total, is required in those cases associated with permanent lung damage. The extent of the resection is determined by the bronchographic findings.

Carcinoma of the Bronchus

Ætiology.—Carcinoma of the bronchus has shown a fifteen-fold increase during the last thirty years. It is ten times more common in men than women, and most frequently occurs during the fifth and sixth decades, but is not unknown in children and young adults. Statistical surveys show that heavy cigarette smoking over many years predisposes to the development of lung cancer. The exposure to other irritants, such as arsenic and radio-active substances (Schneeberg[1] cancer), exhaust fumes, sulphurous smoke and fog, and tarry particles from the roads may possibly be implicated. Workers in the chromate industry have a high incidence of lung cancer.

[1] The name of a district in Germany where these substances are mined.

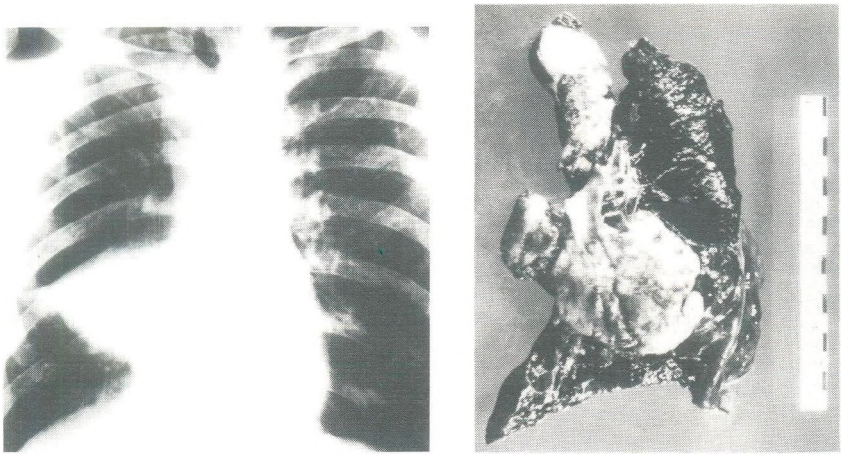
FIG. 764.—Carcinoma of the left upper lobe bronchus. The upper lobe is atelectatic and its bronchi are dilated and filled with mucopus. The lower lobe is normal.

Pathology.—Three main macroscopic types are described :

(1) *Main Bronchus Tumours.*—These arise in the main bronchus or one of its primary or secondary divisions. They produce bronchial irritation and ulceration at an early stage and frequently give rise to bronchial obstruction. They are usually visible through the bronchoscope (figs. 764 and 765).

(2) *Peripheral Tumours.*—These arise from the smaller bronchi and may only be visible with the fibreoptic bronchoscope. They rarely produce secondary lung changes. Some are discovered at routine radiography (figs. 766 and 767).

(3) *Pancoast ' Tumours '.*—These are essentially peripheral lung carcinomas arising at the apex of the lung. The tumour extends early along pre-existing apical adhesions to invade the brachial plexus, sympathetic chain, upper ribs and adjacent vertebræ producing the Pancoast syndrome (lower brachial plexus lesion, Horner's syndrome, an apical shadow, and rib erosion). The lesion is particularly distressing owing to the early production of intractable pain which is difficult to relieve, combined with a relatively slow growth.



(*a*) (*b*)

FIG. 765.—Adenocarcinoma right middle lobe. (*a*) Radiograph showing consolidation of right middle lobe and enlargement of mediastinal shadow. (*b*) Pneumonectomy specimen showing massive tumour in middle lobe and enlarged paratracheal nodes excised with the lung.

Histology.—Three main histological types are recognised.[1]

(1) Squamous cell (epidermoid) carcinoma 50%.

(2) Anaplastic (*a*) Small cell 15% (majority are oat cell).
(*b*) Large cell (5%).

(3) Adenocarcinoma 20%.

The anaplastic carcinomas are poorly differentiated tumours often exhibiting rapid growth with early metastases. They are more common in young individuals.

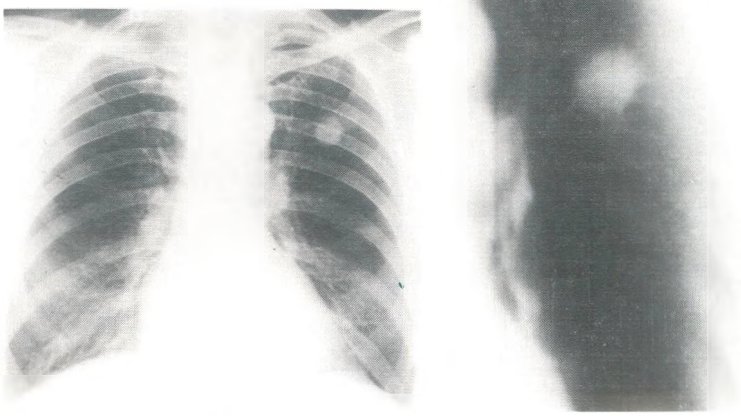
[1] Based on WHO classification.

Henry Khumrath Pancoast, 1875–1939. Professor of Rontgenology, University of Pennsylvania, Philadelphia, U.S.A.
Johann Horner, 1831–1883. Professor of Ophthalmology, Zürich, described his syndrome in 1869.

Metastases.—(1) Direct extension into the mediastinum, the pleura, the chest wall or the pericardium

(2) The lymphatic system to the hilar lymph nodes and thence to the subcarinal nodes or the paratracheal chain. The upper nodes of the chain are continuous with the inferior deep cervical group and may be detected by digital palpation deep to the insertion of the sternomastoid muscle, or by scalene node biopsy.

FIG. 766.—Peripheral type of lung carcinoma, showing typical lobulated outline and absence of secondary changes in the lung.



(*a*) (*b*)

FIG. 767.—Peripheral lung carcinoma. (*a*) Typical ' coin lesion ' in left upper lobe. (*b*) Tomogram shows spidery extensions into surrounding lung typical of carcinomatous invasion.

(3) The blood-stream to the liver, brain, and bone (ribs, vertebræ, pelvis particularly). Other tissues and organs may be involved.

Clinical Features.—Symptoms produced by a carcinoma of the bronchus are many, but the most important are cough, sputum, hæmoptysis, dyspnœa, pain and wheezing. It is important to recognise the early manifestations as it is at this stage that treatment is likely to be most effective. Any of the above symptoms appearing for the first time in a middle-aged individual and tending to become more severe with time, demand early investigation. Only some 10 per cent. of cases present initially with an hæmoptysis; more frequently, the first symptoms may occur suddenly with an influenzal-like illness which fails to resolve satisfactorily. A number start insidiously with cough and dyspnœa, whilst in the case of the chronic bronchitic there may be an alteration in the typical pattern of his symptoms. Occasionally, first symptoms are due to the presence of secondary deposits (especially in the

brain) or invasion of the mediastinum. A diagnosis of unresolved or virus pneumonia should always be made with reservations, and if recovery is not complete, carcinoma should be suspected. Finger clubbing is a frequent and important sign (fig. 768). A small group present with neurological (carcinomatous neuropathy) or arthritic (osteo-arthropathy) (fig. 769) symptoms without chest symptoms. A further group present with symptoms due to abnormal hormonal secretion, resulting in hypokalæmia (muscle weakness), hyponatræmia (water retention), hypercalcæmia (confusion and drowsiness), gynæcomastia or Cushings' syndrome (p. 653). The prognosis tends to be less good in these cases and often an oat cell growth is present. The presence of an underlying lung carcinoma is revealed by chest X-ray.

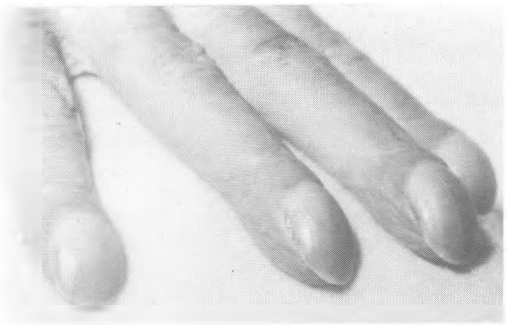


FIG. 768.—Gross finger clubbing in patient with lung cancer.

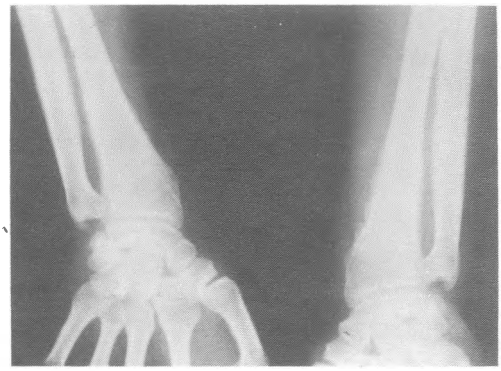


FIG. 769.—Periosteal changes seen in radius and ulna in patient with lung cancer and pulmonary osteo-arthropathy.

Investigations.—The suspicion of a lung cancer demands a thorough investigation. *A chest X-ray* is the most important single investigation and it is usual for some abnormality to be revealed. The appearances vary from that of a hilar or peripheral shadow due to the presence of the tumour to those of obstructive emphysema, atelectasis or consolidation due to secondary lung changes.

Bronchoscopy.—This is not only an essential diagnostic procedure but produces important information regarding operability. The examination will reveal the presence of the tumour in those lesions which are centrally placed and enable a biopsy to be made and the histology determined. Paralysis of a vocal cord, involvement of the trachea, or widening of the main carina, are regarded as evidence of inoperability. It should be emphasised that bronchoscopy may not reveal the presence of some tumours, particularly those arising in the segmental divisions of the upper lobes, or the peripherally placed tumours.

Cytological examination of sputum will reveal malignant cells in about 40 per cent. of cases.

Barium Swallow.—This may provide evidence of extension of the growth

into the mediastinum or the enlargement of mediastinal lymph nodes. If œsophageal distortion is considerable, adequate removal is improbable.

Tomography is useful in demonstrating enlarged mediastinal nodes.

Liver, brain and bone scanning is indicated if involvement of these organs is suspected.

CAT whole body scanning is being increasingly employed for the detection of distant metastases.

Bronchography.—This may provide valuable information in cases of doubtful diagnosis but has little place in the investigations of the proven case.

Scalene Node Biopsy and Mediastinoscopy.—This is a routine practice in some clinics. Nodes from the paratracheal and inferior deep cervical groups may be obtained by these means. If invaded radical surgery is contra-indicated.

Diagnosis.—A positive histological diagnosis is available in about 60 per cent. of cases, whilst malignant cells in the sputum or pleural fluid provide positive evidence in some of the remainder. In about one-third of cases, however, only a presumptive diagnosis can be made.

In the unproven case, a carcinoma is most likely to be confused with chronic suppurative pneumonia, a lung abscess or solid tuberculous lesions, and occasionally with a benign tumour. Careful consideration of the history and response to antibiotics will often provide strong circumstantial evidence against a carcinoma, whilst bronchography and tomography may be helpful in doubtful cases.

Treatment.—Surgical removal of the tumour offers the best prospects for cure, although in selected cases the results of radical radiotherapy are equally good.

The aim of operative treatment must be the adequate removal of the primary tumour and the regional lymph nodes, but with the preservation of as much normal lung tissue as possible.

The extent of the lung resection will thus depend on the site of the tumour. For lesions confined to one lobe *lobectomy* will suffice, but those involving the main bronchus *pneumonectomy* will be required (fig. 770). An intermediate group can be adequately treated by removing the involved lobe and a cylinder of the main bronchus—*sleeve resection*—thus preserving some normal lung tissue. In all cases the paratracheal, subcarinal and para-aortic lymph nodes are removed. Limited resection of the chest wall, pericardium or diaphragm can be carried out should these structures be invaded.

Radical Pneumonectomy

'One lung anæsthesia' is advisable. The feasibility of lung resection should be determined with as little lung disturbance as possible. At an early stage the pulmonary veins from the affected lobe should be tied to reduce the risks of tumour embolism. The remaining vessels are then isolated and divided after securing with double ligatures or a transfixed ligature. The bronchus is divided close to its origin after applying special bronchus clamps. The bronchus is closed with sutures of wire, nylon, or thread, using inter-

rupted figure of eight stitches and occluding a rim of bronchus 3 to 4 mm. wide (figs. 770 and 771).

The paratracheal and subcarinal and para-aortic lymph nodes should be removed with surrounding tissues.

Only 20 per cent. of lung cancers are considered suitable for exploration and of these one in five is found to be too advanced for resection on explora-

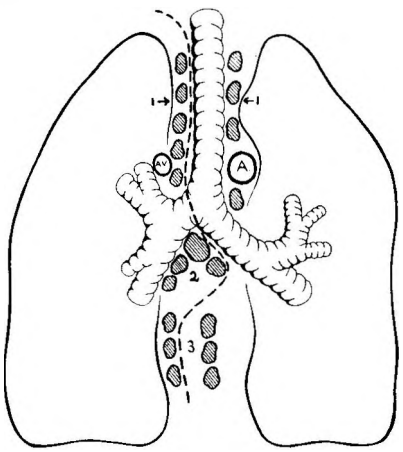


FIG. 770.—Right radical pneumonectomy. The dotted lines indicate the plane of dissection. A.V. = azygos vein. A. = aorta. 1 = paratracheal nodes. 2 = subcarinal nodes. 3 = para-aortic nodes.

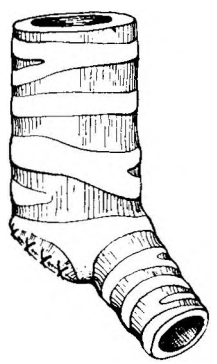


FIG. 771. — Sutured bronchus after right pneumonectomy. Note oblique division, absence of stump, and figure-eight stitches.

tion. The mortality of radical resection is 10 per cent. Five-year survival (25–40%) is largely dependent on histological type and staging at the time of surgery as follows:

Stage I.—Growth confined to the lung.

Stage II.—Involvement of broncho-pulmonary lymph nodes at hilum.

Stage III.—Involvement of tracheo-bronchial (mediastinal) lymph nodes.

Stage IV.—Spread of growth outside the chest.

The following are contraindications to radical resection :

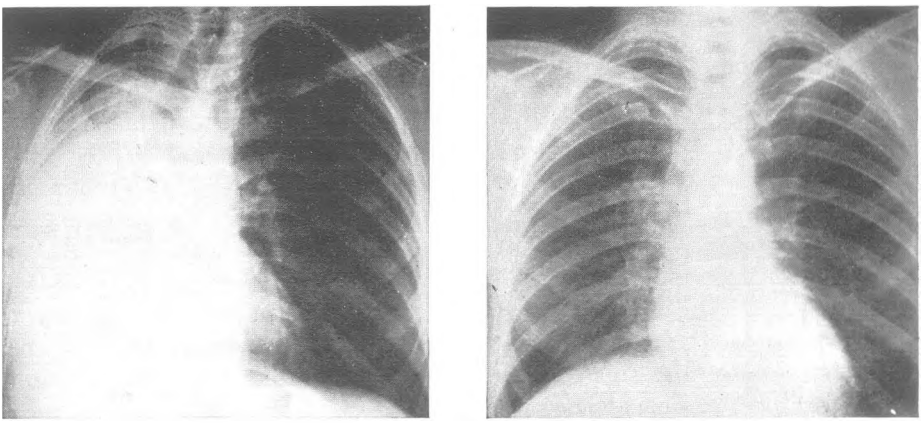
(1) A growth which involves the trachea or the origin of the main bronchus.
(2) Evidence of spread into the mediastinum with involvement of the œsophagus, recurrent laryngeal nerve or vena cava.
(3) Extensive invasion of the chest wall (fixed chest pain).
(4) Evidence of distant metastases.
(5) Patients over the age of seventy, unless remarkably fit for years.
(6) Patients with poor respiratory reserve. (This can usually be assessed on clinical grounds with simple exercise tests).
(7) Patients with serious ischæmic heart disease or generalised arterial disease.
(8) Patients with chronic bronchitis with persistent signs in the normal lung who are likely to be severely crippled by dyspnœa and bronchitis after operation (respiratory cripple).

Palliative Resection.—At thoracotomy it is advisable to remove the lung with the primary tumour whenever possible even though the growth cannot

be completely removed. Patients are better off without the primary tumour and die more peacefully from secondary deposits. Occasionally a deliberate palliative resection is justified in cases with repeated hæmoptyses or with much pulmonary suppuration.

Interstitial Irradiation.—This is carried out by the insertion of radioactive grains of gold ^{198}Au. It is a useful method of palliation in those tumours found to be irremoveable at thoracotomy and after palliative or incomplete resections. Gold grains can also be inserted into intrabronchial tumours through the bronchoscope.

X-ray Therapy.—Radical X-ray therapy, particularly with super-voltage machines, is employed in cases which are considered operable from the point of view of the tumour, but inoperable on other grounds. Survival rates of some years have been recorded but no extensive series is available (figs. 772 and 773). Palliative X-ray therapy is designed to relieve superior



FIGS. 772 and 773.—Main bronchus carcinoma, showing total atelectasis of right lung before irradiation, and aeration of the lung after treatment.

vena caval obstruction, bronchial obstruction and the pain of bone involvement. Considerable relief is obtained by such treatment, but survival is not appreciably lengthened.

Pre-operative X-ray therapy has given disappointing results largely due to an increased incidence of broncho-pleural fistulæ following resection.

Post-operative treatment is used occasionally where small areas of growth have not been removed (e.g. on the chest wall). Only fit patients should be treated if serious debilitating reactions are to be avoided.

Chemotherapy.—The use of cytotoxic drugs alone has been disappointing. Some benefit may follow their administration combined with surgery.

Secondary Lung Tumours.—Secondary deposits in the lungs from carcinomas and sarcomas occur frequently. The deposits are usually multiple but occasionally occur singly. In the absence of other evidence of malignant disease solitary lesions should be excised. A 3-year survival of 30 per cent. is obtained overall but the results with testicular teratoma are excellent with many patients surviving for 10 or 15 years.

Lung Abscess

A lung abscess is a collection of pus in the lung substance. All cases are secondary to some pre-existing condition, such as foreign bodies, tumours, or bronchostenosis, or to the inhalation of infective material. There is no such condition as primary or idiopathic lung abscess, and in all cases the cause should be elucidated.

Classification.—*A. Due to specific pneumonias.*—(1) Staphylococcal, (2) Actinomycotic, (3) Friedländer's pneumonia, (4) Amœbic.
B. Due to bronchial occlusion.—(1) Carcinoma, (2) Benign tumours, (3) Intrabronchial foreign bodies, (4) Sputum plugs (post-operative atelectasis).
C. Due to vascular embolism.—(1) Infarct, (2) Pyæmic. (Common amongst drug addicts.)
D. Traumatic.—(1) Infected hæmatoma of lung, (2) Foreign body in lung.
E. Aspiration (inhalation) pneumonia.—Non-specific suppurative pneumonia.

The commonest group is that due to an aspiration pneumonia. In these cases infected material, usually from the upper respiratory passages, is inhaled into the lung, where various grades of infection ensue from a relatively benign bronchopneumonia to more severe states with necrosis, suppuration and abscess formation.

The most important factors in the production of a lung abscess are:
(1) A source of infected material (dental sepsis, sinus infection, vomit, operations on the upper respiratory tract).
(2) A disturbance of the natural defence mechanisms of the body by depression of the cough reflex (with drugs, in coma, or after anæsthesia or alcohol).
(3) A disturbance of the normal mucous production and ciliary action of the bronchial mucosa (Negus).

Pathology.—Infected material is inhaled into the bronchial tree and obstructs one of the smaller bronchi. The resulting atelectatic segment is invaded by pathogenic organisms producing pneumonic consolidation. The commonest organisms are *Hæmophilus influenzæ* and Pneumococci but invasion by mouth organisms (anaerobic streptococci and spirochætes), coliform organisms and cross infection with drug-resistant staphylococci sometimes occur and may be detected by microscopy and culture of the sputum. Suppuration and necrosis develop in varying degrees within the involved segment, first with the production of a suppurative pneumonitis which later matures into an abscess. As pus accumulates, tension rises and eventually the abscess ruptures into the bronchus. This may be followed by complete expectoration of all sloughs and pus, and the inflammation may then subside. More commonly, however, a state of chronic infection with persistence of the abscess occurs (fig. 774). Progressive involvement of adjacent lung tissue or spread to other parts of the lung is possible.

Clinical Features.—The onset is often acute with influenza-like symptoms and variable toxæmia, but usually without any localising symptoms. After some days of ill-defined illness, pleural pain and a dry irritating cough may appear; the latter becomes worse and the patient may notice an offensive odour in his breath. About the tenth day, rupture of the abscess results in the sudden expectoration of considerable quantities of offensive

Carl Friedländer, 1847–1887. Pathologist, Friedrichshain Hospital, Berlin. Described this bacillus in 1882.
Sir Victor Ewings Negus, 1887–1974. Surgeon, Ear, Nose and Throat Department, King's College Hospital, London.

blood-stained pus. With successful treatment, resolution occurs slowly with reduction of toxicity and diminution of cough and sputum, but healing takes several weeks and can only be regarded as complete when cough and sputum have disappeared and radiographs are clear. Chronicity is suggested by the persistence of cough with the expectoration of purulent sputum and the presence of a cavity on X-ray (fig. 775).

FIG. 774.—Chronic lung abscess showing irregular cavity filled with inspissated pus and sloughs and surrounded by an area of chronic pneumonia.

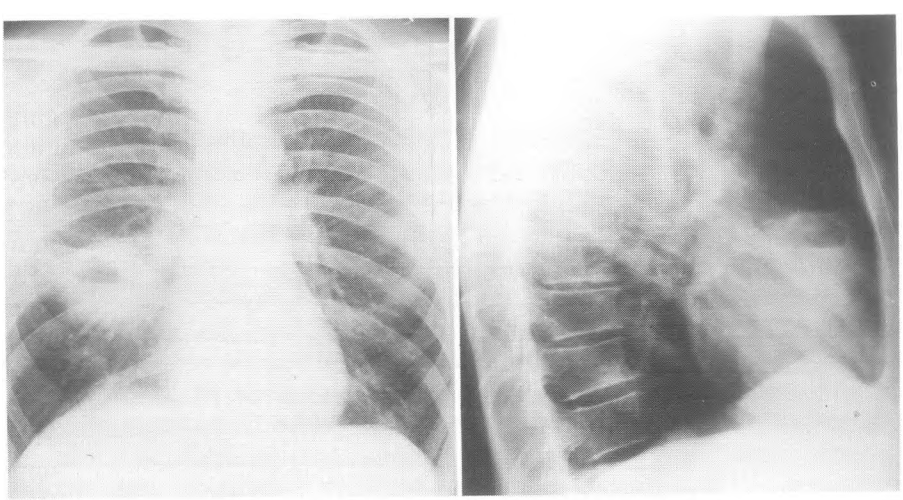
Complications.—(1) Spread of the disease to other parts of the lung by direct extension or by bronchial embolism: this is suggested by an exacerbation of symptoms and fresh radiographic changes.

(2) *Empyema.*—This may occur spontaneously or following surgical drainage but is less common following the introduction of antibiotics. Persistent fever, pleural pain, and signs of a pleural effusion call for exploration of the pleura with a needle.

(3) *Cerebral Abscess.*—Pyæmic emboli reach the brain through the paravertebral system of veins (Collis). The resulting abscesses are usually multiple but occasionally single. The onset is usually accompanied by headaches and fever. A fit is occasionally the first symptom. The incidence of cerebral abscess has fallen markedly since the introduction of antibiotics.

(4) *Secondary Hæmorrhage.*—Hæmorrhage may occur from a drained or an undrained abscess. It is often no more than blood-staining of sputum or discharge, but occasionally severe or fatal hæmorrhage occurs.

Diagnosis.—The diagnosis is suggested by a history of a sudden, acute influenza-like illness, followed later by coughing with purulent sputum



(*a*) (*b*)

FIG. 775 (*a*) and (*b*)—Abscess in anterior segment of right upper lobe showing cavity with fluid level and surrounding zone of consolidation.

John Leigh Collis, Contemporary. Professor of Thoracic Surgery, United Birmingham Hospitals, England.

and the demonstration of an area of lung consolidation, with or without cavitation. All cases must be investigated to determine the cause by:

(1) Bronchoscopy to exclude foreign bodies or neoplasm.
(2) Complete bacteriological examination of the sputum.
(3) Examination of the teeth and upper respiratory tract.

Treatment.—Eighty-five per cent. of lung abscesses resolve with conservative measures, and surgery is only required for the minority which do not respond. Medical treatment consists of:

(1) *Chemotherapy.*—Penicillin is the antibiotic of choice as it can be given in very large doses (2 to 8 million units daily) for long periods (four to six weeks) with little risk of side-effects. Other antibiotics should be used if the organisms are resistant to penicillin. The response to antibiotics is often slow and treatment must be continued for some weeks until there is no further clinical or radiological improvement.

(2) *Postural Drainage.*—This should be carried out for periods of up to one hour three or four times a day with the patient placed in such a position that the abscess lies above the appropriate draining bronchus.

(3) At the same time, percussion over the site of the abscess should be carried out to encourage drainage.

Conservative treatment is continued whilst there is clinical and radiological evidence of improvement, or until symptoms have disappeared and the X-ray is clear.

Surgical treatment is indicated in those cases where resolution does not occur with the above regime. In the majority of cases a formal resection (lobectomy) is possible. External drainage of the abscess—once the standard operation—is now only occasionally required in patients who show no response to chemotherapy and who remain too ill for resection.

Lung Cysts

These may be divided into four groups:

(1) *Epithelial Cysts.*—These are developmental in origin and may be solitary and large or multiple and small. They are lined by respiratory epithelium and may have traces of cartilage, muscle or glands in their wall.

(2) *Emphysematous Cysts.*—These include a wide variety of conditions where the normal alveolar framework is destroyed or disrupted, resulting in large air spaces which may become further distended with air.

(3) *Parasitic cysts*, of which the commonest is the hydatid.

(4) *Pseudo-cysts.*—Certain inflammatory conditions of the lung result in cavity formation, and in many instances the cavities may closely resemble epithelial cysts. The cavities, when chronic, are often lined by squamous epithelium. These pseudo-cysts may occur in association with staphylococcal pneumonia, pulmonary tuberculosis, or following a lung abscess. Their true nature is often suggested by their history, course, and radiological features, but the distinction is often difficult.

Epithelial Cysts.—These are often associated with other congenital abnormalities (cervical rib, pulmonary stenosis, patent ductus) (fig. 776). They appear as spherical shadows in the X-ray with a thin, sharply defined

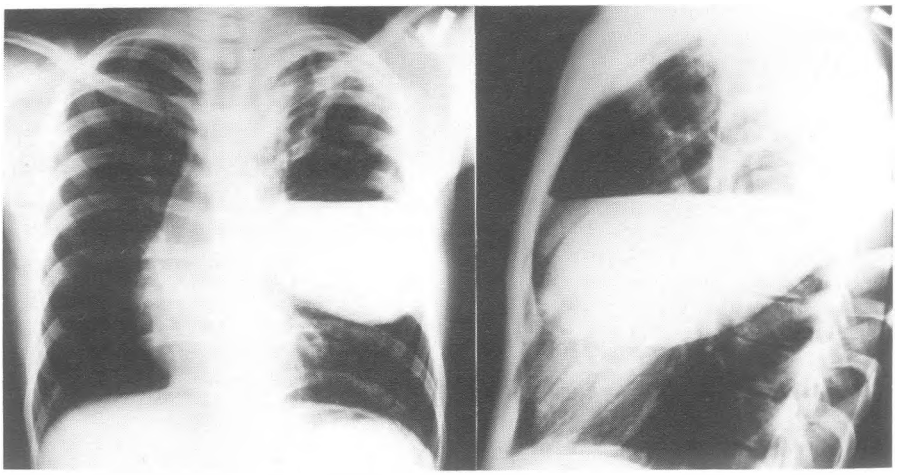


FIG. 776.—P.A. and lateral X-ray of large air and fluid containing congenital lung cyst occupying the greater part of the upper lobe. Removal by lobectomy.

wall. They may contain air, fluid, or both air and fluid. Symptoms may be produced by:

(1) The size of the cyst which compresses the lung and produces dyspnœa, tightness in the chest, etc. This is particularly so in infants and children where the thorax is small and the cyst often relatively large.

(2) Infection is common with the production of fever, cough and sputum. Radiography will demonstrate a fluid level in the cyst. Infection is usually readily controlled with antibiotics, but recurrence is frequent and removal indicated.

(3) Hæmorrhage from cysts is usually associated with infection.

Spontaneous pneumothorax is uncommon with epithelial cysts (cf. emphysematous cysts.)

Treatment.—Solitary cysts producing symptoms should be excised. Symptomless cysts can be kept under observation. Infection of multiple cysts should be controlled by antibiotics if excision is not practicable.

Emphysematous Cysts.—Although many cases are acquired and due to degenerative changes in the lung, others cannot be explained on this basis. The latter may be due to a congenital deficiency of the elastic tissue of the lung, or to absence of cartilage in the walls of the bronchus. Either of these conditions may lead to abnormal distension of the lung with rupture of the alveolar walls. Progressive coalescence of alveolar spaces combined with distension with air may result in the development of an enormous cyst (figs. 777 and 778). These cysts have no epithelial lining, and when large the remnants of the more resistant bronchi and blood vessels are seen stretching across the

space. The changes may be limited to a small area of the lung, such as a segment or a lobe, or the condition may be generalised and bilateral.

Clinical Features.—The most important symptom is dyspnœa, which is due to a combination of compression of normal lung tissue by the cyst, increase in the dead space, and a poor gaseous exchange. Emphysematous cysts are frequently associated with chronic bronchitis. In such cases persistent coughing tends to distend the cysts still further, whilst the presence of bronchial secretions impairs respiratory function still more. The presence of bronchitis is a serious complication which renders radical treatment more hazardous.

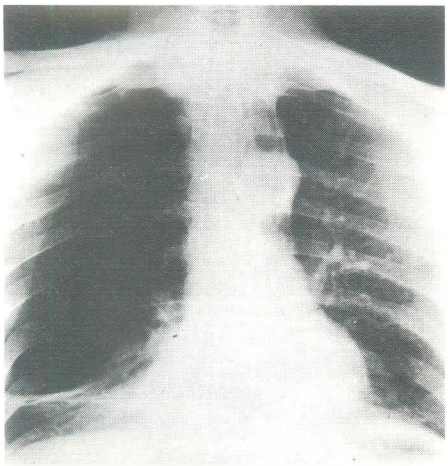


FIG. 777.—Giant emphysematous cyst of right upper lobe with compression of the lung and depression of the diaphragm.

FIG. 778.—A giant emphysematous cyst which presented through the wound on opening the chest.

Spontaenous pneumothorax is a common and serious complication and may be the first presenting feature. Infection and hæmorrhage are unusual, (cf. epithelial cysts).

Treatment.—Emphysematous cysts which are not associated with generalised emphysematous changes should be excised, but if changes are widespread, excision is impracticable. Obliteration by plication with multiple sutures has given good results; it is applicable to multiple cysts and does not remove potentially functioning lung tissue. Removal of the pulmonary autonomic nerves can be combined with the above procedures; its chief value lies in the reduction of bronchial spasm. In cases complicated by a spontaneous pneumothorax, pleurodesis should be carried out to prevent further attacks of this serious complication.

Hydatid disease of the lung (Chap. 45) has been largely eliminated from such countries as Australia and Iceland by control of dogs and the proper slaughtering of sheep. The disease remains endemic in Asia and parts of the Middle East but is now rare elsewhere.

Pulmonary hydatid cysts occur in about 15 per cent. of all cases; the cysts are usually solitary, but multiple or bilateral cysts are not uncommon. The disease is more common in children and young adults.

The cases may present in any of the following ways:

(1) On routine clinical or radiological examination and without symptoms.
(2) Dyspnœa, pain or tightness in chest due to the presence of a large cyst.
(3) Hæmoptysis due to ulceration into the bronchus.
(4) Expectoration of watery fluid and ' grape skins ' due to rupture into the bronchial tree. This event may produce severe distress with suffocating symptoms or even death if the leak is a large one. Anaphylactic shock and urticaria may occur.
(5) Symptoms due to secondary infection of the cyst, viz, cough, purulent sputum and fever.

Radiology is the most helpful investigation:

(1) An uncomplicated cyst appears as an almost spherical, sharply defined dense homogeneous opacity.
(2) Communication with the bronchial tree may result in a crescentic cap of air overlying the cyst (perivesicular pneumo-cyst).
(3) Rupture of the cyst permits air to enter the cyst and the laminated membrane collapses. This floats on any fluid which may be present, producing an irregular projection above the fluid level (' water-lily ' appearance) (fig. 779).
(4) Infection results in disintegration of the laminated membrane with appearances simulating a lung abscess.
(5) Varying degrees of pneumonitis, atelectasis, or pleural effusion may accompany any of the above and modify the radiological appearance.

Diagnosis.—The most important point is to remember the condition. A history of residence in an endemic area may support the diagnosis. The intradermal test of Casoni will give a positive result in 75 per cent. of uncomplicated cysts provided fresh hydatid fluid is used. An eosinophilia of over 4 per cent. is suggestive of the condition.

FIG. 779.—Hydatid cyst. ' Water-lily ' appearance.

Treatment.—Removal of the cyst is indicated in all cases owing to the considerable risk of serious or even fatal complications. The earlier operation of marsupialisation has been almost completely replaced by thoracotomy with enucleation of the cyst or lobectomy. Enucleation is employed for uncomplicated cysts without significant associated lung damage. Extreme care and utmost gentleness is required to remove the laminated membrane intact without spilling the contents with the risk of infecting the pleura or wound. The adventitia is carefully incised and the laminated membrane will usually slowly extrude itself. It should never be grasped with forceps as it is friable and is sure to tear. Extrusion can be expedited by gentle positive pressure by the anæsthetist. After removal, any bleeding-points or open bronchi are secured and the residual space can be obliterated by mattress sutures if desired. The pleura is drained as some leakage of air is inevitable.

Complicated cysts are best treated by a more formal resection (lobectomy), particularly if there is any surrounding lung damage.

Secondary Pleural Hydatid Disease.—This is an occasional but serious complication of rupture of a cyst into the pleura or of ill-executed surgery. Multiple cysts grow widely throughout the pleura and may invade the chest wall, diaphragm, or mediastinum. Radical pleuropneumonectomy offers the only hope of eradicating the disease.

Hepato-Bronchial Fistula (fig. 940) is caused by the rupture of an infected hepatic cyst into the lung. The infected material traverses the subphrenic and pleural spaces (both being obliterated by adhesions) before discharging into the lung. It results in the production of bile stained sputum. Treatment demands a thoracotomy with resection of the affected lobe; the track through the diaphragm must be enlarged to expose the underlying residual cyst which is drained through an independent opening.

Tomaso Casoni, 1880–1933. Physician, Ospedale Coloniale Vittorio Emanuele III, Tripoli, Libya.

Pulmonary Œdema

Blood in the lung capillaries is separated from air in the alveolar spaces merely by the thin capillary wall and the alveolar membrane. Gaseous diffusion occurs readily across this membrane and any disturbance of the physico-chemical balance in the blood may permit the transudation of fluid from the capillaries into the alveolar spaces.

Initially, transudation occurs into the interstitial tissues of the lung, producing dyspnœa and cyanosis; later, when fluid reaches the alveoli, thin frothy blood-stained fluid is expectorated, often in considerable quantities. Coarse râles are audible throughout both lungs and radiographs show consolidated areas spreading from each hilum towards the periphery ('bat's wing' appearance).

Causes.—(1) Obstruction to the flow of blood through the left side of the heart as in left ventricular failure and mitral stenosis.

(2) Increased volume of blood reaching the lungs as may occur after excessive transfusion of blood or saline.

(3) Disturbances of osmotic pressure in the blood.

(4) Traumatic, such as follows the inhalation of certain noxious gases (phosgene, mustard gas), hot air, smoke or steam.

(5) Inflammatory, as in influenzal pneumonia.

(6) Associated with certain brain injuries and lesions of the brain stem.

(7) Fat embolism.

Treatment is urgently required along the following lines:

(1) Correction of the precipitating cause.

(2) Support of the heart and circulation with aminophylline (250 mg. intravenously), digitalis (0·5 mg. digoxin intravenously) pressor agents and oxygen.

(3) Morphine, which should be given intravenously, is particularly effective in left ventricular failure.

(4) Diuretics such as Frusemide (Lasix) 40 mg. intravenously, repeated in 4 hours if necessary.

(5) Positive Pressure Ventilation is the most effective method of controlling pulmonary œdema and should not be delayed when the above measures are not immediately effective. Initially it can be employed with a cuffed plastic endotracheal tube, but tracheostomy will be necessary if ventilation is required for more than a week to ten days.

SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

In a book of this nature it is not possible to give more than an outline of the principles involved in the surgical treatment of pulmonary tuberculosis. Such treatment is usually an incident in the long-term management of the case, and as such can only be satisfactorily determined by consultation and discussion between physician and surgeon.

Principles of Treatment.—(1) *Rest.*—This can be either local or general, and is an essential part of all forms of treatment. General rest is

obtained in bed, whilst local rest is achieved by the various methods of collapse therapy, phrenic paralysis, and pneumoperitoneum.

(2) *Chemotherapy.*—The introduction of various anti-tuberculous drugs since 1950 has revolutionised the treatment of all forms of tuberculosis and has resulted in a world-wide reduction in the prevalence and importance of this disease (p. 37).

Currently, best results are obtained with the following regime: streptomycin 0·75 g, isoniazid 300 mg, and rifampacin 450 mg in daily doses. Ethambutol is substituted for streptomycin in doses of 25 mg per kg for patients over 40 to avoid the risks of VIIIth nerve damage in older patients from streptomycin. Streptomycin (or ethambutol) are discontinued after two months whilst isoniazid and rifampacin are continued for a total of nine months. Subsequent relapses are very rare.

Pyrazinamide, para-amino salicylic acid and thiocetazone are sometimes used in place of rifampacin which is currently more expensive but has the advantage of requiring only a single daily dose.

(3) *Relaxation.*—Tuberculosis heals by the production of fibrous tissue resulting in the contraction of the diseased area and the collapse and obliteration of the destroyed areas and cavities. Complete contraction and healing is often prevented by anatomical structures (bony chest wall), so that artificial methods of relaxing the diseased lung are required in many instances. These are divided into the minor and major methods. The former include artificial pneumothorax, phrenic paralysis, and pneumoperitoneum, whilst the latter include thoracoplasty, extrapleural pneumothorax, and plombage.

(4) *Excision of Diseased Lung.*—Prior to the introduction of streptomycin, excision of tuberculous lungs usually met with disaster. Antibiotics used before, and as a cover for operation, have transformed the situation and resection has become the surgical therapy of choice. Resection has the advantage over collapse therapy of removing the major foci of disease, leaving the body with the less formidable task of dealing with the residual disease.

(5) *Drainage of Pus.*—Apart from the drainage of an empyema, this method has little application now, though in the past tuberculous lung cavities were treated by catheter drainage (Monaldi), or open external drainage (cavernostomy).

Management.—All cases should be treated with bed rest and chemotherapy for several months before any active measures are undertaken. In many instances prolonged treatment will result in complete arrest of the disease; in others a tuberculous cavity, a positive sputum, or bronchial damage requires more active treatment. Surgical measures are almost entirely employed for dealing with residual lesions which persist after adequate conservative treatment. Resection is the treatment of choice in nearly 90 per cent. of all cases requiring surgical aid. Thoracoplasty is employed only when the risks of resection are unduly high or the disease too widespread for safe removal.

Apart from the failures of medical treatment and the persistence of un-

Vincenzo Monaldi, Contemporary. Director, Tuberculosic Clinic, University of Naples, Italy.

stable foci, resection is indicated in patients with destroyed lobes, in tuberculous bronchiectasis and in large solid lesions (tuberculoma). Where there is an empyema as well, surgical treatment will probably be required.

Thoracoplasty.—This has been the standard method of treatment employing major collapse therapy. The operation is now performed infrequently owing to the greater efficiency and safety of lung resection. It is mainly used for cavities in the upper lobe associated with a persistently positive sputum and

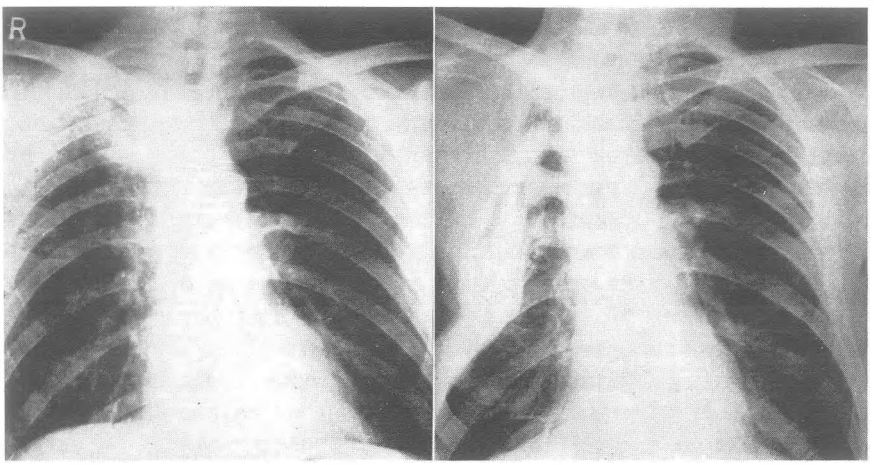


FIG. 780.—Chronic tuberculous cavity at the right apex.

FIG. 781.—Same case as in fig. 780 after thoracoplasty.

resistant organisms (figs. 780 and 781), especially where extensive disease makes resection too hazardous.

The aim of the operation is to relax the diseased area from all sides so that it will retract concentrically towards the hilum. Lateral relaxation is produced by removal of the upper ribs whilst apical relaxation is achieved by separating the lung from the apex and the mediastinum. The original operation was introduced by Sauerbruch and consisted of removal of ribs only (lateral thoracoplasty), but this operation has been replaced by Semb's modification which includes apicolysis in addition to rib removal.

Lung Resection.—Since the introduction of streptomycin, resection of tuberculous lesions has been widely practised and has given good results. The operation has the advantage of removing the greater part of the diseased tissue, in leaving no external deformity, and disturbing respiratory function very little. But it cannot be safely employed in those with extensive disease. Before embarking on resection it is necessary to determine by tomography and bronchography whether the lung and bronchi to be left behind are free from disease. Pre-operative bronchoscopy is advisable, and if active tuberculous bronchitis is discovered, resection should be delayed until it is controlled by further chemotherapy. At operation it is advisable to remove all the obvious tuberculous disease, although small healed nodules may be safely left behind (fig. 782). The remaining lobes must be prevented from

Ernst Ferdinand Sauerbruch, 1875–1951. Successively Professor of Surgery at Zurich, Switzerland, and at Munich and Berlin, Germany.
Carl Boye Semb, 1895–1971. Professor of Surgery, University of Oslo.

over-expanding either by temporary phrenic paralysis or by performing a small apical thoracoplasty. Over-distention of the lobe is likely to cause reactivation of dormant foci. It is unwise to carry out a resection if the organisms are resistant to all antibiotics.

Other Measures.—Minor collapse procedures, such as artificial pneumothorax, phrenic paralysis, and pneumoperitoneum and variations of the operation of thoracoplasty, such as extra-pleural pneumothorax and plombage, have been rendered obsolete by the continued efficiency of chemotherapeutic agents.

FIG. 782.—Upper lobe of left lung (surgical specimen) showing large cavity at apex (upper arrow) smaller cavity below and tuberculous pneumonia.

POST-OPERATIVE PULMONARY COMPLICATIONS

The pulmonary complications of general surgery are the cause of serious morbidity and an appreciable mortality, and yet they can be largely prevented by adequate pre-operative and post-operative care.

Predisposing Factors.—(1) *Type of Operation.*—Operations on the upper abdomen and on the upper respiratory tract are more commonly implicated than others, but no operation is exempt. Operations for septic conditions are more prone to be followed by complications.

(2) *Sex.*—Males are more commonly affected than females.

(3) *Age.*—Complications are particularly frequent in the very young and the elderly.

(4) *Chronic Bronchitis.*—Chronic bronchitis is an important predisposing condition.

(5) *Smoking.*—Probably through the production of chronic bronchitis, heavy smokers are more prone to complications than non-smokers. Morton records 60 per cent. of complications occurring in heavy smokers and less than 10 per cent. in non-smokers.

(6) *Anæsthetic.*—The anæsthetic plays some part though this is less than is usually thought. Trauma to the tracheo-bronchial tree should be avoided, and prolonged post-operative unconsciousness is undesirable.

(7) *Pain.*—Post-operative pain is an important factor as it restricts coughing and deep breathing.

(8) Lack of mobility in bed and dehydration predisposes to venous stasis and thrombosis.

(9) Abdominal distension due to paralytic ileus (p. 1052).

(A) Excessive Bronchial Secretions

Abnormal bronchial secretions account for the majority of the chest complications of surgery. Many patients are a little 'chesty' following even the most trivial operation. This 'normal' post-operative bronchitis, however, may lead to very serious and perhaps fatal sequelæ unless adequately controlled.

Preventative Measures.—Adequate time should be devoted to preparing patients for operations of election. This can usefully be done in a regular

Hugh John Vivian Morton, Contemporary. Anæsthetist, Hillingdon Hospital, Uxbridge, Middlesex, England.

pre-operative clinic where breathing exercises and coughing instruction is given, dental sepsis is attended to and bronchitis treated by antibiotics. The latter may be continued into the postoperative period. A routine chest X-ray may reveal unsuspected tuberculous or other lesions. Smoking of cigarettes should be forbidden for several days before operation.

The following complications may be encountered, but it should be realised that one may merge into another, or neglect of one frequently leads to the production of more serious conditions.

(1) *Bronchitis.*—This is the commonest complication and is important in that it is often the precursor of more serious lesions. It may arise *de novo* or represent an exacerbation of a pre-existing bronchitis. The attack may vary considerably from a simple post-operative cough with mucopurulent sputum to severe suppurative bronchitis. Signs in the lungs are generalised, and there are no radiographic changes to be seen.

(2) *Bronchopneumonia* (Aspiration Pneumonia).—This is usually a sequel to the above. Patchy consolidation occurs with more profound systemic changes. The commonest organisms are *Hæmophilus influenzæ* and pneumococci but cross-infection with ward organisms such as drug-resistant staphylococci and *P. pyocyaneus* may occur. Their presence should be sought by microscopy and culture of the sputum. Signs are more localised and bronchial breathing may be heard. Radiographs will reveal patchy mottling.

(3) *Atelectasis.*—This is produced by occlusion of a bronchus by viscid secretions of mucus or pus and is usually a sequel to bronchitis. The obstruction leads to absorption atelectasis of the involved lobe. Secondary invasion of the lobe by pathogenic organisms results in particularly serious changes. Depression of the cough reflex by pain or sedation and poor ventilation are important predisposing factors. Atelectasis usually occurs after the second post-operative night, frequently following heavy sedation. Secretions accumulate during sleep, and by morning have blocked the bronchus. The patient does not feel quite so well and there may be slight fever, tachycardia and breathlessness. Cough and sputum may not be obvious as the latter cannot be expectorated. Signs consist of restricted movement of the affected side of the chest with diminished breath-sounds and impaired percussion note over the affected lobe. Radiography will reveal the dense opacity of the atelectatic lobe.

(4) *Lung Abscess.*—This usually follows bronchopneumonia or atelectasis, and is often the result of ineffective early treatment. It is a particularly serious complication often resulting in spreading suppuration throughout the lung by organisms which are by then often resistant to all drugs.

(5) *Empyema.*—This is an occasional complication of any of the above; its management is no different from the average case.

Treatment.—*Prophylactic.*—At the conclusion of an operation the tracheo-bronchial tree should be aspirated if any secretions are suspected. Early return of consciousness and the cough reflex are desirable. Subsequently coughing and breathing exercises combined with regular rolling

of the patient from side to side are desirable. Pain should be controlled by repeated small doses of analgesics of which pethidine (50–100 mg.) is the most useful, as it does not depress respiration.

The Established Case.—It is important to encourage the expectoration of sputum by all possible means. Much can be done by the physiotherapist and the nurse, whilst steam inhalations and saline expectorants help to loosen the sputum. When the secretions cannot be adequately removed naturally, they must be removed by catheter suction. If the simpler measures are not soon effective *bronchoscopy* should be carried out. This can readily be performed in the ward if the patient is too ill to move. It should be performed with minimal local anæsthesia so that the cough reflex is preserved. A careful and unhurried clearance of the whole bronchial tree should be the aim. Bronchoscopy usually results in the aeration of an atelectatic lobe, but the condition may recur and further bronchoscopies be required. In such cases it is often preferable to perform a *tracheostomy*, particularly when secretions are profuse or the patient dyspnœic. Tracheostomy by reducing the ' dead space ' greatly improves the efficiency of respiration; it also provides easy access for repeated and atraumatic aspiration of the tracheo-bronchial tree. In addition to post-operative complications it has an important place in the management of severe chest injuries, head injuries, bronchopneumonia and poliomyelitis.

Subphrenic Abscess (p. 972).

Broncho-pleural Fistula

This complication is one peculiar to thoracic operations involving resection of lung tissue. Several factors may be involved in its production.

(1) Poor surgical technique in closing the bronchus (sutures too tight or imperfectly placed).

(2) Infection or infiltration of the bronchial stump (pyogenic, tuberculous or malignant).

(3) Indifferent healing (elderly and debilitated patients, and after deep X-ray therapy).

Symptoms usually appear towards the end of the first week after operation; fever and blood-stained sputum are the first signs. Subsequent developments depend on the size of the fistula.

A small leak allows air to enter the pleural cavity producing a pneumothorax and collapse of the lung whilst pleural fluid enters the bronchial tree giving rise to a persistent cough with much thin blood-stained sputum.

A larger fistula may give rise to serious flooding of the bronchial tree and the development of a tension pneumothorax.

Treatment.—The first essential when a fistula is suspected is to anticipate and prevent flooding of the bronchial tree by early aspiration of fluid from the pleural cavity and by nursing the patient with the affected side down.

A small leak may be controlled by repeated aspirations or by temporary tube drainage until the fistula heals.

Larger fistulæ should be treated at an early stage by re-opening the chest and resuturing the bronchus. The suture line should be reinforced by a pedicled intercostal muscle graft. If the pleural cavity is already infected, however, it is better to drain the pleura and carry out bronchial repair at a later date.

Pulmonary Embolism (see p. 175).

Between 2 and 3 per cent. of all hospital deaths are due wholly, or in part, to pulmonary embolism. The condition is by no means limited to surgical wards. The clot most commonly originates in the veins of the calves, thighs or pelvis. Two types of venous lesions occur :

(1) *Thrombophlebitis.*—The thrombosis is secondary to infection or trauma. There is a marked inflammatory reaction and the resulting clot is firmly adherent to the vessel wall and is rarely dislodged.

(2) *Phlebothrombosis.*—Clotting is secondary to venous stasis and there is no inflammatory reaction in the veins. The clot is soft and insecurely attached to the vessel wall. Detachment of the clot is easy, particularly as the condition is often unsuspected and movements uninhibited (p. 176).

Clinical Features.—Pulmonary embolism is the most dangerous complication of phlebothrombosis. Much depends on the size of the embolus. *Small emboli* may be silent but the cumulative effect of many such episodes leads to a reduction in the pulmonary vascular bed resulting in pulmonary hypertension. Patients require permanent anticoagulants. *Medium emboli* lodge in branches of the pulmonary artery and result in pulmonary infarction without any hæmodynamic disturbances. Classically the onset is abrupt with pain in the chest, dyspnœa, and hæmoptysis. Chest radiographs reveal a triangular infarcted segment. Signs of venous thrombosis may or may not be present. Anticoagulants should be administered without delay and the patient immobilised for one week. Recurrent emboli are frequent. *Large emboli* lodge in the main pulmonary artery or one of its major branches. They produce serious hæmodynamic disturbances and may be rapidly fatal. Confusion with coronary thrombosis is easy but an E.C.G. is often of diagnostic value, showing right ventricular strain in pulmonary infarction. A massive pulmonary embolus may produce immediate death from ventricular fibrillation or complete obstruction of the circulation. Such cases often have an urgent desire for a bed-pan but are dead before one is produced. Those who survive complain of severe præcordial pain, tightness in the chest, and marked dyspnœa. Shock is marked and the patient becomes grey and cold with a rapid, feeble pulse, low blood pressure, and raised venous pressure. Death may occur at any moment, but the longer the patient survives the better are his prospects of recovery.

Management of Massive Pulmonary Embolism.—Patients fall into one of two categories:

(*a*) Those who succumb within 2 hours of the embolism, and

(*b*) Those who survive this critical period.

In the former group cardiac arrest is frequent and closed cardiac massage

will be required. This may serve to dislodge the clot and permit a limited pulmonary circulation. There is insufficient time to mount cardio-pulmonary bypass, and pulmonary embolectomy with temporary inflow occlusion should be attempted.

In the second group a reasonable though precarious cardiac output is maintained and there is time to carry out essential investigations, to transfer the patient to a special centre or prepare cardio-pulmonary bypass. Confirmation of the diagnosis is essential by angio-cardiography before definitive treatment is started. The cardiac catheter is left in the pulmonary artery and 600,000 units of streptokinase is injected followed by 100,000 units hourly for 72 hours. Laboratory control of coagulation and lysis are essential. Improvement in cardiac output should occur within 6 hours. Thrombolytic therapy should be continued for 3–4 days in favourable cases. If deterioration sets in, pulmonary embolectomy should be performed. Heparin should not be used with fibrinolysins.

Pulmonary Embolectomy (Trendelenburg Type).—The pulmonary artery is exposed through a left fourth space thoracotomy. Several stay sutures are placed on either side of the proposed incision in the pulmonary artery. A clamp is placed across the base of the pulmonary artery and aorta through the transverse sinus. The pulmonary artery is incised and clot removed with a sucker. Both branches of the pulmonary artery must be explored with the sucker end. After 90–120 seconds of occlusion the arteriotomy is controlled with a clamp and the occlusion clamp across aorta and pulmonary artery removed.

Pulmonary Embolectomy with Bypass.—Anæsthesia is not started until the pump is primed, the surgical staff scrubbed and all in readiness.

A mid sternal splitting incision gives quick access to the heart. A large venous tube is inserted into the right atrium and arterial return effected through a small incision in the ascending aorta. Bypass is then established and the pulmonary artery opened and clot removed with sponge forceps or a sucker. Careful exploration of the pulmonary arteries on both sides should be carried out and all fragments of clot removed.

Pulmonary Embolectomy with Inflow Occlusion.—This procedure is advised where bypass facilities are not available. The heart is exposed through a median sternotomy, the pericardium is opened longitudinally. Tapes are placed around both cavæ. With the tapes tightened, the right ventricle rapidly becomes empty and the pulmonary artery can be opened and clot removed with forceps and suction. Venous occlusion should only be maintained for about 60 seconds but can be reapplied several times to enable all clot to be removed.

Venous Thrombectomy.—Removal of residual clot by a Fogarty catheter is advocated by many surgeons to reduce the likelihood of further emboli and to help re-establish a normal circulation in the affected limb. Ligation or plication of the inferior vena cava is advocated by some to prevent further emboli reaching the lungs (p. 177).

LUNG TRANSPLANTATION

Transplantation of the lung has been successfully carried out on many occasions but to date the longest survivor died 10 months after the operation. Many technical problems such as ischæmia at the bronchial suture line and stenosis of the vascular anastomoses remain to be solved. In addition, the problems of rejection are not fully solved. (See Chap. 61.)

THE DIAPHRAGM

Herniation of an abdominal viscus through the diaphragm into the chest may occur through a congenital or acquired defect; the former occur at

Friedrich Trendelenburg, 1844–1924 (see footnote, p. 75). Described pulmonary embolectomy in 1908. The operation was first performed successfully by Martin Kirschner (see footnote, p. 354) in 1924.
Thomas J. Fogarty, Contemporary. Assistant Professor of Surgery, Stanford University, San Francisco, California, U.S.A.

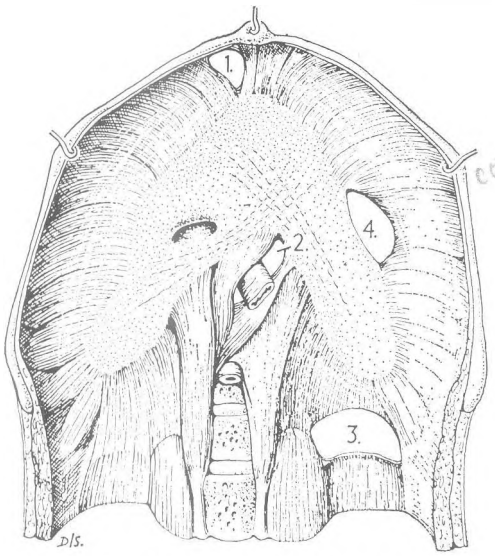


FIG. 783.—The usual sites of congenital diaphragmatic herniæ. 1. Foramen of Morgagni. 2. Œsophageal hiatus. 3. Foramen of Bochdalek (pleuro-peritoneal hernia). 4. Dome.

certain well-recognised points (fig. 783) and are due to a failure of fusion of the various elements which make up the diaphragm. Such herniæ usually have a well-defined sac and adhesions are uncommon. Other congenital malformations may be present.

The acquired group are either traumatic or post-operative. They may occur anywhere in the diaphragm and extensive adherence to surrounding structures is usual. Stomach, small or large bowel, omentum or spleen are commonly found occupying the hernia. Liver and kidney are less frequently seen.

Symptoms may only occur when either obstruction or strangulation supervene. Many patients, however, complain of vague dyspeptic symptoms but if the hernia is large, especially in children, respiratory embarrassment may occur. In infants a diaphragmatic hernia is one cause of acute respiratory embarrassment demanding urgent surgical correction. The lung on the affected side is often hypoplastic.

Surgical repair is recommended in all cases in order to avoid serious complications. Access is best obtained through a low thoracotomy incision. The sac is excised and the defect is repaired by a double row of non-absorbable sutures. Occasionally reinforcement with fascia lata is required. In infants and small children with large hernias there may be difficulty in accommodating the abdominal viscera in the abdomen.

(1) **Eventration.**—This condition is due to defective development of the whole or part of the muscle of the diaphragm; the latter consists of a sheet of fibrous tissue covered with pleura and peritoneum. The thin, flaccid diaphragm is raised and immobile. Symptoms are uncommon and the condition is usually discovered on routine X-ray. If respiration is embarrassed plication is indicated.

(2) **Œsophageal Hiatus Hernia.**—This is the commonest of congenital abnormalities but may also occur as an acquired condition in the middle-aged or elderly. It is considered on p. 795.

(3) **Hernia through the Foramen of Morgagni.**—The defect lies between the sternal and costal attachments of the diaphragm; it is more common on the right side (fig. 784).

(4) **Hernia through the Foramen of Bochdalek** (Pleuro-peritoneal Canal).—The canal may remain open with free communication between the

Giovanni Battista Morgagni, 1682–1771. Professor of Anatomy, Padua, Italy, for 56 years; from 1715 until his death in his 90th year.
Victor Alexander Bochdalek, 1801–1883. Professor of Anatomy, Prague, Czechoslovakia.

abdominal and pleural cavities. The hernia in such instances has no sac and the abdominal organs move freely in the chest, sometimes nearly filling the hemithorax and displacing the heart to the opposite side. More commonly the canal is covered by pleural or peritoneal membranes but muscle is absent, and in these cases the hernia has a sac and is usually smaller.

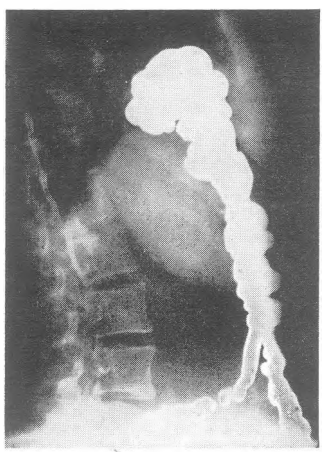


FIG. 784. — Colon occupying a Morgagni hernia. (*Dr. Oliver Smith, Birmingham.*)

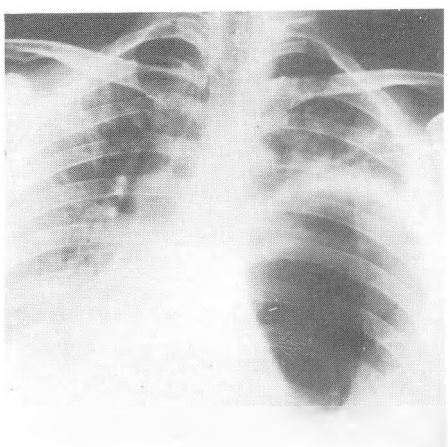


FIG. 785.—A large traumatic diaphragmatic hernia due to a penetrating thoraco-abdominal wound with a retained foreign body.

(5) **Hernia through the Dome.**—These are usually left-sided and may occur anywhere in the diaphragm. They are commonest, however, at the junction of the muscular and tendinous portions.

(6) **Traumatic Diaphragmatic Hernia.**—The diaphragm may be torn directly by a missile (thoraco-abdominal wound) (fig. 785) or ruptured by severe lower thoracic or abdominal compression (crush injury) without an external wound.

The defect may occur anywhere in the diaphragm in the former instance, but with crush injuries the dome is usually torn. Herniation may occur immediately with damage to liver or spleen or be delayed for some considerable time. A peritoneal sac is not present and adhesions between the lung and the herniated viscera always occur. Dyspeptic and obstructive symptoms are common and strangulation occasionally occurs.

Repair of the hernia should always be undertaken.

(7) **Post-operative Diaphragmatic Hernia.**—Occasionally herniation follows thoraco-abdominal operations, particularly those involving an intrathoracic œsophagogastric anastomosis. Herniation may occur around the new hiatus or through the sutured diaphragm. Great care should be taken in suturing the diaphragm to the stomach or jejunum in these cases, whilst the diaphragm is best closed with a reasonable overlap of the edges and with two rows of sutures.

Diagnosis may be extremely difficult as there are many other causes of

vomiting, pain, etc., in the post-operative period. Radiography is often of little use as the suspected area is obscured by effusion, etc.

An early exploration is advisable if the condition is suspected.

MEDIASTINAL TUMOURS

The term ' tumour ' covers a wide variety of conditions which are often difficult to distinguish, but precise diagnosis is not of great practical importance as the majority of innocent ' tumours ' require removal, and those which are not amenable to surgery, e.g. secondary carcinoma, lymphosarcoma, lymphadenoma and reticulosis, are usually readily recognised. Innocent ' tumours ' should be removed owing to the risks of malignant degeneration or compression or involvement of the mediastinal structures. The most important feature in differential diagnosis is the position of the tumour in the mediastinum, and a useful practical classification is as follows :

(1) **Anterior Mediastinal Tumours**
Retrosternal goitre.
Thymic tumours and cysts.
Persistent or enlarged thymus.
Teratoma.
Pleuro-pericardial cyst.

(2) **Posterior Mediastinal Tumours**
Neurogenic tumours (neurofibroma, ganglioneuroma).
Foregut cysts (paratracheal, para-œsophageal cyst, gastric cyst of mediastinum).
Œsophageal hiatus hernia with thoracic stomach.

(3) **Tumours arising anywhere in the Mediastinum**
Lymphadenopathies (these are often multifocal and the lesions are characteristically lobulated). Secondary carcinoma, tuberculous adenitis, lymphadenoma, reticulosis, and sarcoids are the commonest varieties.
Lymphogenous cysts.
Lipoma.
Fibroma.

Diagnosis in the difficult lymphadenopathies can often be made by a scalene node biopsy or mediastinoscopy.

The majority of the solitary lesions require operative removal if serious complications are to be avoided.

Retrosternal Goitre (p. 623).—This produces an anterior mediastinal tumour, often of characteristic shape and distribution. It is frequently bilateral though usually larger on one side. The tumour has a broad base superiorly which merges and cannot be separated from the tissues of the neck on radiological examination. Calcification is not uncommon as the tumour has probably been present for many years. This is one of the few tumours which compress the trachea (scabbard trachea, fig. 786).

Removal through a cervical incision is usually possible, and only occasionally is a sternal-splitting approach required.

THE THYMUS

Anatomy.—The gland originates as a diverticulum of the third and sometimes the fourth pharyngeal pouch on each side. Epithelial in origin, it soon assumes a lymphoid character. The body of the thymus consists of two lobes closely applied to

one another in the middle line, overlying the upper part of the pericardium and the great vessels, and extending upwards into the base of the neck. Each lobe is overlapped by pleura, and above this level has a slender pole passing to the isthmus of the thyroid. The blood supply of the gland is derived mainly from the internal mammary arteries. After puberty, the thymus commences to atrophy, and is replaced by fat, but even up to, and after, the age of fifty, the gland still contains a considerable amount of characteristic lymphoid tissue with epithelial elements, notably Hassall's corpuscles.

The Thymus and Immunity.—Most of our knowledge about the relationship of the thymus to immune processes is derived from observations made on thymectomised young mice and other mammals. Immunologically-active lymphocytes first appear in the thymus and are later seeded in other conglomerations of lymphoid tissue. Early thymectomy results in a depletion or absence of these cells in the body. In addition there may be depletion in immunoglobulin levels and some antibody responses are decreased or delayed. The thymectomised mice will accept homograft transplants with little or no evidence of rejection.

Hyperplasia of the thymus occurs in toxic goitre, acromegaly, in some cases of Addison's disease, after castration, after bilateral adrenalectomy, and in myasthenia gravis. In infancy the gland may be enlarged (fig. 787). In the past this condition

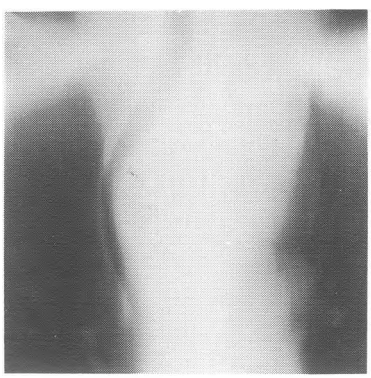


FIG. 786.—Tomogram from patient with retrosternal goitre showing displacement and compression of the trachea.

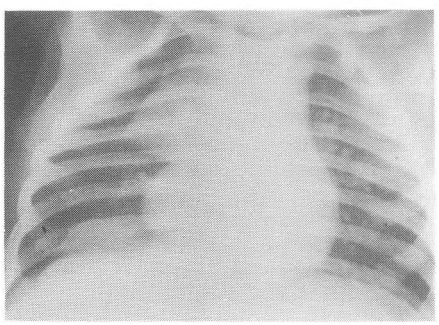


FIG. 787.—Typical sail-shaped upper mediastinal shadow due to enlarged thymus in an infant.

was called '*status thymo-lymphaticus*', and thought to be an explanation of sudden death under anæsthesia in children. This theory is no longer acceptable. Enlargement of the gland in infants may compress the trachea causing stridor and wheeziness (thymic asthma). Symptoms usually subside spontaneously but if severe and persistent, treatment with radiotherapy or surgery may be required.

Tumours of the thymus are not uncommon. Classification is difficult, but Thompson and Thackray describe three main histological types: (1) Epithelial. (2) Lymphoid. (3) Teratomatous. The epithelial type is the commonest. Cyst formation, lobulation by fibrous tissue, necrosis, and calcification are sometimes seen. Between 10% and 20% of thymic tumours are associated with myasthenia. Conversely, about 15% of myasthenics are found to have a thymic tumour; half are invasive and malignant and associated with severe and intractible disease; the remainder are histologically indistinguishable from normal thymic tissue and are non-invasive and do not appear to influence the severity of the myasthenia nor its response to treatment. Thymic tumours occupy the anterior compartment of the superior mediastinum. The majority of tumours are discovered by routine radiography. They are not dense radiologically unless calcified and are thus not visible on postero-anterior radiographs unless large enough to project beyond the mediastinal contour. In the lateral film they may be visible in the normally translucent area of the anterior superior mediastinum (fig. 788). Tumours visible on both sides of the

Arthur Hill Hassall, 1817–1894. General Practitioner, Microscopist and Chemical Analyst, Kensington, London.
Robert Edward Mervyn Thompson, Contemporary. Reader in Bacteriology, Middlesex Hospital, London.
Alan Christopher Thackray, Contemporary. Professor of Morbid Histology, Middlesex Hospital, London.

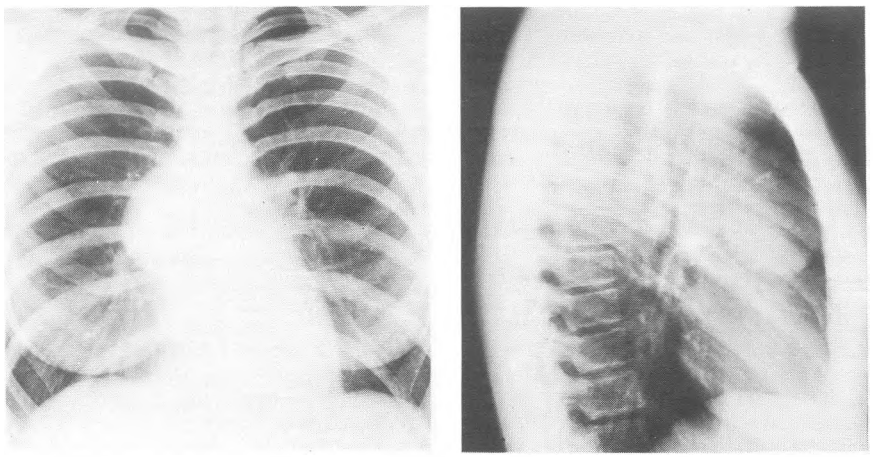
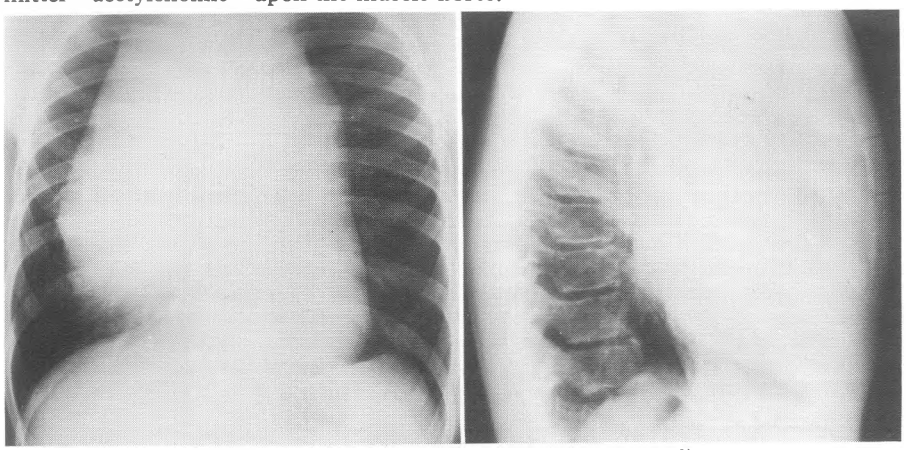


FIG. 788.—PA and lateral radiograph of thymic cyst. The mass occupies the anterior superior mediastinum.

normal mediastinal contour are likely to be invasive and malignant (fig. 789). A few may produce signs of superior mediastinal obstruction.

Removal of the tumours is advised in the majority of patients owing to the very real risks of progressive growth and the development of invasive characteristics. The tumour is approached by a sternal-splitting incision which gives excellent access and facilitates complete removal. In those instances where the lesion is invading the surrounding structures widely a histological diagnosis should be obtained before starting radiotherapy. This can be achieved by an anterior mediastinal exploration through a small incision in the second or third intercostal space.

Myasthenia Gravis.—In this disease the transmission of motor-nerve impulses at the neuromuscular junctions is blocked by interference with the action of the transmitter—acetylcholine—upon the muscle fibres.[1]



(*a*) (*b*)

FIG. 789 (*a*) and (*b*).—PA and lateral radiograph of malignant thymoma. The tumour presents on both sides of the mediastinum. In the lateral film it is seen to lie anteriorly.

[1] At a postgraduate course in 1934, Mary Walker, a Scottish doctor working in London, learned that physostigmine counteracts curare, and gave the former to a myasthenic patient. Luckily this patient was spared the side-effects (vomiting). This breakthrough met with fierce opposition, but she was vindicated by the successful series of Dr. L. P. E. Laurent, Physician, West London Hospital who reported remission during pregnancy (see text), and the introduction of prostigmine (neostigmine) which has less side-effects.

Clinical Features.—The disease occurs in both sexes and commences, as a rule, in early adult life. The essential symptoms are profound fatigue after modest exertion, and transient paresis of voluntary musles. Muscles supplied both by the cranial and spinal nerves are affected, the extrinsic ocular muscles being involved most constantly. Usually ptosis (fig. 790), a squint, and diplopia are the first symptoms. Later manifestations are drooping of the mandible, with weakness of mastication and difficulty in swallowing. The voice becomes weak, and in some cases death occurs within a few months from involvement of the muscles of respiration. Usually the disease runs a chronic progressive course, but partial remissions occur. The reflexes remain normal, and there is no sensory loss. Occasionally myasthenia gravis complicates primary thyrotoxicosis. Should a patient with myasthenia gravis become pregnant, remission of symptoms during pregnancy often occurs.

FIG. 790.—Myasthenia gravis. Before neostigmine. *(Arthur Makey, F.R.C.S., London.)*

FIG. 791.—Myasthenia gravis. One hour after neostigmine. *(Arthur Makey, F.R.C.S., London.)*

Confirmatory Tests.—(1) Edrophonium chloride (Tensilon), 10 mg intravenously relieves signs and symptoms of myasthenia in 1 minute though they usually return in 10 minutes. Tensilon is only used diagnostically in this way because it has such a short action. Alternatively 2 mg neostigmine and 0·65 mg atropine can be given but the response is delayed for ½–1 hour (figs 790 and 791). (2) The affected muscles often show the myasthenic reaction—fatigue with faradic, but not with galvanic, stimulation.

Radiography, particularly a lateral radiograph, of the superior mediastinum should always be undertaken. In 15% of cases a thymic neoplasm is revealed. Apart from tumours, there is no X-ray evidence of thymic enlargements in myasthenic patients.

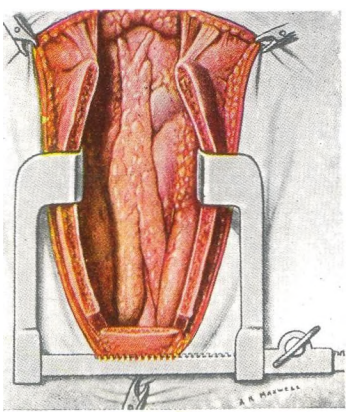


FIG. 792.—Exposure of a tumour of the thymus by splitting the sternum. *(Sir Geoffrey Keynes, F.R.C.S., London.)*

Treatment.—Medical.—Orally, from 4 to 15 tablets (15 mg. each) of neostigmine can be given in the twenty-four hours. They should be so spaced as to enable meals to be taken without difficulty in mastication. Pyridostigmine (mestinon) is a related longer-acting compound which can be used in combination with, or instead of, neostigmine. It is particularly valuable for nocturnal administration. In critical cases neostigmine may be given in a slow drip infusion. Oral potassium salts given in large dosage (12 g per day) are often beneficial. Ephedrine and Guanidine may also be of benefit. Atropine should be administered if bowel activity is increased by the anti-cholinesterase drugs.

Surgical Treatment.—Thymectomy is advised for those patients who are not adequately controlled by medical measures and especially for the more severe cases. Best results are obtained in younger patients especially females with a short history and in these the indications are less strict. The operation, however, should not be denied the elderly male patient with a long history if medical management is proving difficult. The presence of a thymic tumour makes a good operative result less likely and some recommend pre-operative radiotherapy in such cases.

Sir Geoffrey Langdon Keynes, Contemporary. Consulting Surgeon, St. Bartholomew's Hospital, London.

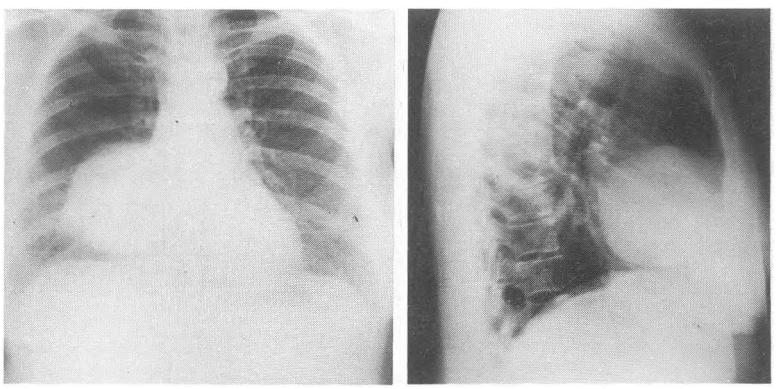
Thymectomy.[1]—Muscle relaxants are currently advised by many anæsthetists on the basis that 24 hours of artifical ventilation after operation has many advantages and permits the clinician to omit all drugs during this period.

The gland is best approached by a vertical full sternal splitting incision. The pleural cavities should be avoided if possible but if opened an intercostal drainage tube should be inserted at the end of the operation and connected to an under-water seal.

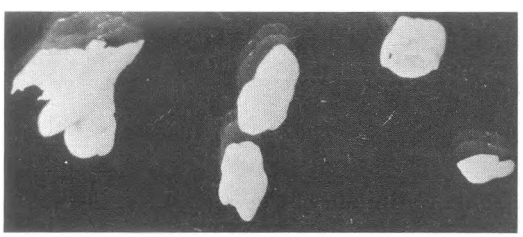
The gland is readily found as it is usually larger than normal. It is dissected carefully from its surroundings and removed in its entirety. The thymic vein passes directly backwards from the posterior aspect of the thymus to the left innominate vein and should be carefully displayed and divided after ligation. The mediastinum and the pleural cavity should all be drained for at least 24 hours.

After operation the patient is best managed in an intensive care unit, for respiratory problems are the main concern. An immediate post-operative chest radiograph is important to exclude a pneumothorax or a hæmothorax. Artificial ventilation through an endotracheal tube for 24 hours is advisable. Thereafter assisted respiration can usually be reduced, provided there is no respiratory infection and the patient's respiratory muscles are strong enough. Tracheal suction may be required. A naso-gastric tube will allow the crushed anticholinesterase drugs to be given into the stomach, which is the most effective route, but they should be withheld until their need is apparent so as to prevent the serious complication of a cholinergic crisis.

Results of Treatment.—The operation mortality is approximately 5%. Complete remission is obtained in nearly 50% of favourable cases (young females with a short history) whilst many of the remainder are able to manage more satisfactorily on smaller doses of drugs. In the older age group the results are less dramatic and are often delayed. In patients with invasive thymic tumour the results may be disappointing even when the tumour has been totally excised.



(*a*)



(*b*)

FIG. 793.—(*a*) PA and lateral radiograph of patient with a mediastinal teratoma. The tumour lies anteriorly and with a pedicle attached beneath the aortic arch. (*b*) X-ray of excised tumour showing formation of teeth.

[1] Alfred Blalock, 1899-1964. Professor of Surgery, Johns Hopkins University, Baltimore, was the first to perform thymectomy successfully for myasthenia gravis in 1939.

Pre-operative radiotherapy is only occasionally employed at the present time being usually reserved for the acute case with a radiologically visible tumour.

Teratoma.—The commonest 'inclusion' tumour is the teratoma; true dermoid cysts are only occasionally encountered. The tumours are almost always situated anteriorly with a pedicle attached deeply beneath the aortic arch (fig. 793). They are often slightly lobulated and may project into either side of the chest. Irregular areas of calcification, bone or tooth formation are sometimes seen.

Rapid increase in size without malignant change is sometimes associated with pregnancy. Occasionally a positive Aschheim Zondek test is obtained in non-pregnant patients. Infection and malignant transformation are not uncommon complications. Removal is indicated in all cases, and is best carried out through a wide lateral thoracotomy.

Mediastinal Cysts.—These are usually developmental in origin and may be associated with the trachea, bronchi, or œsophagus (foregut cyst), pericardium (pleuro-pericardial, spring-water cyst), or lymph nodes (lymphogenous cysts).

They are characteristically low-tension cysts and so may alter their shape with posture, respiration or after the induction of an artificial pneumothorax.

Foregut cysts are likely to become infected and may rupture into the trachea, œsophagus, lung or bronchus. Removal is desirable.

Neurogenic Tumours.—These have already been considered under chest-wall lesions (p. 721). They consist of neurofibromas arising from the intercostal nerves and ganglioneuromas from the sympathetic chain. They lie posteriorly in the costovertebral gutter. Occasionally they extend through the intervertebral foramen into the spinal canal (dumb-bell tumours). Malignant change may occur.

Selmar Aschheim, Contemporary. Formerly Honorary Professor of Gynæcology, University of Berlin.
Bernhard Zondek, 1891–1966. Professor of Obstetrics and Gynæcology, Hadassah Medical School, Hebrew University, Jerusalem, Israel.
Aschheim and Zondek described this test for pregnancy in a joint paper in 1928.

CHAPTER 41

THE HEART AND PERICARDIUM

SPECIAL METHODS OF INVESTIGATION

MODERN cardiac surgery demands precise methods of investigation to provide accurate anatomical and physiological details of the various cardiac abnormalities. It is only when such information is available that a precise diagnosis can be made and surgical treatment planned. The most important available methods are :

(1) **Radiology** (standard techniques and fluoroscopy). Such examinations provide details of the cardiac contour and variations in size of the chambers; it also provides information about the pulmonary circulation.

(2) **Angiocardiography** (figs. 794 and 795).—The injection of 76% Urografin in doses of 1 ml. per kg. is used to outline the chambers of the

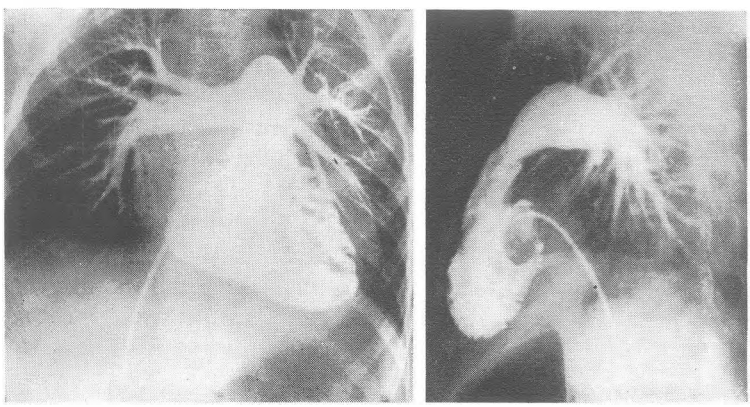


FIG. 794.—Normal angiocardiogram showing the right side of the heart and pulmonary arteries.

heart and great vessels and films can be taken at frequent intervals during the passage of the opaque material through the heart. Important anatomical information concerning the size and shape of the heart chambers, the presence of a shunt or of obstruction or incompetence of a valve may be provided. The aorta and pulmonary arteries and their branches will also be outlined. Ciné-angiography gives a dynamic picture of the circulation through the heart and is especially suitable for demonstrating valvar regurgitation.

(3) **Cardiac Catheterisation.**—A fine catheter is introduced through a vein or an artery into the heart where its passage can be followed by fluoroscopy and blood samples and pressure tracings recorded. This investigation supplies functional as well as anatomical data (fig. 796).

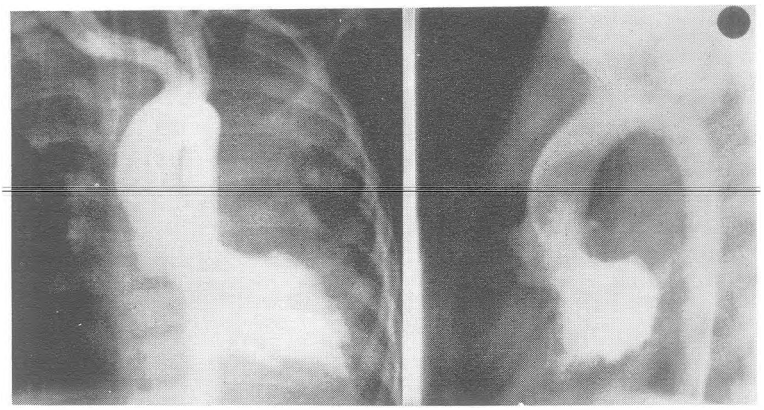


FIG. 795.—Normal angiocardiogram showing the left side of the heart and aorta.

(4) **Measurement of Cardiac Output.**—Cardiac output is an important measurement of left ventricular function, which in turn may affect both the risks of operation and the subsequent relief of symptoms. Output can be estimated by the Fick principle after measurement of the oxygen consumption and the venous and arterial oxygen contents, by thermodilution techniques, or by the use of tracer dyes. The determination, originally laborious, has been simplified by computerised techniques.

(5) **Echocardiography.**—Beams of high speed ultrasound waves are directed through the heart. Whenever the vibrations cross a boundary or interface, vibrations will be reflected back to the transmitting source provided the reflecting surface lies at right angles to the original beam. By accurate placing of the transmitter the movements of the aortic leaflet of the mitral valve, the anterior and posterior walls of the heart and the ventricular septum can all be recorded and important information obtained (fig. 797 (*a*) and (*b*)). Thickening, calcification and poor mobility of the anterior mitral cusp can be clearly demonstrated and indications for closed or open mitral valvotomy determined from this investigation alone thus avoiding the need for cardiac catheterisation and angiocardiography. Similarly pericardial effusions and atrial tumours can be diagnosed with certainty. Recent developments have included investigations of all forms of congenital heart disease and of left ventricular function by observing movements of the ventricular walls.

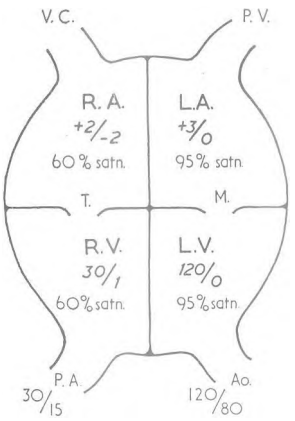
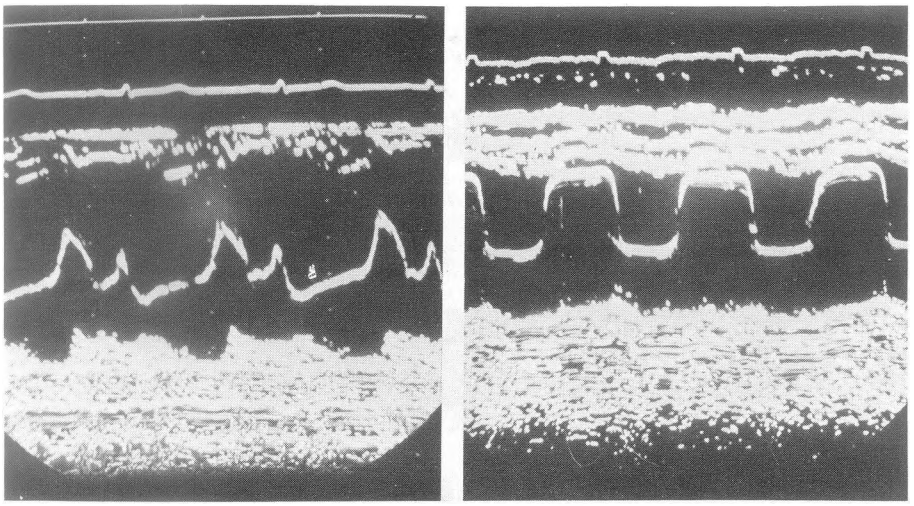


FIG. 796.—Normal intracardiac pressures and oxygen saturations.

CARDIO-RESPIRATORY RESUSCITATION

In cardiothoracic emergencies the heart and lungs should be considered as one unit: where there is circulatory failure, both pulmonary and cardiac

Adolf Fick, 1829–1901. German Physiologist.



(*a*) (*b*)

FIG. 797.—Movements of anterior leaflet of mitral valve as recorded by ultrasound, (*a*) normal cusp; (*b*) mitral stenois. The flat top of the tracing indicates reduced movement of valve leaflets during diastole. (*D. Gibson, London.*)

support will be required. Although in the early stages of a crisis only one element may be affected, its failure very rapidly implicates the other. Thus, respiratory failure rapidly leads to cardiac arrest and vice versa.

Circulatory failure is common in civil life—electrocution, drowning, asphyxia, poisonous gases and major trauma account for many cases. In addition, circulatory arrest may occur in any hospital department. It is more commonly associated with major surgical procedures especially those performed for cardiac conditions, but minor operations and diagnostic procedures are not immune. If lives are to be saved, it is essential that the mechanism of production and the methods of correction of circulatory arrest should be clearly understood. Not only should vulnerable departments be supplied with the necessary basic instruments for dealing with the emergency, but both medical and nursing personnel should be thoroughly familiar with the procedure of resuscitation.

Causes of Cardiac Arrest.—The following are the factors usually responsible for cardiac arrest :

(1) **Anoxia.**—Myocardial anoxia is probably the most frequent and important precipitating cause of arrest. It can be produced by a variety of means, such as respiratory obstruction, serious hæmorrhage, a sudden severe fall in blood pressure or coronary air embolism. Hæmorrhage from the heart is always more serious than that from the peripheral circulation owing to a marked decrease of cardiac output and rapid fall in blood pressure. Most cardiac manipulations produce a temporary fall in blood pressure, but if they are repeated or prolonged, severe and irreversible changes may develop.

(2) **The State of the Myocardium.**—A healthy myocardium will stand moderate manipulation and anoxia without ill effect, but a diseased myocardium has less reserve, and relatively minor disturbances may cause failure.

(3) **Toxic and Irritating Drugs.**—Certain drugs have a direct depressing or irritating action on the myocardium whilst others affect it indirectly through the

circulation. In the latter group, the vasodilators are the most important; these include many anæsthetic agents, procaine, etc. Chloroform, cyclopropane and adrenaline may produce cardiac irregularities, whilst marked excess of potassium may lead to cardiac arrest.

Types of Cardiac Arrest.—Sudden acute cardiac failure may be produced by:

(1) **Circulatory Obstruction.**—By a massive pulmonary embolism, air embolism, or accidental occlusion of one of the main vessels.

(2) **Asystole.**—This is usually due to severe myocardial depression. The heart beat becomes slow and feeble and finally stops. The myocardium is flabby, dilated, and cyanosed.

(3) **Ventricular Fibrillation** is usually associated with an irritable heart as a result of manipulation, trauma or drugs. In such cases, the myocardium is less depressed and more likely to recover than in the former group.

Management of Cardiac Arrest.—Speedy recognition is essential if the circulation is to be restored before irreversible damage has been done to the brain. Diagnosis is based on three signs (*a*) absent carotid pulse (*b*) absent or gasping respiration (*c*) dilated pupils. Disappearance of the pulse is the most important sign. If the carotid pulse cannot be felt, cardiac arrest must be assumed and appropriate action taken. If the circulation can be restored within three minutes of cessation, recovery is possible, but it is unlikely after an interval of ten minutes.

Vulnerable departments should prepare a simple chart outlining essential procedure for guidance to both medical and nursing staff.

Management is divided into two phases:

(*a*) **Emergency.**—These measures are carried out by the man on the spot and consist of *mouth to mouth ventilation* and *closed cardiac massage*. These can both be performed by one operator in the ratio of one breath to ten chest compressions, but are more efficiently carried out by two. (Full technical details are available in all first-aid manuals.) These two measures should be continued without interruption until a heart beat or spontaneous respirations return or until expert help arrives. If performed effectively, the vital organs can be kept alive for a few hours by these measures alone. Correction of metabolic acidosis as outlined in next section should be started as soon as possible.

(*b*) **Subsequent Management.**—Further management depends on the arrival of expert help and appropriate equipment (endotracheal tubes, ventilating apparatus, cardiograph, electrical defibrillator and thoracotomy set).

1. *Ventilation:* At the earliest possible moment an endotracheal tube should be passed and mechanical ventilation started.

2. *Cardiograph:* Electrocardiographic monitoring should be provided as soon as possible in order to determine the type of cardiac arrest (asystole or ventricular fibrillation) and demonstrate any signs of recovery.

3. *Cardiac massage:* Closed massage should be continued until cardiography is available. Further action depends on the type of rhythm.

(*a*) If ventricular fibrillation is present, attempt external defibrillation. If this is unsuccessful, give an intravenous or intracardiac injection of 100 mg. lignocaine or 250 mg. of procaine amide, or 5 ml. of 10 per cent. calcium

chloride, and attempt defibrillation after an interval of two to three minutes with continued cardiac massage. As a last resort expose the heart and carry out open massage and drug administration.

(*b*) If asystole has developed, inject 10 ml. of 1:10,000 adrenaline or 0·01 mg. isoprenaline into heart. This should induce ventricular fibrillation in which case proceed as in 3(*a*) above. If there is no response, open the chest and carry out open massage with further injections of adrenaline or isoprenaline.

(*c*) **Open Cardiac Massage.**—The chest should be opened through the fifth intercostal space on the left side by an intercostal incision extending from the sternum to the axilla. The wound does not bleed as there is no circulation. The only instrument required to permit bi-manual massage is a rib-spreader, but should this not be immediately available massage is quite possible with one hand. Massage is most efficient with two hands with the pericardium widely opened. One hand should be placed above and the other below the ventricular mass, and massage should be carried out with the flat of the hand and fingers rather that with the finger-tips. The rate should be about 50 times per minute, though this is guided by the need for a sufficient pause to permit diastolic filling. Massage should always be carried out gently as considerable damage to the myocardium is possible if excessive force is used. The object of cardiac massage is to improve the tone of the myocardium, to increase the coronary flow, and ensure a supply of oxygenated blood to the nervous centres. The arrested heart is distended, flaccid, and cyanosed, but after massage it should become smaller, firmer, and pinker. When tone has returned, attempts should be made to obtain normal beating. Before closing the chest pacemaker wires, should be attached to the left ventricle in case of further episodes of arrest.

4. *Correction of Metabolic Acidosis:* All patients develop acidosis after cardiac arrest and its presence may prevent resuscitation. Sixty ml. of an 8·4 per cent. solution of sodium bicarbonate (1 mEq. per ml.) should be given intravenously as soon as possible and further doses administered to restore the pH to normal levels (p. 100).

Later Management.—After the restoration of normal rhythm and a normal blood pressure, a careful watch should be maintained in case of recurrence. Adequate steps should be taken to correct any metabolic acidosis. The main late complications are related to cerebral and renal damage resulting from the period of circulatory arrest. If return of consciousness is delayed, cerebral dehydration as described on p. 437 should be carried out. Total body hypothermia with reduction of temperature to 32°C. may help to limit cerebral damage (p. 767). If renal activity is impaired, the measures described under renal anuria (Chap. 55) should be instituted.

THE SCOPE OF CARDIAC SURGERY

Operative treatment of cardiac abnormalities is advancing steadily, particularly with the advent of new techniques for controlling the circulation. The procedures can be, for practical purposes, placed in one of three groups :

(1) **Extracardiac Operations.**—These are carried out on the main vessels outside the heart or on the pericardium. The ventricles or atria are not directly interfered with so that cardiac function is not unduly disturbed. Examples include ligation of a patent ductus, excision of coarctation, systemic-pulmonary anastomoses, resection of aneurysms, pericardectomy and certain operations for ischæmic heart disease.

(2) **Closed Intracardiac Operations.**—These are blind intracardiac procedures performed by instrument or finger and controlled by touch. Access is obtained to the interior of the heart through either the ventricular or atrial walls or through the base of one of the great vessels. Cardiac action is interfered with to some extent so that irregularity of heart action is liable to be encountered. Mitral valvotomy is the only closed procedure still used regularly.

(3) **Open Cardiac Operations.**—The desire of every surgeon is to operate with safety under direct vision on the open and motionless heart. The following procedures have come near to achieving this goal :

(*a*) *Extra-corporeal Circulation.*—Several clinically proven heart-lung machines are now in regular use. Basically each consists of a pump and an oxygenator. Blood is withdrawn from the venæ cavæ, passed through the oxygenator and returned into the arterial circulation through the femoral artery or ascending aorta (fig. 798). The blood is thus diverted completely from the heart and lungs but a good supply of well oxygenated blood is made available to the vital organs. Heart-lung machines provide the surgeon with periods for intracardiac surgery of between two or three hours with safety. By the use of an extracorporeal circulation many complicated cardiac anomalies can be corrected and such conditions as ventricular septal defects, Fallot's tetralogy (p. 775) and valvar abnormalities are being so treated.

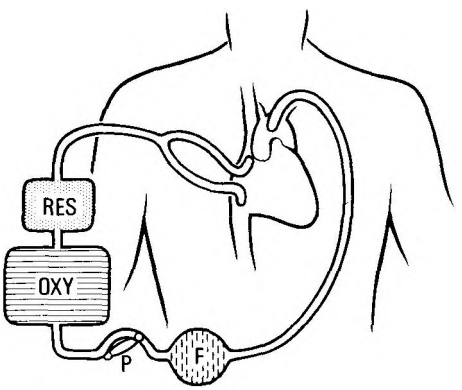


FIG. 798.—Cardio-pulmonary bypass: cannulation sites in right atrium and ascending aorta and apparatus. Res = Reservoir; Oxy = Oxygenator; P = Pump; F = Filter.

Technique.—The majority of cardiac abnormalities can be treated through a median sternotomy incision. The heart is exposed through a vertical incision. Both venæ cavæ and the ascending aorta are circumnavigated with tapes. Immediately prior to cannulation the patient is heparinised with 3 mg/kg body weight. The ascending aorta is cannulated with a plastic tube through a purse string suture placed in the aortic adventita and each cava is similarly cannulated through the right atrial wall. The cannulæ are connected to the heart-lung machine after careful elimination of all bubbles of air. At the completion of the operation the cannulæ are removed and the heparin is counteracted with protamine (6 mg/kg body weight). During heart-lung bypass the efficiency of the extracorporeal circulation is monitored by observing urinary output and cerebral activity, by measurement of blood gases and determination of serum potassium.

(*b*) *Hypothermia.*—The metabolic needs of the body can be considerably reduced by cooling the tissues, thus enabling the latter to survive short periods of total deprivation of oxygen or longer periods of partial deprivation. Originally used as a sole technique, surface cooling of the body to 28–30°C gave a surgeon about 10 minutes of circulatory arrest before endangering the

brain. This time permitted the performance of simple cardiac operations (closure of atrial defects and relief of pulmonary stenosis) but the technique has been almost entirely replaced by cardiopulmonary bypass. Greater safety with further reductions in body temperature can only be achieved with the use of cardiopulmonary bypass as ventricular fibrillation usually ensues at a temperature of about 25°C. With this technique, however, the body temperature can be reduced to 15–18°C and periods of up to 45 minutes or more of circulatory arrest can be safely tolerated. This technique is now widely used in the surgery of congenital heart disease in infants.

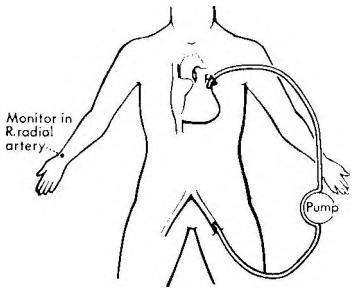


FIG. 799.—Left heart by-pass.

(*c*) *Left Heart By-pass.* — Left atrio-femoral or left heart by-pass is a useful technique which facilitates operations on the thoracic aorta. The left atrium is cannulated and blood diverted with a pump into the femoral artery (fig. 799). The aorta can now be cross-clamped and the left ventricle continues to supply the head and upper extremities with blood whilst the by-pass caters for the lower limbs and abdominal viscera.

(*d*) *Femoro-femoral Bypass.*—The femoral artery and vein are each cannulated and the blood is passed through an oxygenator before return to the body via the femoral artery. Indications for its use are similar to left atrio-femoral bypass where the left atrium is not readily accessible.

(*e*) *Vascular Shunts.*—Aortic anomalies such as aneurysms or coarctation can be bypassed by shunting blood from the left subclavian to the femoral artery. The use of a special heparinised tube for the creation of the shunt eliminates the necessity of general body heparinisation.

(*f*) *Myocardial Preservation.*—For surgery of the left heart (mitral valve, aortic valve and coronary arteries) the operator requires a motionless relaxed heart for an appreciable length of time. Much attention has recently been given to the protection of the heart muscle during this period. Various methods in vogue employ one or more of the following: (*a*) Reduction in cardiac work by use of total heart-lung bypass and venting of the left ventricular cavity, (*b*) cessation of heart beat, (*c*) cooling of myocardium, (*d*) cannulation and perfusion of the coronary arteries, (*e*) employment of cardioplegic agents injected into the coronary vessels.

THE PERICARDIUM

Cardiac Tamponade.—A rapid accumulation of fluid in the pericardial sac will compress the heart and prevent diastolic filling; this results in an increase of the venous pressure and a reduction of the cardiac output. Occurring rapidly (e.g. after injuries or cardiac surgery), it produces a shock-like state and if not relieved may prove fatal. The cardiac dullness and silhouette are enlarged and the heart sounds weak; the jugular venous pressure is raised and the pulse may be paradoxical (weaker on inspiration). Immed-

iate relief can be obtained by aspiration but, if symptoms recur, pericardial exploration will be required.

Aspiration of the Pericardium.—The pericardium should be explored with a short-bevelled needle introduced alongside the xiphoid process in an upward and backward direction with the needle at an angle of 45 degrees to the skin. This site has the following advantages:

(*a*) It does not transgress the pleura.

(*b*) It is more likely to encounter fluid which frequently gravitates between the diaphragm and the heart.

(*c*) It is less likely to damage the coronary vessels.

Acute Pericarditis.—Purulent pericarditis is now a rare complication of septicæmia, pyæmia, pneumonia or empyema. Signs and symptoms may be vague, consisting of fever, tachycardia or retrosternal pain. A friction rub may be heard and felt. X-rays may show a large pear-shaped cardiac outline, whilst the electrocardiograph shows a characteristic elevation of the S-T segment. The pericardium should be aspirated and suitable antibiotics injected. Drainage of the pericardium is required if aspirations prove ineffective in controlling the infection.

Chronic Pericarditis (Constrictive Pericarditis; Pick's Disease).—In this condition there is marked thickening, fibrosis and calcification of the pericardium which confines the heart in a rigid inelastic casing preventing it from filling in diastole and emptying in systole. The majority of cases are a sequel to a tuberculous pericarditis, but occasional cases follow purulent pericarditis or a traumatic hæmopericardium.

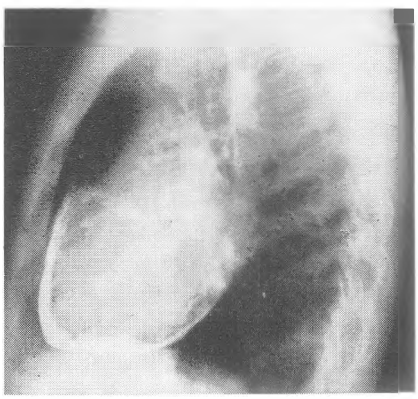


FIG. 800.—A calcified pericardium.

Hæmodynamics. — Decreased diastolic filling leads to an accumulation of blood on the venous side. The jugular veins are engorged, pleural effusions may occur, the liver is enlarged, and ascites and œdema of the ankles may appear. On the arterial side, the blood pressure is low with a small pulse volume due to a reduced cardiac output. Fluoroscopy shows decreased or absent cardiac pulsation and pericardial calcification is commonly seen (fig. 800).

Treatment.—Surgical removal of the constricting pericardium is the only effective treatment; this will allow the heart to fill and empty normally.

The patient should be prepared by salt and water restriction, diuretics, and aspiration of pleural or ascitic fluid. At operation (pericardectomy) it is essential to remove the thickened pericardium from the ventricles, but thickened or calcified plaques covering the atria or venæ cavæ should also be removed (Holman).

CONGENITAL HEART DISEASE

Approximately six babies in every thousand live births have a congenital cardiac abnormality. Many are severe and complicated so that survival beyond a few weeks or months is unlikely. Of those who do survive infancy,

Friedel Pick, 1876–1926. Professor of Laryngology, Prague, Czechoslovakia. Described this disease in 1896.
Emile Frederic Holman, 1890–1977. Professor of Surgery, Stanford University, San Francisco, California, U.S.A.

the expectation of life is often markedly reduced so that surgical correction is desirable wherever possible.

The lesions are divided into those associated with a normal arterial oxygen saturation (acyanotic group) and those with reduced saturation (cyanotic group).

Acyanotic Congenital Heart Disease

I. PATENT DUCTUS ARTERIOSUS

This is one of the commonest abnormalities, accounting for 15 per cent. of all types. It may occur as an isolated condition (simple patent ductus) or as part of a more complex abnormality (complicated patent ductus). The mechanism of normal closure of the ductus and the causes of patency are not fully understood.

Hæmodynamics—The ductus connects the left pulmonary artery with the aorta just distal to the origin of the left subclavian artery. As the pressure is higher in the aorta than in the pulmonary artery, blood will flow from the former into the latter vessel. As much as two to four times the normal cardiac output can flow through a patent ductus. On the systemic side, the systolic blood pressure rises and the diastolic pressure falls, producing a water-hammer or collapsing pulse. The amount of blood in the lungs is markedly increased and the pulmonary vessels are dilated (pulmonary plethora) and their pulsation increased (hilar dance). The additional circulation of blood through the lungs and the left side of the heart results in left ventricular hypertrophy.

Clinical Features.—Many cases are only discovered on routine examination in childhood, but a minority give rise to cardiac failure during the first year. Soon after birth, when the pressures in the pulmonary and systemic circuits are nearly equal, only a systolic murmur is audible and diagnosis is difficult. As the child grows, the difference between pressures increases and the typical continuous machinery murmur develops; this is best heard in the second and third spaces to the left of the sternum. Breathlessness on exertion and retardation of growth may be present in childhood, but symptoms are often absent until complications ensue. Fluoroscopy shows enlargement of the left ventricle with markedly enlarged pulsatile pulmonary arteries. The diagnosis is usually obvious from the presence of the continuous murmur, an increased pulse pressure with a water-hammer pulse, and the X-ray findings. Atypical cases should be investigated by cardiac catheterisation.

Complications.—(1) *Cardiac Failure.*—This is the most important complication especially in infants and probably accounts for one-third of the deaths. Failure is preceded by progressive cardiac enlargement.

(2) *Bacterial Endocarditis.*—Before the advent of antibiotics this was a frequent and fatal complication accounting for one-third of all deaths. Chemotherapy now permits control of the majority, but relapse is likely unless the ductus is ligated.

Prognosis.—The outlook for a simple patent ductus is difficult to determine. The disease is common in children and rare in adults. It is unlikely

that a patent ductus ever closes spontaneously, and it must be assumed that some patients die from cardiac failure in early adult life.

Treatment.—The results of surgical closure are so satisfactory and the mortality rate is at such a level (less than 1 per cent.) that ligation can be recommended in all cases. Recanalisation is rare. Division and suture is practised by Gross and others in order to reduce the risks of recanalisation, but is probably only necessary for the short wide ductus which cannot be safely ligated.

The operation is carried out through a left-sided postero-lateral incision through the fourth space. The mediastinal pleura is incised over the aortic arch and the vagus nerve and its recurrent branch are retracted posteriorly. The areolar tissue is removed from the surface of the ductus and the latter gently mobilised by blunt dissection. Occlusion is achieved by using two stout non-absorbable ligatures placed at either end of the ductus with a transfixion ligature between (Blalock).

II. COARCTATION OF AORTA

Narrowing of the aorta may occur at any site, but, in the vast majority of cases, the stenosis lies immediately beyond the origin of the left subclavian artery in close relationship to the ductus or ligamentum arteriosum. The lesion accounts for 6 per cent. of all congenital cardiac anomalies.

Two main types are described according to the relationship of the ductus to the coarctation:

(1) **Post-ductal Type. Adult Coarctation.**—Sixty per cent. of all cases are of this type. The ductus is patent in a minority and enters the aorta above the coarctation. The stenosis is abrupt with an internal lumen of 1 to 2 mm. Externally the aorta shows a characteristic indentation.

(2) **Pre-ductal Type. Infantile Coarctation.**—Here a patent duct enters the aorta below the coarctation. Unoxygenated blood may pass from the pulmonary artery into the aorta and thence to the lower trunk and extremities. These areas are cyanosed, whilst the head, neck and upper extremities are pink (differential cyanosis). The stenosed aortic segment may be long and include one or more of the great vessels. Forty per cent. of patients have other cardiac lesions. The mortality in the first year of life is considerable.

Clinical Features.—The obstruction results in hypertension above and hypotension below the constriction with collateral vessels linking the two areas. Symptoms can be divided into three groups:

(1) Those of hypertension above the block; headaches, irritability, excessive heart action, throbbing and pulsation in head and neck.

(2) Those due to hypotension below the block; cold legs and intermittent claudication.

(3) Complications secondary to hypertension; left ventricular failure, intracranial hæmorrhage, rupture of the aorta.

Characteristically there is marked pulsation of the carotid and subclavian arteries with evidence of left ventricular enlargement. Systolic murmurs are frequently audible over the præcordium and elsewhere over the thorax, due usually to the collateral vessels, although sometimes caused by associated aortic valve disease. Blood pressure in the arms is considerably elevated. Collateral vessels can be seen and felt over the thorax, particularly in the scapular region. Femoral, dorsalis pedis and posterior tibial pulses may

Robert Edward Gross, Contemporary. Ladd Professor of Children's Surgery, Harvard University, Boston, Massachusetts, U.S.A.
Alfred Blalock, 1899–1964. Professor of Surgery, Johns Hopkins University, Baltimore, Maryland, U.S.A.

be absent or weak. The femoral pulse, if present, is delayed when compared with the radial. Oscillometry reveals defective pulsation in the lower limbs. X-rays show evidence of left ventricular enlargement, a prominent ascending aorta and an absent or abnormal aortic knuckle (fig. 801).

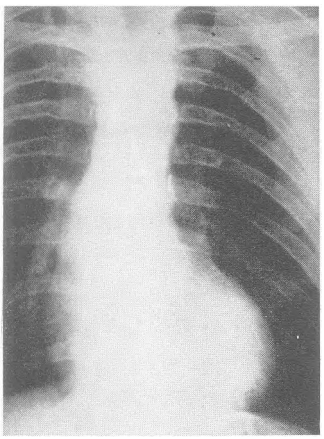


FIG. 801.—Coarctation of aorta showing a prominent ascending aorta, double aortic knuckle and rib notching.

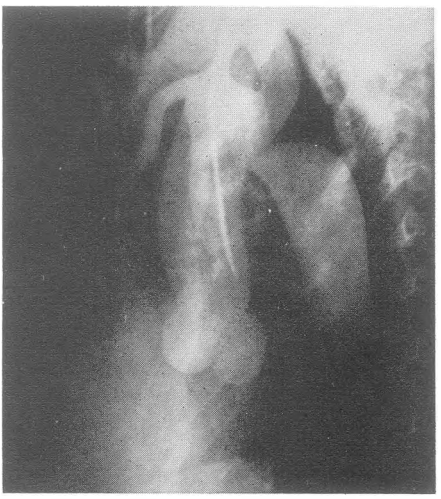


FIG. 802.—Coarctation of aorta. Aortogram showing the stenosis and the marked collateral vessels.

Notching of the ribs due to large tortuous intercostal vessels is seen in adults but rarely in children under six.

Investigations.—Venous angiocardiography or retrograde aortography (fig. 802) are employed to demonstrate the actual site and type of the coarctation, but these investigations are by no means essential pre-operatively if an aortic graft is available at the time of operation.

Prognosis.—The condition is a serious one and it is estimated that very few patients survive beyond the age of forty-five years; the majority die from the effects of hypertension.

Treatment.—Both Crafoord and Gross independently in 1945 demonstrated that the stenosis could be excised and an end-to-end anastomosis safely performed. The operation is now a well-accepted one and the mortality should not be above 5 per cent. In the majority, excision with an end-to-end anastomosis is possible. Occasionally a graft is required to bridge a wide defect in cases with a long hypoplastic segment or with a post-stenotic aneurysm. Aortic homografts were originally employed but they calcify and narrow with age and have been superseded by grafts of crimped Teflon or Dacron (p. 146). All children and young adults should be operated upon.

The operation is carried out through a left postero-lateral incision through the fourth intercostal space. The large anastomotic vessels in the chest wall are ligated. The aorta is mobilised widely both above and below the constriction, great care being

Clarence Crafoord, Contemporary. Professor Emeritus of Thoracic Surgery, Karolinska Institut, Stockholm.

taken not to injure the large thin-walled intercostal and mediastinal vessels arising from the distal segment. The ligamentum arteriosum is divided: the aorta is clamped above and below the constriction and the latter excised (fig. 803). An end-to-end anastomosis is performed using a fine (000 or 0000) eversion suture. The clamps should be released slowly and any leak from the suture-line controlled by pressure.

For patients with long coarctations a plastic reconstruction may be easier than an end-to-end anastomosis. The constricted zone is incised longitudinally and an oval patch of Dacron is inserted. An alternative approach developed by Hamilton, which is particulary suitable for infants, uses the left subclavian artery as an autologous onlay patch. All procedures in the young may need operative revision later in life.

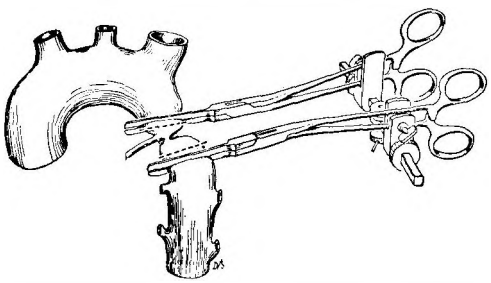


FIG. 803.—Coarctation of aorta. The mobilised aorta is clamped prior to excision and anastomosis. The clamps are held in position with the adjustable Brom's Vice.

Results of the Operation.—The over-all mortality rate for all cases is under 5 per cent., but in children it is considerably lower. A normal blood pressure in both upper and lower limbs is usual in children; in adults some elevation in blood pressure usually persists, but the pressure in the lower limbs often rises above that in the upper limbs and the femoral pulse is no longer delayed. Relief of symptoms is the rule.

III. CONGENITAL VALVAR ABNORMALITIES

Developmental abnormalities can occur with any of the valves, resulting in stenosis, regurgitation or combined lesions. Less frequently obstruction may occur in the subvalvar region.

Stenotic lesions in the pulmonary or aortic valves are commonest (all other varieties are rare). Classically the commissures are fused to form a dome-shaped valve with a small central orifice. Bicuspid valves and other complex varieties are not uncommon. The stenotic valve produces an ejection systolic murmur and thrill and evidence of Right (pulmonary stenosis) or Left (aortic stenosis) Ventricular Hypertrophy.

Both lesions can be corrected by open operation with heart-lung bypass with good results. The operation of closed pulmonary valvotomy (Brock) is rarely employed now.

IV. ATRIAL SEPTAL DEFECT

Defects of the septum between the atria account for 7 per cent. of congenital cardiac anomalies; they allow blood to flow from the left to the right atrium (L–R shunt) so that the right side of the heart and lungs are overfilled whilst the left side receives less blood than usual. Although the deformity may cause little disability during childhood and early adult life, it is a serious condition with troublesome symptoms appearing in middle life associated with the onset of atrial fibrillation.

The defects are of three main types depending on which part of the developing atrial complex is defective:

(*a*) *Secundum defect:* commonest and simplest; lies centrally in septum; caused by failure of septum secundum; direct suture closure often possible.

Gerard Brom, Contemporary. Academisch Ziekenhuis, Leiden, Holland.
David Ian Hamilton, Contemporary. Cardiac Surgeon, Royal Liverpool Children's Hospital.
Russell Claude Brock (Lord Brock of Wimbledon), 1903 1980. Surgeon, Guy's Hospital and Brompton Hospital, London.

(*b*) *Primum defect:* caused by failure of septum primum; often associated with abnormal cleft of the mitral valve and mitral regurgitation; occurs low in the septum—there is no septal tissue separating mitral and tricuspid valves; always requires a patch for closure.

(*c*) *Sinus venosus defect:* due to failure of incorporation of sinus venosus into the atrium proper; occurs high in the septum and often associated with abnormal pulmonary venous drainage; usually requires a patch for closure.

Signs are due to over-filling and -activity of the right side of the heart and lungs. The right ventricle is palpably enlarged and active. The pulmonary arteries are dilated and actively pulsatile (hilar dance) and increased flow produces a pulmonary systolic murmur. The second heart sound is widely split due to bundle branch block. The E.C.G. shows right bundle branch block and right ventricular hypertrophy (secundum defect).

Closure of the defect is readily performed by direct suture under vision with the aid of extracorporeal circulation.

The more complicated atrial defects (ostium primum: atrio-ventricular canal) can be corrected but the risks of operation are higher.

V. VENTRICULAR SEPTAL DEFECT

Isolated defects of the ventricular septum are one of the commonest congenital lesions (22 per cent.). In addition, they occur frequently as a part of more complex anomalies (Fallot's tetralogy, transposition, etc.).

The defect varies from 1 to 3 cm in diameter, most often situated in the fibrous part of the septum close to the aortic and tricuspid valves.

The defect allows the passage of blood from the left to the right ventricle resulting in over-filling of the right heart, pulmonary plethora and hypertension.

The lesion is a serious one, over 50 per cent. of patients dying during the first few months of life.

The defect is repaired with the aid of an extracorporeal circulation using either direct sutures or inserting a patch of Dacron or pericardium. The operative approach is either through the anterior wall of the right ventricle or from the right atrium via the tricuspid valve.

The operative mortality is 10 per cent., and the most serious complications are due to the production of complete heart block or incomplete closure of the defect. In infants a stout ligature can be applied around the pulmonary artery (pulmonary banding) to restrict the flow of blood into the lungs. Closure of the defect and removal of the pulmonary artery band is carried out at a later date.

Cyanotic Congenital Heart Disease

The cyanotic heart lesions include a number of different abnormalities, the commonest of which is Fallot's tetralogy (fig. 804). The abnormalities are multiple and complex and often difficult to determine accurately, but all have central cyanosis due to an intracardiac shunt from the right to the left side. From the surgical point of view the state of the pulmonary cir-

Etienne Louis Arthur Fallot, 1850–1911. Professor of Medicine, Marseilles, France.

culation is by far the most important consideration. If this is defective (pulmonary ischæmia), then surgical means can be taken to improve it and thereby increase exercise tolerance and decrease cyanosis. If, however, the lungs are overfilled (pulmonary plethora), or the pressure considerably raised (pulmonary hypertension) it may be difficult or impossible to carry out a corrective procedure with safety. The exact type of surgical procedure will depend on obtaining accurate hæmodynamic and anatomical details of the abnormality by angiocardiography and cardiac catheterisation.

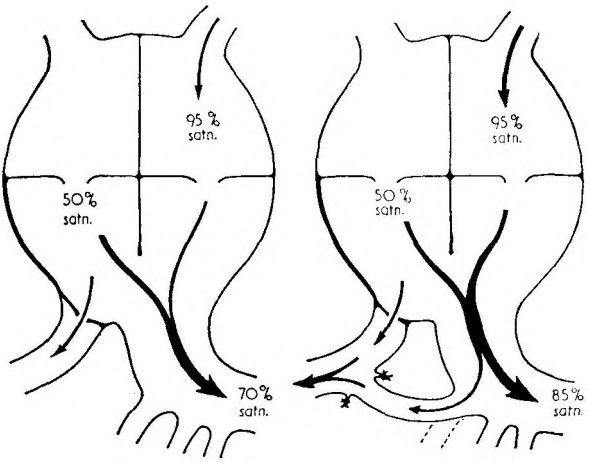


FIG. 804.—Hæmodynamics of Fallot's tetralogy before and after Blalock's operation. Note the ventrivular septal defect, the over-riding aorta, pulmonary stenosis and right ventricular hypertrophy which make up the tetralogy (cf. fig. 796).

I. FALLOT'S TETRALOGY

This is the commonest of the cyanotic conditions and accounts for about 10 per cent. of all cases of congenital heart disease.

The tetrad originally described by Fallot in 1888 consists of (1) stenosis of the pulmonary tract; (2) a ventricular septal defect; (3) an aorta which straddles the ventricular defect and over-rides both ventricles and (4) right ventricular hypertrophy.

The patient is usually undersized with considerable physical disability including central cyanosis and finger clubbing. The heart is small and quiet and there is a systolic murmur and thrill in the pulmonary area. All degrees of severity are encountered from those who are only cyanosed with exercise, to others who are deeply and permanently cyanosed in whom the arterial oxygen saturation rarely rises above 60 per cent. The mortality in infancy in this latter group is considerable, but those of less severity may survive into adult life.

All patients require careful investigation with cardiac catheterisation, angiocardiography and dye studies to provide accurate anatomical details of the lesion and indicate the most favourable type of operation.

Fallot's Tetralogy is capable of complete surgical correction and this should be the aim in every case. However, in the very young and in those patients with hypoplastic right ventricular outflow tract some surgeons advise a palliative anastomotic operation designed to increase the pulmonary blood flow, to improve the patient's clinical condition and perhaps help the hypo-

plastic outflow tract to develop. Definitive total correction would then be carried out at a later date.

(i) **Anastomotic Procedures.**—These aim at increasing the blood flow to the lungs by creating a shunt between the aorta or one of its branches and the pulmonary artery. The Blalock operation is the most popular and consists of an anastomosis between the left subclavian artery and the left pulmonary artery.

A posterolateral thoracotomy through the fourth space gives good access. The pulmonary artery is mobilised and doubly clamped as it lies in the lung hilum. The subclavian artery is fully mobilised and divided near the first rib. The proximal end of the subclavian is then anastomosed to the upper border of the pulmonary artery (fig. 805). A continuous thrill can be felt over the anastomosis on releasing the clamps.

Alternative anastomotic procedures are available where a Blalock operation cannot be done or in infants. Descending aorta—left pulmonary artery (Potts), ascending aorta—right pulmonary artery (Waterston–Cooley) or superior vena cava—right pulmonary artery (Glenn) are those most frequently employed.

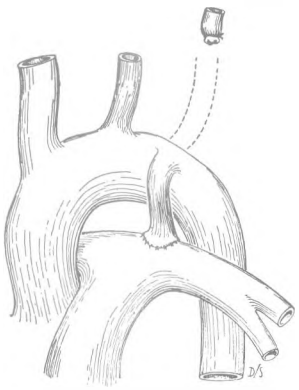


FIG. 805. — Subclavian-pulmonary anastomosis (Blalock) for Fallot's tetralogy.

(ii) **Complete Correction.**—This should be considered in every case but may be deferred in infants and the more severe examples where palliative anastomotic procedures are performed initially.

The operation is carried out under cardio-pulmonary bypass with additional hypothermia. The ventricular septal defect is closed with a Dacron patch. The pulmonary valvar and infundibular obstruction is relieved and an out-flow tract patch of Dacron inserted to enlarge the tract if necessary.

The operative mortality is about 10%. The results of total correction are dramatic and gratifying. Long term follow-up demonstrates continued good health.

II. TRANSPOSITION OF THE GREAT VESSELS

After Fallot's tetralogy this is the next most common cause of cyanotic congenital heart disease and frequently presents with severe symptoms in infancy. The aorta arises from the right ventricle and the pulmonary artery from the left ventricle. The greater and the lesser circulations are thus separated and life is only possible if there is a communication between the two through an atrial or a ventricular defect or a patent ductus arteriosus. Death in the neonatal period is common due to inadequate communications between the circuits.

The child is deeply cyanosed and severely incapacitated. The lungs may be plethoric or oligæmic depending on associated pulmonary stenosis. Diagnosis is confirmed by cardiac catheterisation and angiocardiography.

Management in infancy is aimed at improving the shunting between the circuits by creating or enlarging an atrial defect. This is effected in infants

Willis John Potts, 1895–1968. Surgeon, Children's Memorial Hospital, Chicago, Illinois, U.S.A.
David James Waterston, Contemporary. Surgeon, Hospital for Sick Children, Great Ormond Street, London.
Denton A. Cooley, Contemporary. Professor of Surgery, Baylor University, Houston, Texas, U.S.A.
William W. L. Glenn, Contemporary. Professor of Surgery, Yale University, New Haven, Connecticut, U.S.A.

by rupturing the septum with a balloon catheter passed transvenously through the foramen ovale (Balloon Septostomy: Rashkind). Alternatively, portions of the septum can be excised by the technique of Blalock and Hanlon. If the child survives the first year with or without surgical aid a more radical correction can be contemplated, either by an ingenious transposing of the atria developed by Senning and Mustard, or a re-alignment of the arteries as described by Rastelli.

ACQUIRED HEART DISEASE

Inflammatory and degenerative changes are responsible for the majority of the acquired lesions of surgical interest although cardiac injuries and tumours constitute a small but important group. Rheumatism has a predilection for the mitral and aortic valves, syphilis for the aortic valve and aortic wall, whilst degenerative lesions are frequent in the aortic valve, coronary vessels and myocardium.

Mitral Valve Disease

Mitral valvulitis is the commonest sequel of rheumatic carditis. The original attack of rheumatism may be so mild as to pass unnoticed. Only half the patients with mitral stenosis give a history of rheumatic fever or chorea. The condition affects women more frequently than men, and symptoms are commonest during the third and fourth decades.

Pathology.—The cardiac manifestations of rheumatism are widespread with involvement of all three layers, but it is mainly the mitral and aortic valve involvement which leads to later disturbances. The essential lesion is the Aschoff nodule. These produce swelling and roughening of the valve cusps and fibrin is deposited on the roughened surfaces. Later organisation with fibrosis and calcification may occur. As a result the valve leaflets become fused, thickened, rigid and immobile. All grades are encountered from simple fusion of the commissures and mild fibrosis (but with preservation of mobility) to grossly calcified, fixed and functionless valves. The valve orifice is oval or slit-like with an average opening of 1 cm. in its long axis in those patients submitted for operation.

The mitral valve mechanism may be affected by lesions other than rheumatism. Acute or subacute bacterial infection, ruptured chordæ tendineæ, papillary muscle dysfunction and after myocardial infarction. Most of these conditions result in mitral regurgitation.

Hæmodynamics.—Disease of the leaflets or subvalvar apparatus may result in obstruction to forward flow or the regurgitation of blood from the left ventricle to the left atrium. Often combinations of stenosis and regurgitation are present. In either case there is a reduction of forward flow from the left ventricle and a build up of blood in the lungs.

Clinical Features.—These can be considered briefly under four headings:

(1) Those due to a low cardiac output: tiredness, shortness of breath on exertion, a small volume pulse and pale, cold extremities.

(2) Those due to disturbances of the pulmonary circulation: exertional dyspnœa, pulmonary œdema, nocturnal dyspnœa or asthma, hæmoptysis and bronchitis.

William Jacobson Rashkind, Contemporary. Surgeon, Children's Hospital, Philadelphia, Pa., U.S.A.
Cyril Rollins Hanlon, Contemporary. Professor of Surgery, St. Louis University Hospitals, Missouri, U.S.A.
Åke Senning, Contemporary. Professor of Surgery, Zurich.
William T. Mustard, Contemporary. Associate Professor of Cardiovascular Surgery, Hospital for Sick Children, Toronto, Canada.
Gian Carlo Rastelli, 1934–1970. Consultant in Cardiovascular Surgical Research, Mayo Clinic, Rochester, Minn., U.S.A.
Karl Albert Ludwig Aschoff, 1866–1942. Professor of Pathology, Freiburg, Germany. Described the Aschoff nodule in 1904.

(3) Those due to failure of the right ventricle : congestive failure heart with engorged veins, large liver, ascites and œdema.

(4) Systemic embolism from clot dislodged from the left atrial appendage.

Investigations.—These are required to determine the type and severity of the valve lesion and the presence or not of additional lesions such as other valve lesions or coronary artery disease which may complicate management.

Investigations may include radiology (fig. 806), echocardiography (fig. 797), cardiac catheterisation and angio-cardiography.

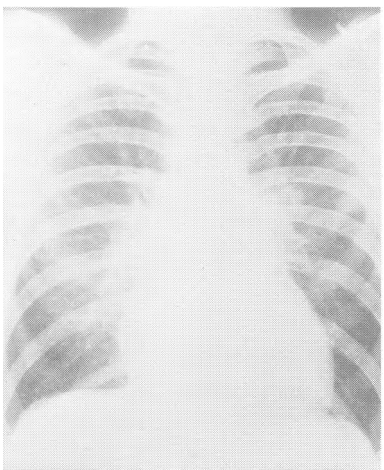


FIG. 806.—Mitral stenosis showing enlargement of the 'pulmonary bay' and the pulmonary arteries.

Indications for Operation.—Operation should be considered in any patient who is being inconvenienced by symptoms which cannot be controlled by medical means. Patients with little disability or few symptoms should be deferred. At the other end of the spectrum, patients in severe congestive heart failure should be seriously considered for operation though at a higher than average risk. Regrettably their powers to recuperate after a successful operation are often limited.

Left ventricular function is probably one of the most important factors in determining risks, whilst in older patients the presence of significant coronary artery disease may affect the outcome.

There are few contraindications to operation in the symptomatic patient. There are certain factors which may cause delay: (1) evidence of active rheumatism such as fever, raised E.S.R., joint pains, high anti-streptolysin titre; (2) any septic focus especially dental should be attended to; (3) all fibrillating patients should be adequately controlled with digitalis; (4) all patients with congestive heart failure should have optimum treatment before surgery; (5) pregnancy is no bar to mitral valve surgery if symptoms are demanding; (6) patients who have sustained a cerebral embolus should wherever possible have cardiac surgery deferred for three months to allow maximum improvement and stability in the cerebral condition.

Mitral Valve Surgery

Closed mitral valvotomy was one of the earliest and most successful of cardiac operations. Good long-term results were obtained in patients with suitable valves, viz. soft, mobile, non-calcified valves without regurgitation. The operation has largely been superseded by open valvotomy though it is still practised in some centres. The advantage of an open procedure is that in addition to separation of the commissures the surgeon can repair the valve,

can separate fused chordæ tendineæ and reduce the circumference of the annulus if this is stretched (annuloplasty).

Careful pre-operative preparation, with a period of rest, fluid and salt restriction, diuretics and breathing exercises is necessary. All cases should be digitalised as there is a 30 per cent risk of atrial fibrillation developing in the post-operative period in cases in normal rhythm. Patients with a history of embolism and those with pulmonary hypertension should be given oral anticoagulants for several weeks before operation. The anticoagulants should be continued through the operation into the postoperative period.

Closed Valvotomy.—A good result from a closed valvotomy can be expected where the valve is mobile and neither thickened nor calcified. Regurgitation should be absent and the patient in sinus rhythm. The operation is carried out through a left antero-lateral thoracotomy. The valve is split with an expanding dilator (Tubbs) inserted through a stab wound in the apex of the left ventricle with the right index finger inserted into the left atrium through its appendage to control the split. The appendage is amputated at the end of the procedure to minimise risk of embolism.

Open Valve Repair (Open Valvotomy).—This is carried out with the aid of cardiopulmonary bypass through a sternal splitting incision. The valve is carefully inspected with the ventricle both relaxed and distended. Fused commissures can be carefully and accurately incised and fused chordæ tendineæ carefully separated. Any tears or irregularities of the leaflet mechanism can be repaired. If the annulus is dilated and producing regurgitation an annuloplasty using two semi-purse-string sutures around the posterio-medial commissure is done. The purse strings are slowly tightened until the regurgitation from the distended ventricle is controlled.

Valve Replacement.—Replacement of the valve is required for a grossly diseased valve or where attempts at open valve repair have failed. The valve is excised and a suitable prosthesis sutured to the annulus with interrupted mattress sutures. The surgeon has a choice of ball valve or flap valve mechanical prosthesis or a tissue valve (porcine heterograft) (fig. 808).

Postoperative Care.—In the immediate postoperative period it is essential to maintain normal blood gases (by ventilation) and normal electrolytes. Potassium is the most important after mitral valve surgery as total body potassium is likely to be low and a low serum potassium may provoke serious rhythm disorders.

Cardiac output is best monitored by observing skin temperature, cerebral function and urinary output in addition to blood pressure and pulse rate. A diminished cardiac output may be improved by reducing peripheral resistance with an infusion of salbutamol (20 mg in 500 ml) or sodium nitroprusside (50 mg in 100 ml), or by infusion of inotropic agents such as isoprenaline (1 mg in 100 ml), adrenalin (1 mg in 100 ml) or dopamine (200 mg in 100 ml). Infusions are given at a rate of 1–5 drops per minute depending on the response.

Rhythm disorders are not infrequent and often difficult to control. The patient should be adequately digitalised and the serum potassium must be within normal limits. If control is still not possible intravenous lignocaine, propanalol (Inderal) or verapamil (Cordilex) should be used but with caution.

All patients with mechanical prostheses should be anticoagulated with Warfarin. Dosage should start as soon as the drainage tubes are removed. A prothrombin ratio of 2:1 is desirable.

Results of Treatment.—Closed mitral valvotomy gives good long term

Oswald Sydney Tubbs, Contemporary. Formerly Cardiothoracic Surgeon, St. Bartholomew's Hospital, London.

results in 75% of patients and with a mortality of approximately 5%. However, not all patients with dominant stenosis are suitable. Open valvotomy provides a better chance of a perfect operation but also a risk of failure demanding valve replacement. Operative risks are similar to those of closed valvotomy. Mitral valve replacement is employed for more severely disabled patients and consequently carries a somewhat higher mortality.

Long Term Results.—Restenosis can occur after both open and closed valvotomy in which case either a second valvotomy or a valve replacement will be required. All mechanical prostheses are potentially thrombogenic and need anticoagulation. This is effected with oral anticoagulants (Dindevan, Warfarin) in doses sufficient to maintain a prothrombin time of twice normal. If anticoagulants cannot be used or prove ineffective drugs to prevent platelet adherence (aspirin, dipyridamole (Persantin)) can be used additionally. In spite of this systemic embolism still occurs in a small number of patients.

All prostheses are foreign bodies in the circulation and are liable to become the focus of infection by bacteria, yeasts or fungi. The infection may seriously damage the prosthesis or its moorings leading to malfunction. Infection

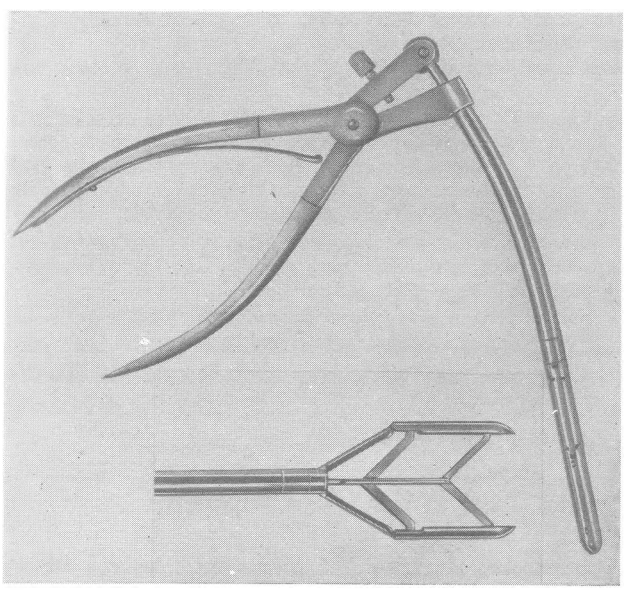


FIG. 807.—Transventricular adjustable mitral valve dilator. (*Tubbs.*)

can sometimes be controlled with suitable antibiotics though often this can only be achieved by excising the prosthesis, sterilising the tissues and replacing with a new valve.

Valve Prosthesis.—The perfect artificial heart valve has yet to be designed. The surgeon today has the choice of three basic types—ball valve, disc or hinge valve and tissue valve.

The ball valve (fig. 808(*a*)) is simple in construction and with a simple mechanism

unlikely to go wrong. It is thrombogenic and the poppet is partially obstructing the forward flow. The disc valves (fig. 808(*c*)) are less obstructive but more thrombogenic than the ball valve and the mechanism is more complicated and therefore more liable to develop malfunction. Damage to the red cells with hæmolysis and anæmia can occur with both types of mechanical valve. Tissue valves (fig. 808(*b*)) have the best flow patterns and are least thrombogenic but their durability remains suspect. The present popular glutaraldehyde-treated porcine heterograft would appear to have a life of at least 7–10 years.

Aortic Valve Disease

The aortic valve may be affected by developmental, inflammatory or degenerative processes, resulting in stenosis, incompetence or combined lesions.

Aortic Stenosis.—The obstruction impedes the forward flow of blood from the left ventricle producing angina, syncope and effort dyspnœa. The left ventricle is hypertrophied and a coarse systolic murmur and thrill are discernible in the aortic area. The arterial pulse is slow-rising and sustained (anacrotic or plateau pulse) and the pulse pressure is small.

The lesion is serious; children are liable to sudden death and adults with symptoms have a life expectancy of only a few years.

Surgery, with the aid of extracorporeal circulation, plays an important role in the management of the condition. *Open valvotomy* with division of fused commissures is often possible in children. *Valve replacement* will be required for grossly destroyed or heavily calcified valves.

Aortic Regurgitation.—A leaking aortic valve produces considerable left ventricular dilatation in addition to hypertrophy. Symptoms are similar to those of aortic stenosis. In addition to an aortic systolic murmur, a diastolic murmur is audible along the left sternal edge. The pulse is characteristically quick rising (Corrigan or water-hammer) with a high systolic and low diastolic pressure resulting in a large pulse pressure.

Isolated cases are capable of correction by suture (tears) or patching (perforations), but the majority are best treated by excision and replacement.

Aortic Valve Replacement.—This is carried out with the aid of heart lung bypass through a sternal splitting incision. The valve is exposed with an incision in the aortic root after cross-clamping the aorta.

During the operation, which may take an hour or more, steps must be taken to protect the myocardium from deprivation of blood and oxygen. This can be achieved by the cannulation and perfusion of each coronary with blood from the heart-lung machine. Alternatively a solution of cardioplegic and protective drugs at a temperature of 4°C can be injected into the coronary vessels. This cools and paralyses the heart and allows the surgeon approximately 45 minutes of safe myocardial ischæmia without perfusion of the coronaries.

The valve is carefully excised and calcium dissected from the valve ring. The prosthesis is sutured to the valve ring with interrupted mattress sutures. The surgeon has a choice of mechanical valves—a caged ball valve (fig. 808(*a*)) or tilting disc valve (fig. 808(*c*)), a free homograft (Ross and Barratt-Boyes) or mounted heterograft (fig. 808(*b*)).

Sir Dominic John Corrigan, 1802–1880. Physician, Jervis Street Hospital, Dublin. Described the water-hammer pulse of aortic incompetence in 1832.
Donald Nixon Ross, Contemporary. Cardiac Surgeon, National Heart Hospital, London.
Sir Brian Barratt-Boyes, Contemporary. Cardiac Surgeon, Auckland, New Zealand.

Results of Aortic Valve Replacement.—Early mortality is approximately 5%. The later mortality and morbidity is determined by valve dysfunction, thrombo-embolism, hæmolysis or infection. Associated lesions, especially

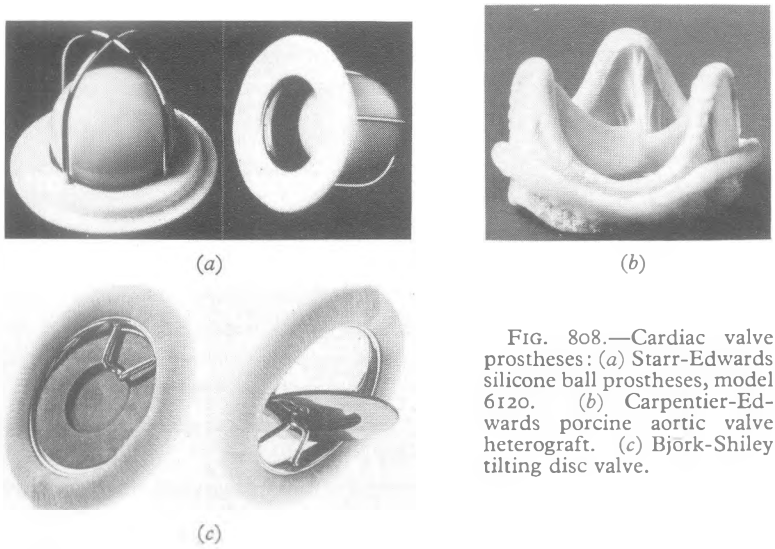


FIG. 808.—Cardiac valve prostheses: (*a*) Starr-Edwards silicone ball prostheses, model 6120. (*b*) Carpentier-Edwards porcine aortic valve heterograft. (*c*) Björk-Shiley tilting disc valve.

coronary artery disease, are important causes of later mortality. Late deaths at 5 years are approximately 10%. The clinical results are very satisfactory.

Cardiac Tumours

Tumours of the heart are relatively uncommon; they may occur in the ventricle or atrium. The commonest is the myxoma of the atrium (80 per cent of all tumours) and of these the majority (75 per cent) occur in the left atrium.

Left atrial myxomas simulate mitral stenosis; they obstruct the flow of blood through the left atrium producing pulmonary venous engorgement and hypertension. Systemic embolism and postural syncope are important symptoms.

The condition simulates mitral stenosis but is distinguished from it by a shorter and more progressive history, evidence of toxicity (fever, raised E.S.R., anæmia) and disturbances of plasma proteins and by ultra-sound (echocardiography, p. 763).

The lesion can be demonstrated by angiography, and can be completely removed with the aid of an extracorporeal circulation.

Disorders of Rhythm

Heart block or complete atrio-ventricular dissociation may occur as a manifestation of certain congenital cardiac lesions, as a complication of ischæmic heart disease or the result of surgical treatment of ventricular or allied defects. The commonest cause remains degeneration of the conduction tissue in old age. In all cases, the atrial impulse fails to stimulate the ventricle owing to interference with the conduction of the bundle of His. The ventricles beat independently at a rate of 25 to 50 per minute.

The dangers of this situation are three-fold:

(*a*) The slow rate impairs cardiac output and may lead to heart failure.

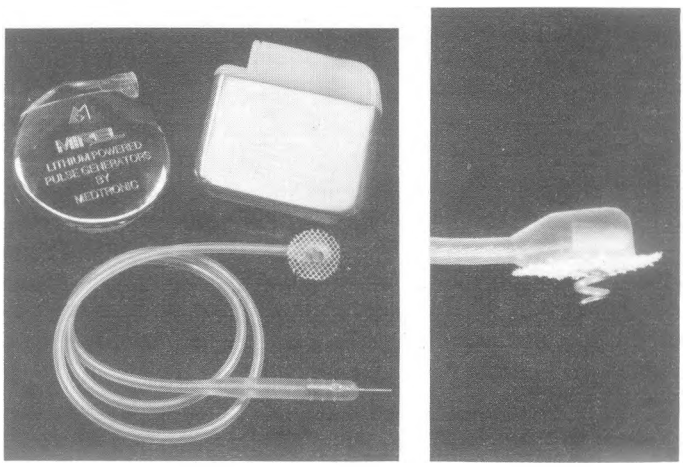
(*b*) A sudden slowing or cessation of the beat may produce syncope (Stokes-Adams attack), or death.

Albert Starr, Contemporary. Professor of Surgery, University of Oregon, Portland, Oregon, U.S.A.
Alain Carpentier, Contemporary. Cardiac Surgeon, Hôpital Broussais, Paris.
Viking Olaf Björk, Contemporary. Cardiac Surgeon, Karolinska Sjukhuset, Stockholm, Sweden.
Wilhelm His (Junior), 1863–1934. Professor of Medicine, Berlin. Described the atrio-ventricular bundle in 1893.
William Stokes, 1804–1878. Regius Professor of Medicine, Dublin. Described this condition in 1846, and drew attention to the cases reported by Adams in 1827.
Robert Adams, 1791–1875. Regius Professor of Surgery, Dublin.

(*c*) A sudden change from sinus rhythm to heart block may produce syncope.

Although the ventricular rate can sometimes be increased by sympathetic-otonic drugs, of which ephedrine and isoprenaline are the most effective, the response to both drugs becomes less with continued use.

Artificial electrical pacemakers have been designed to stimulate the heart at appropriate rates and may be of either the *fixed rate* or *demand* types. The stimulating electrode is applied to the ventricle from the *endocardial* or



(*a*) (*b*)

FIG. 809.—Implantable pacemakers: (*a*) Two varieties (Medtronic and Telectronic) of lithium-powered generators and pacemaker lead. These units should operate for at least 7 years. (*b*) Close-up view of the pacemaker electrode which is inserted into the myocardium.

epicardial aspect. With the introduction of lithium-powered generators, pacemaker systems (fig. 809) should now last at least seven years before requiring replacement.

Ischæmic Heart Disease

Atherosclerosis of the coronary arteries occurs with increasing frequency in Western civilisation. Between 1951 and 1975 the proportion of deaths in England and Wales due to ischæmic heart disease rose from 10·5% to 26·0% of the total.

Narrowing of the coronary arteries deprives the myocardium of oxygen. When severe enough, chest pain is produced (angina = claudication). Total occlusion results in death of myocardial tissue (infarction = necrosis). Angina thus indicates a serious deprivation of myocardial blood supply which may lead to infarction and death if allowed to progress.

Surgery has a role to play both in the relief of angina by revascularisation of the myocardium and in the management of the complications of infarction.

The natural history of angina pectoris depends primarily on the extent of the coronary arterial disease and the functional state of the left ventricle.

Full investigations including coronary arteriography and left ventriculography are therefore necessary before surgery can be considered. Patients with relatively mild arterial disease and angina that can be readily controlled by β blockers should be treated medically. The remainder should be considered for surgical treatment.

Aorto-Coronary Bypass Graft.—Here a length of autogenous saphenous vein is used to circumvent a narrowed coronary artery. Proximally it is attached to the ascending aorta and distally to the coronary artery beyond the occlusion (fig. 810). Up to five grafts may be required to nourish all ischæmic areas. Angiographic criteria for bypass grafting include significant proximal stenoses of vessels with good distal lumens supplying a viable segment of myocardium with an adequate vascular runoff. The risk of such operations depends almost entirely upon the left ventricular function. Where this is good the mortality rate is 3–5%. Most patients gain relief from their angina and there is increasing evidence that prognosis is improved in those with triple vessel disease. Most grafts that occlude do so early and graft patency in most series is about 80% at four years after operation.

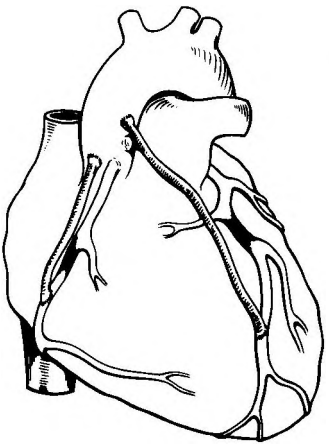


FIG. 810.—Diagrammatic representation of double coronary vein graft to obstructed right coronary artery and left anterior descending.

Left Ventricular Aneurysm.—Myocardial infarction results in death of muscle. Healing is by scar tissue. The scar may stretch and form an aneurysm. The presence of an aneurysm adversely effects the function of the heart and results in heart failure. Fibrin is deposited on the walls of the aneurysm and may be dislodged as an embolus. The aneurysm should be resected under cardiopulmonary bypass and the left ventricle reconstituted by suturing the edges of the aneurysm together.

The operative risks are approximately 10% and prospects of long term survival are quite good.

Other Complications of Infarction.—The interventricular septum or the papillary muscles supporting the tricuspid or mitral valves may be damaged after infarction. Rupture of the septum (acquired VSD) and papillary muscle dysfunction of the mitral valve may result. Either lesion seriously affects cardiac function but both can be corrected surgically.

Support of the circulation by means of intra-aortic balloon pump counterpulsation may be useful before and after the operation.

AORTIC ANEURYSMS

The surgery of the aorta has made considerable strides since the introduction of aortic grafts and the advent of hypothermia and methods of cardiopulmonary by-pass. It is now possible to occlude the aorta with safety for

reasonable periods, and to bridge considerable defects, so that the two chief deterrents to reconstructive aortic surgery have largely disappeared.

Aortic aneurysms due to syphilis or degenerative changes may be fusiform, saccular (fig. 811) or dissecting. The diagnosis may be straightforward, but is often notoriously difficult, although angiocardiography has provided a useful diagnostic procedure.

FIG. 811.—Saccular aneurysm of the ascending aorta.

Treatment of Aneurysms.—Insertion of Colt's umbrella and Blakemore's wire and wrapping of aneurysms in polythene has been entirely superseded as methods of treatment by more direct procedures made possible by the use of heart-lung by-pass or left atrio-femoral by-pass. (p. 768).

(1) *Excision and Repair. Aneurysmorrhaphy* (*Matas*).—Applicable to smaller saccular aneurysms. Consists of excision of the sac with direct suture of the neck. Not applicable to fusiform aneurysm or saccular aneurysms with a wide neck (fig. 812 and also fig. 143).

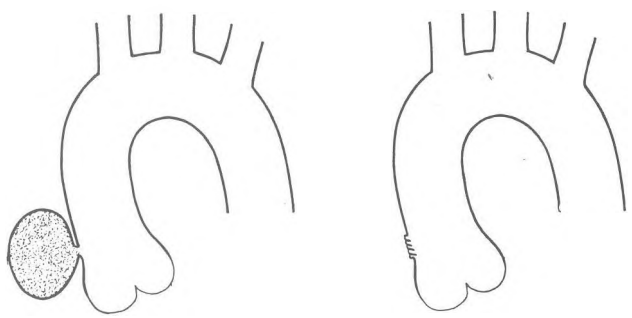


FIG. 812. — Saccular aneurysm. Excision and repair.

(2) *Excision and Grafting*.—This involves excision of the aneurysm and adjacent aorta and its replacement with a prosthesis (woven, crimped Dacron) (fig. 813). Heart-lung by-pass is required for aneurysms of the

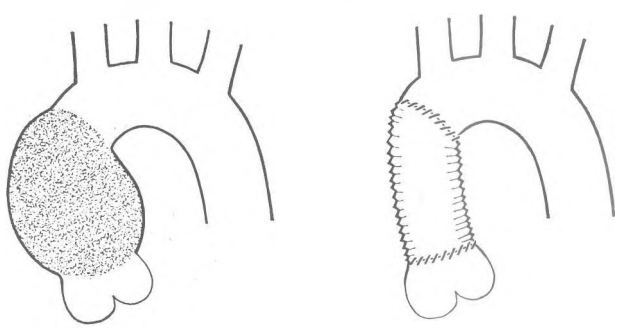


FIG. 813. — Fusiform aneurysm of the ascending aorta. Excision and grafting.

George Herbert Colt, 1878–1957. Surgeon, Royal Infirmary, Aberdeen, Scotland.
Arthur Hendley Blakemore, Contemporary. Associate Professor of Surgery, Columbia University College of Physicians and Surgeons, New York, U.S.A.
Rudolph Matas, 1860–1957. Professor of Surgery, Tulane University, New Orleans, Louisiana, U.S.A. *(see footnote, p. 149).*

ascending aorta or aortic arch, but the simpler left atrio-femoral by-pass or heparinised vascular shunt (p. 768) is all that is required for aneurysms of the descending thoracic aorta.

(3) *Excision and Plastic Repair.*—The aneurysm is excised and the aortic defect is repaired by an insert of woven Dacron (fig. 814). This method is

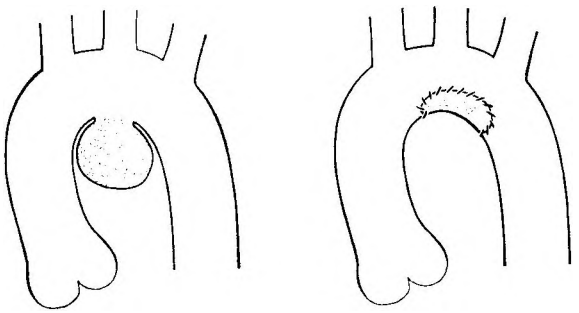


FIG. 814. — Aneurysm of the aortic arch. Localised excision and plastic repair.

applicable to saccular aneurysms of the aortic arch and avoids the elaborate anastomoses required for replacement of the arch.

Dissecting Aneurysms.—These are produced by a split in the intima and internal portion of the media, allowing blood to enter the media and extend both distally and proximally resulting in a double lumen aorta. The aneurysms may rupture externally or into the pericardium with fatal results, may undermine and dislodge the attachments of the aortic valve, producing aortic incompetence or occlude vital branches of the aorta (carotid, renal, etc.) with serious consequences. Dissection may be related to systemic hypertension or defective aortic wall as in Marfan's disease.

The lesion is a serious one with a high mortality within a few days of onset, but a minority survive for some years. In the early stages dissection may be limited by the use of hypotensive drugs. Operative treatment with excision of the point of entry and re-anastomosis offers some hope of limiting further dissection by allowing blood to clot in the outer lumen (fig. 815). Some form of by-pass circulation is necessary.

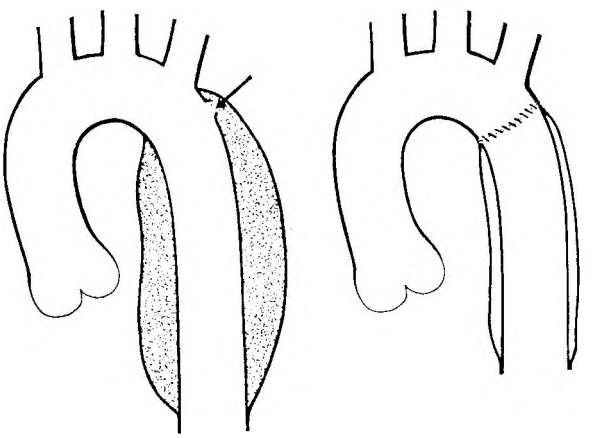


FIG. 815.—Dissecting aneurysm of the descending aorta. Excision of area of rupture and reconstitution of the aorta. The arrow indicates the site of the intimal tear.

Bernard Jean Antonin Marfan, 1858–1942. (*See footnote, p. 282.*)

Vascular Rings (Dysphagia Lusoria[1]).—Abnormal development of the aortic arch and its branches (e.g. double aortic arch, right subclavian arising from descending aorta) may result in obstruction of the trachea and œsophagus by vessels which surround them both in front and behind. The anomaly may pass unnoticed, but occasionally in children severe dyspnœa, stridor and dysphagia may develop and require urgent treatment. Occasionally symptoms only occur later in life due to enlargement of the vessels.

A barium swallow reveals a posterior indentation of the œsophagus by the abnormal vessel. If symptoms are severe, thoracotomy should be performed and the abnormal vessel divided or transposed.

CARDIOTHORACIC TRAUMA

Road accidents and other high-speed injuries may cause acute traumatic rupture of the aorta. This usually occurs between the left subclavian artery and the ligamentum arteriosum. Aortic rupture should always be suspected in deceleration injuries where there is widening of the mediastinum and unexplained hypotension. The diagnosis is confirmed by aortography. The natural history of traumatic rupture of the aorta is poor and emergency operation is indicated for either repair or replacement with a Dacron graft. These patients often have other extensive injuries and the recent introduction of heparinised vascular shunts (p. 768), thereby avoiding systemic heparinisation during the period of aortic clamping, has been an important advance.

Stab wounds of the chest may penetrate the lungs or heart. After initial clinical assessment a chest radiograph should be taken and the patient carefully observed. A pneumothorax or hæmothorax should be treated by intercostal tube drainage. Most stab wounds of the heart present with signs of hæmorrhage or cardiac tamponade but providing the cardiovascular function remains stable it is usually reasonable to defer exploratory operation.

CARDIAC TRANSPLANTATION

The first human heart transplant was performed by Christiaan Barnard in 1967. During the last decade considerable advances have been made by Dr. Shumway's group at Stanford University and a patient undergoing transplantation at that institution can now anticipate 67% and 50% chances of being alive after one and four years respectively.

The main reasons for this improved outlook include better selection of recipients, earlier detection and better management of acute rejection episodes, fewer postoperative infections, and a declining incidence of coronary atheroma in the transplanted heart.

Patients selected for transplantation should have terminal heart disease not amenable to further medical or conventional surgical treatment. They should preferably be less than 50 years of age, should have no active infection, and the pulmonary vascular resistance should not be unduly elevated. There

[1] Lusus = game, sport or jest. Condition originally described as ' Lusus naturæ '—a jest or sport of nature. First noted by Bayford in 1761.

David Bayford, 1739–90. Surgeon and Physician, Lewes, Sussex. He was born probably in the village where Mr. McNeill Love lived in his later years. He was granted the degree M.D. by the Archbishop of Canterbury, a rare means of obtaining a medical degree which still exists today.

Christiaan Barnard, Contemporary. Professor of Cardiac Surgery, Groote Schuur Hospital, Cape Town, South Africa.

Norman Shumway, Contemporary. Professor of Surgery, Stanford University, California, U.S.A.

should be ABO blood group compatibility between donor and recipient and a negative lymphocyte crossmatch between donor cells and recipient serum. The degree of HLA tissue typing mismatch does not seem to affect the prognosis. Permanent immunosuppression with corticosteroids and azathioprine is necessary and most late complications are related to this. The advent of a new effective and non-toxic immunosuppressive agent would further expand the indications and reduce the cost of the procedure.

CHAPTER 42

THE ŒSOPHAGUS

Surgical Anatomy.—The œsophagus is a fibromuscular tube 25 cm long occupying the posterior mediastinum and extending from the cricopharyngeal sphincter to the cardia of the stomach; 4 cm of this tube lies below the diaphragm. The musculature of the upper one-third is mainly striated, giving way to smooth muscle below. It is lined by squamous epithelium which is replaced by specialised epithelium at the level of the hiatus similar to gastric mucosa but without oxyntic and peptic cells (fig. 816). This specialised mucosa lines the lower 3 cm. The parasympathetic nerve supply is mediated by the vagus through an extrinsic and intrinsic plexus. The intrinsic plexus has no Meissner's network which is present elsewhere through the alimentary canal, and Auerbach's plexus is present only in the lower two-thirds.

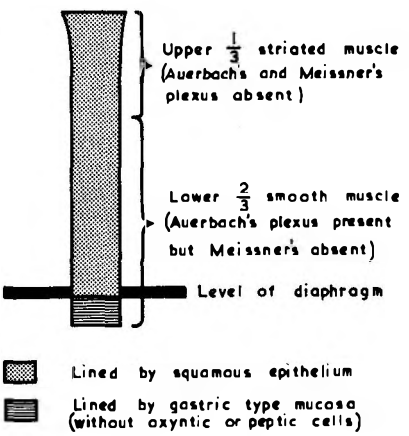
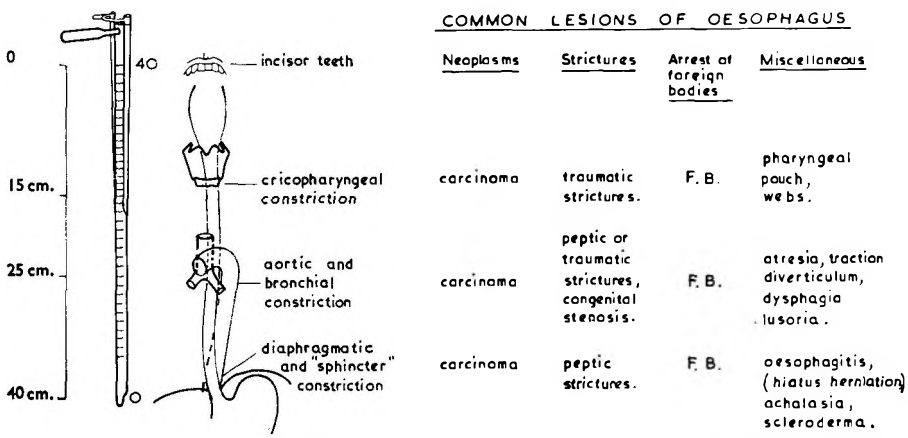

Upper 1/3 striated muscle
(Auerbach's and Meissner's plexus absent)
Lower 2/3 smooth muscle
(Auerbach's plexus present but Meissner's absent)
Level of diaphragm
Lined by squamous epithelium
Lined by gastric type mucosa (without oxyntic or peptic cells)

FIG. 816.—The anatomical structure of the œsophagus.

There are three physiological constrictions in this tube with distinct lesions at each level as shown in fig. 817. The figures 15, 25 and 40 are very important to the œsophagoscopist. They represent the situations of anatomical narrowing where difficulty may be experienced in the passage of instruments and where foreign bodies may be arrested. They are also the sites of election for innocent strictures and for carcinoma of the œsophagus.



COMMON LESIONS OF OESOPHAGUS

	Neoplasms	Strictures	Arrest of foreign bodies	Miscellaneous
cricopharyngeal constriction	carcinoma	traumatic strictures.	F. B.	pharyngeal pouch, webs.
aortic and bronchial constriction	carcinoma	peptic or traumatic strictures, congenital stenosis.	F. B.	atresia, traction diverticulum, dysphagia lusoria.
diaphragmatic and "sphincter" constriction	carcinoma	peptic strictures.	F. B.	oesophagitis, (hiatus herniation) achalasia, scleroderma.

FIG. 817.—Common lesions at various levels of the œsophagus. An œsophagoscope is drawn to the left of the diagram. (*L. R. Celestin, F.R.C.S., Bristol.*)

Physiology.—The main function of the œsophagus is to form part of a co-ordinated mechanism transferring food from the mouth to the stomach. The initial

Georg Meissner, 1829–1905. Professor of Physiology, Göttingen, Germany.
Leopold Auerbach, 1828–1897. Professor of Neuropathology, Breslau, Germany. (Now Wroclaw, Poland.)

movement of food through the oropharynx is induced voluntarily and involves sequential contraction of the oropharyngeal musculature together with simultaneous closure of the nasal and respiratory passages and opening of the upper œsophageal or cricopharyngeal sphincter. The body of the œsophagus then sweeps the food bolus by an involuntary peristaltic wave, through a relaxed gastro-œsophageal sphincter zone, into the stomach.

The cricopharyngeal sphincter is normally closed at rest and serves as a protective mechanism against regurgitation of œsophageal contents into the respiratory passages. Failure of it to relax on swallowing may pre-dispose to the development of a pharyngeal pouch (propulsion diverticulum, p. 692).

At the lower end of the oesophagus there is a physiological sphincter which together with other anatomical mechanisms (p. 796) prevent materials refluxing along the natural pressure gradient between the abdominal and thoracic cavities. The tone of the lower œsophageal sphincter is influenced by gastrointestinal hormones, anticholinergic drugs and smoking.

INVESTIGATION OF THE DISEASES OF THE ŒSOPHAGUS

The œsophagus being inaccessible by palpation the history is of special importance but the physical signs are of lesser value.

Dysphagia is the term used to describe difficulty (not pain) in swallowing; there are two types—oropharyngeal and œsophageal. The type of dysphagia is important. It may be dysphagia for solids or fluids, intermittent or progressive, precise or vague in its appreciation. *Pain* may be present. Painful dysphagia is usually due to œsophagitis. *Regurgitation*—it is important to record the volume, contents, presence of blood or bile and the reaction to litmus. Loss of weight, anæmia, cachexia and change of voice are also important symptoms.

Radiography is a most valuable investigation. A *straight film* will show an opaque foreign body and the site of its arrest (figs. 821, 822). A *barium swallow* is essential: screening will show the motility and films, taken as necessary, will delineate size, distortion or the presence of a space-occupying lesion. It is important to take a film with the patient in the prone head-down position to assess the degree of regurgitation.

Œsophagoscopy is essential. Endotracheal anæsthesia with a short-acting relaxant permits of easy access. The key to safety lies in the correct position of the patient on the table. A sandbag is placed behind the third dorsal vertebra in the supine position. An assistant holds the head and controls its position during the various manœuvres of the tube (fig. 818). Every stage must be accomplished under *direct vision*—at no time must the instrument be passed blindly (see Perforation, p. 793).

Adequate suction and a good light are very important.

Fibre-optic œsophagoscopy—is simpler and hence safer. It has disadvantages however, in that the view (end-on) is limited, biopsies are smaller and the 'feel' where a tumour is present is less informative.

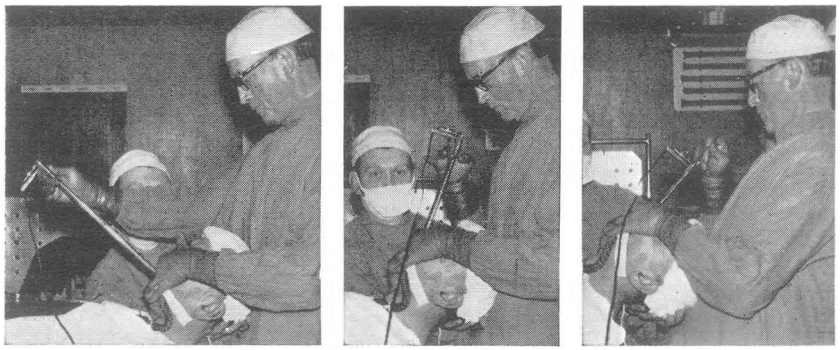
Motility Studies.—Since the work of Code these have come to play an important part in diagnosis. A unit made up of three tubes with open tips or small water-filled balloons is swallowed by the patient. The tubes (or balloons) are spaced at 5 cm apart and each is attached to a pressure transducer. In this way it is possible to record pressure changes in the viscus during swallowing and to see if peristalsis is propagated normally. It has perhaps its greatest use in the diagnosis of diffuse spasm (presbyœsophagus, fig. 844) scleroderma (p. 804) or the lesser degrees of hiatal hernia (Earlam).

CONGENITAL ABNORMALITIES

Well-known abnormalities are:

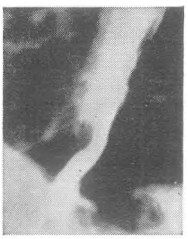
1. Atresia with or without tracheo-œsophageal fistula.
2. Stenosis.
3. 'Short' œsophagus with hiatus hernia.
4. Dysphagia lusoria.

Charles F. Code, Contemporary. Professor of Physiology, The Mayo Clinic, Rochester, Minnesota, U.S.A.
Richard John Earlam, Contemporary. Surgeon, The London Hospital, London.



(*a*) (*b*) (*c*)

(*d*)



(*e*)

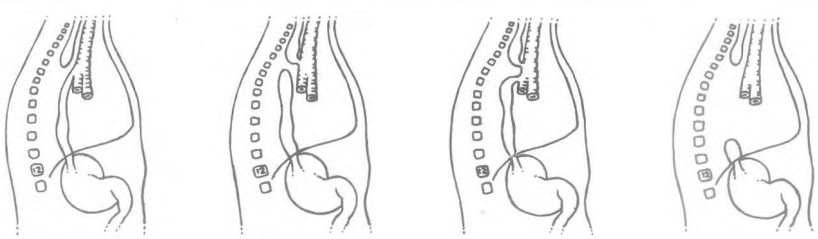
FIG. 818.—Œsophagoscopy. (*a*) With the patient's shoulders slightly raised and neck fully flexed, the œsophagoscope is guided by the endoscopist's left hand over the dorsum of the tongue to the posterior pharyngeal wall. (*b*) The head is slightly extended and the instrument has been passed under vision behind the endotracheal tube. The tip of the instrument is now behind the cricoid cartilage and on levering the larynx forwards the crico-pharyngeal fold is seen. (*c*) As the head and neck are gradually extended the instrument is advanced under vision. The mid-thoracic œsophagus is now being inspected. (*d*) The head and neck are now completely extended and the œsophagoscope has advanced until the 40-cm mark is opposite the upper incisor teeth. At this level the cardia normally comes into view but in this case the œsophageal carcinoma (*e*) is seen.

(*Norman C. Tanner, F.R.C.S., London.*)

Congenital atresia of the œsophagus is usually associated with a tracheo-œsophageal fistula. Referring to fig. 819, it will be seen that in 85 per cent. of cases it is the *lower* pouch that communicates with the trachea.

It is highly important to be cognisant of this abnormality, because its recognition within forty-eight hours of birth, and subsequent surgical correction, is the only hope of survival.

Clinical Features.—The new-born babe regurgitates all its first, and every feed. Saliva pours, almost continuously, from its mouth. This is



(*a*) 85 per cent. (*b*) 2 per cent. (*c*) 1 per cent. (*d*) 12 per cent.

FIG. 819.—Congenital œsophageal atresia. (*a*) Lower pouch opening into the trachea. (*b*) Upper pouch opening into the trachea. (*c*) Both pouches opening into the trachea. (*d*) Both pouches ending blindly. Usually the mid-œsophagus is missing entirely.

the sign of œsophageal atresia—to no other condition does this phenomenon appertain. Attacks of coughing[1] and cyanosis are liable to occur on feeding. The abdomen becomes distended (dilatation of the stomach), due to air swallowed from the tracheal fistula (fig. 819 (*a*)).

Clinical Confirmation of the Diagnosis.—A No. 10 soft rubber catheter is introduced into the œsophagus through the nose. Should an obstruction be encountered about 10 cm from the nostril, the diagnosis is practically certain

Radiological Confirmation.—On no account should barium emulsion be given in these cases. Injection of 1 ml. of lipiodol down the catheter will demonstrate the obstruction. During this examination the supine position is advised, because in the rare event of the atresia belonging to categories (*b*) or (*c*), the medium is less likely to enter the trachea. In all cases the lipiodol should be aspirated after the radiograph has been taken. Another radiographic finding of significance is air in the stomach and jejunum, which is indicative of a communication between the lower pouch and the trachea.

Pre-operative Treatment.—Surgical intervention is urgent. Aspiration pneumonia is nearly always present and penicillin should be given. Everything by mouth is forbidden.

Operation.—The best approach is through a right-sided thoracotomy incision at the level of the fifth intercostal space (fig. 820). The azygos vein having been divided between ligatures, the upper segment of the œsophagus is located by a catheter within it, and freed gently from the surrounding structures. Having been separated from the trachea, the lower segment is transected just sufficiently far from the abnormal communication to permit the opening in the trachea to be closed. An opening is made into the blind upper segment and anastomosis carried out between this opening and the lower segment.

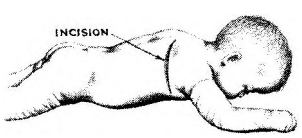


FIG. 820. — Thoracotomy for congenital œsophageal atresia.

Post-operative Care.—A special nurse skilled in aspirating the pharynx, and an oxygen tent-incubator are highly desirable.

Prognosis.—Aspiration pneumonia with acid digestion of the lungs and infection superadded is exceedingly likely to supervene when there has been delay in making the diagnosis. Added to this there is a danger of post-operative leakage from the anastomosis into the pleural cavity, a disaster that is minimised by freeing the distal segment for a considerable distance and thus decreasing tension. These dual complications result in a mortality approximating 40 per cent.

Œsophageal Stenosis.—This is a congenital organic narrowing of the lumen of the œsophagus and is a cause of dysphagia. Commonly it occurs at the same site as atresia and is treated by dilatation. Those in the lower one-third are usually not congenital but due to such conditions as œsophagitis, ulceration or achalasia.

Congenital Short Œsophagus (p. 796).

Dysphagia Lusoria or **Œsophageal Compression by an Abnormal Artery** is discussed on p. 787.

FOREIGN BODIES IN THE ŒSOPHAGUS

All manner of swallowed foreign bodies have become arrested in the œsophagus: coins, pins, and dentures (fig. 821) head the list. When the object contains radio-opaque material, an urgent X-ray examination is called for. The presence of a non-opaque foreign body in the œsophagus can often be confirmed by a barium swallow (fig. 822). Whenever possible the patient should be screened before œsophagoscopy is undertaken.

[1] In type (*a*) the patient coughs up frothy mucus, perhaps tinged with bile.

The foreign body having been visualised (fig. 823), and, if necessary, manipulated into a favourable position, it is grasped with suitable forceps introduced through the œsophagoscope. The œsophagoscope, together with the forceps still grasping the foreign body, is then gently withdrawn.

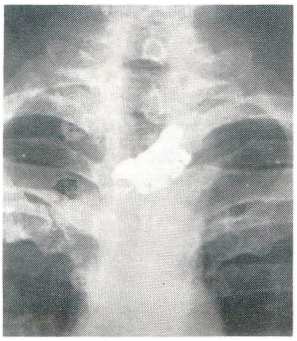


FIG. 821.—False teeth impacted in œsophagus. (*The late Dr. James F. Brailsford, Birmingham.*)

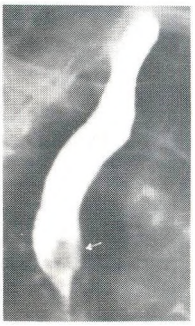


FIG. 822.—Damson-stone outlined by barium, indicated by the arrow.

FIG. 823.—An 'old' penny in the œsophagus as seen through an œsophagoscope.

INJURIES

Perforation of the œsophagus can result from the inexpert use of an œsophagoscope or a gastroscope; usually the beak of the instrument is thrust through the thin posterior wall of the pharynx just above the cricopharyngeal sphincter or the wall is crushed between the shaft of the instrument and a rigid, osteoarthritic cervical spine. Perforation just above an œsophageal stricture can occur from œsophagoscopy or bouginage. It also can result from removal of a piece of growth for biopsy, but probably the most frequent cause of perforation is a sharp foreign body plus instrumentation to remove it.

Often the perforation goes unrecognised by the œsophagoscopist until general distress and great pain on swallowing saliva or fluid, and dyspnœa, become apparent. In such circumstances the possibility of a tear should be considered at once. An X-ray is essential; this discloses the presence of air in the mediastinum, the pleural cavity, or in the neck which may be palpable (surgical emphysema). In doubtful cases the radiograph should be repeated after the patient has swallowed a small quantity of lipiodol. When the wall of the œsophagus has been perforated, urgent operation under antibiotic cover is indicated. When the cervical œsophagus has been ruptured, an incision along the anterior border of the sternomastoid (usually the left), with lateral retraction of the carotid sheath, gives access to the site of perforation (fig. 824), which is closed in two layers. If the mid or lower œsophagus is involved, thoracotomy must be performed. In non-malignant cases timely suture is usually followed by recovery.

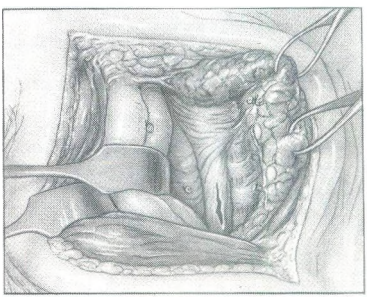


FIG. 824.—Exposure of a tear in the cervical œsophagus. In this case there was surgical emphysema on the right side of the neck.

When the symptoms are delayed, and radiography is negative, the perforation is in all probability a very small one. In this case massive antibiotic treatment with parenteral feeding is usually successful.

Burns and scalds of the œsophagus from swallowing corrosive fluid, if not rapidly fatal, are often the precursor of a dense stricture. The surgeon's aim should

be to minimise the contracture of the inevitable scar and this is best achieved by the use of hydrocortisone and antibiotics. As soon as the patient's general condition permits, gastrostomy is performed. About a week later it is advisable to ascertain the extent of the damage by œsophagoscopy performed with great gentleness.

It is wise to prohibit bouginage for at least three weeks. More than 70 per cent. are controlled by dilatation. The remainder should have resection of the stricture and colon replacement if necessary. The enormous improvement in the outcome of burns and scalds of the œsophagus is due chiefly to the introduction of antibiotics, cortisone, and to the development of œsophageal surgery.

SPONTANEOUS RUPTURE OF THE ŒSOPHAGUS

Ætiology.—Instead of the cricopharyngeus relaxing, as is usual during vomiting, it contracts. The pressure within the œsophagus rises so steeply that the organ bursts at its weakest point. The condition is similar to the linear tears of the cardia in the Mallory-Weiss syndrome (p. 837).

Pathology.—The necropsy findings[1] are remarkably constant. There is a longitudinal tear 1 to 4 cm. in length in the posterior wall of the extreme lower end of the œsophagus, nearly always on the left side. Following perforation, the mediastinum becomes filled with air and gastric contents. Usually about six hours later the pleural membrane gives way, and air and gastric contents gush into the pleural cavity.

Clinical features are also remarkably constant. Following a meal, vomiting occurs. During vomiting, agonising pain is experienced in the thorax, followed by dyspnœa. Board-like rigidity similar to that accompanying perforation of a gastric or duodenal ulcer is often present. Soon surgical emphysema appears in the suprasternal notch; it spreads up and around the neck. Crepitus is often detected here within one hour after rupture. Hyper-resonance and absence of breath-sounds over one lung indicate a pneumothorax. The pain is so intense that even i.v. morphine fails to relieve it.

Radiography.—In very early cases no abnormality is demonstrated; after a few hours mediastinal emphysema is seen; in later cases a hydro-pneumothorax is present.

Paracentesis Thoracis.—Several hours after the perforation, when the extravasated fluid in the mediastinum has ruptured into the pleural cavity, if a hollow needle is inserted into the chest, air and liquid vomitus, acid in reaction, are withdrawn.

Treatment.—Recovery has followed immediate thoracotomy, opening widely the mediastinum, suture of the perforation, and closure of the thorax with drainage. Supportive fluid therapy must be given before, during, and after the operation.

ŒSOPHAGEAL DIVERTICULUM

Pulsion diverticulum is rare, and occurs where a blood vessel enters the œsophagus. The majority are associated with neuro-muscular incoordination which is the cause of occasional dysphagia (fig. 844).

ŒSOPHAGEAL VARICES (see Chap. 45)

PARALYSIS

The passage of food along the gullet is dependent entirely upon involuntary muscular peristalsis. When the neuromuscular mechanism of deglutition is paralysed, as occurs occasionally, notably as a complication of tetanus or diphtheria, ingested material is regurgitated. In established cases the difficulty must be overcome by feeding through a stomach tube.

ŒSOPHAGITIS

This may be *acute*—following burns or scalds, infective (spreading from the pharynx), or peptic, sometimes from the trauma of an indwelling stomach tube; or *chronic*—the most important cause is reflux, either of acid or

[1] Spontaneous rupture of the œsophagus has been found at necropsy in cases of severe head injury and other cerebral lesions, doubtless due to vomiting and retching.

alkaline juices; the latter following œsophago-duodenostomy or œsophago-jejunostomy without a Roux-en-Y loop.

Ætio-Pathology.—The changes are maximal at the lower end of the œsophagus. Essentially there is loss of epithelium with replacement by bleeding granulation tissue. Although the symptoms are experienced by day the damage is done by night when the recumbent position allows the peptic juices to flow into the œsophagus. As soon as inflammation of the epithelium occurs, there is vagal hyperactivity so that the longitudinal muscle contracts and draws the cardia more and more up into the thorax and also hyperacidity is increased. Thus a vicious circle of œsophagitis—longitudinal muscle spasm—upward displacement of the cardia—increased regurgitation ensues. It is likely that the reflux œsophagitis so often encountered during pregnancy is due to a temporary sliding hiatus hernia resulting from increased intra-abdominal pressure; after delivery the hernia usually reduces itself, but it sometimes recurs apart from pregnancy later in life.

Clinical Features.—Reflux œsophagitis is the commonest affection of the œsophagus, but its manifestations are not always clear-cut.

Pain.—In the majority, pain is the presenting symptom. At first, it is retrosternal and later may radiate between the shoulder blades, down either arm or up the side of the neck to the ear. This referred pain may simulate angina and is an expression of the sympathetic innervation. It tends to occur when the patient lies down to sleep and keeps him awake (in contradistinction to the pain of duodenal ulcer which wakes the patient from a sound sleep at about 2 a.m.). It is relieved by sitting upright and sometimes by taking alkalis.

Heartburn with regurgitation of bitter fluid is very common.

Dysphagia.—A complaint that food sticks in the region of the lower œsophagus is a fairly frequent symptom of œsophagitis. It occurs long before any stenosis has developed, and is probably an indication of œdema and spasm. Later, dysphagia is constant and becomes progressively worse as the stenosis develops.

Occult blood is present in the stools in a large percentage of cases.

Secondary anæmia is not uncommon. The œsophagitis may only be uncovered when a routine search is made for the cause of such anæmia.

Diagnosis.—A barium swallow X-ray of the œsophagus reveals no characteristic sign until stenosis commences. *Œsophagoscopy* is essential in order to make the diagnosis. On examination, the mucous membrane of the lower end of the œsophagus is velvety, scarlet, and bleeds readily. Additional white areas of desquamating epithelium may also be seen in the inflamed area. Later one or more ulcers are present (Barrett's ulcers) (fig. 825) and progressive cicatrisation with narrowing of the terminal part of the œsophagus occurs.

Treatment is that of the cause, notably that of hiatus hernia (p. 798). Recently the H_2 antagonist cimetidine has proven a useful adjunct to treatment. It is still not established as a safe long-term remedy (p. 829).

HIATUS HERNIA

TYPES	Sliding or œsophago-gastric	85%
	Rolling or paraœsophageal	10%
	Mixed or transitional	5%

Ninety-eight per cent. of diaphragmatic herniæ (p. 755) occur through the œsophageal hiatus. Sir Astley Cooper was the first to insist that œsophageal

César Roux, 1857–1934. Professor of Surgery and Gynæcology, Lausanne, Switzerland. *(See footnote p. 854.)*
Norman Rupert Barrett, 1903–1979. Surgeon, St. Thomas's Hospital, London.
Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London.

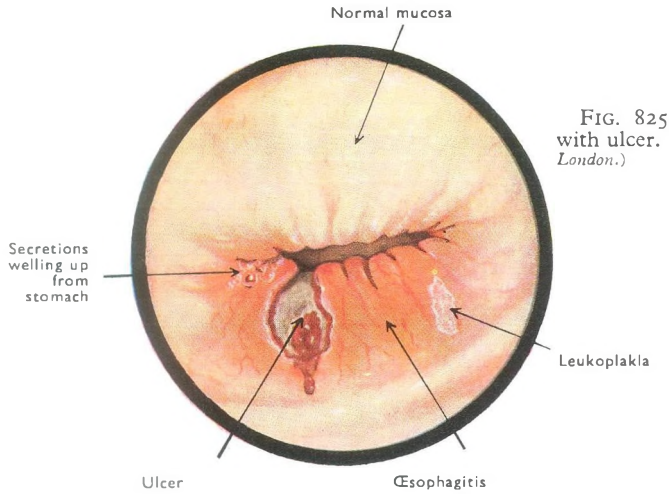


FIG. 825.—Reflux œsophagitis with ulcer. *(N. R. Barrett, F.R.C.S., London.)*

hiatus hernia was acquired, and not congenital. While probably this is true in the majority of cases, the fact is that it may occur in early infancy, and rapidly proceed to stricture and œsophageal shortening, a condition erroneously described as ' congenital short œsophagus '.

Sliding Hiatus Hernia

Anatomy of Cardiac Sphincter.—There is undoubtedly a functional intrinsic sphincter-like mechanism at the cardia although it cannot be demonstrated anatomically. This is expressed as a zone of high intra-luminal pressure which prevents regurgitation from the stomach. This important function is maintained by four

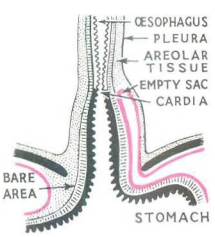


FIG. 826. — Sliding œsophageal hiatus hernia. *(After N. R. Barrett, F.R.C.S., London.)*

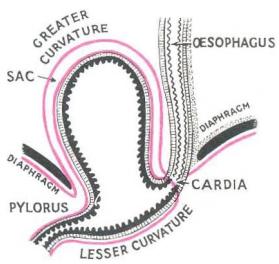


FIG. 827.—Para-œsophageal hernia.

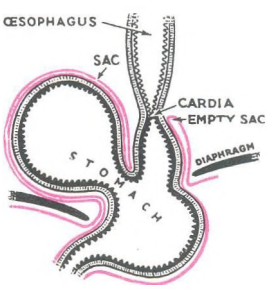


FIG. 828.—Mixed hiatus hernia.

factors: 1, the valvular effects of the œsophago-gastric angle (fig. 829); 2, the pinch-cock action of the right crus (fig. 830); 3, the ' rosette-like ' folds of the gastric mucous membrane at the cardia; 4, the presence of a length (about 4 cm) of intra-abdominal œsophagus (p. 798). Certain gastro-intestinal hormones may affect sphincter tone.

Mechanism of Herniation.—The hernia is small, and usually remains so. The cardiac orifice and a portion of the stomach immediately adjacent pass into the posterior mediastinum, carrying with them a small peritoneal sac applied to the left side of the stomach. The right side of the hernia is derived from the ' bare area ' of the stomach, and consequently is bereft of peritoneum (fig. 826). Branches of the left gastric artery supplying the prolapsed stomach also pass through the hiatus. The sac is always empty, and its fundus lies opposite the junction of the prolapsed stomach

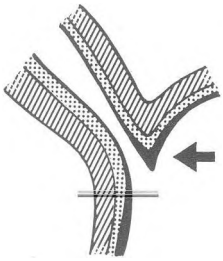


FIG. 829.—The valvular mechanism of the cardia.

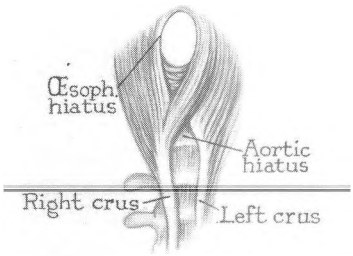


FIG. 830.—The right crus forms the entire musculotendinous diaphragmatic ring around the œsophagus.

(lined by columnar epithelium) and the œsophagus (lined by squamous epithelium). It should be noted that the prolapsed stomach, with its columnar-celled epithelium (black) hangs from the œsophagus proper like a supradiaphragmatic bell. In a less common variety there is massive herniation upwards of the stomach.

The final result is that the phreno-œsophageal ligaments are stretched and the cardio-œsophageal junction is distorted.

Ætiopathology of Herniation.—There are several factors in the development of this hernia:

1. Muscular degeneration of increasing age.
2. Increased intra-abdominal pressure as in large ovarian cysts, pregnancy, increasing weight and the wearing of tight corsets. Trauma may be an initial factor.
3. Increase of fatty tissue in the hiatus with decreased elasticity of the crus as occurs in obese women.
4. Once regurgitation is established, œsophageal spasm and later even œsophageal fibrosis will pull more and more stomach into the chest.

Clinical Features.—These are similar to those of reflux œsophagitis (see p. 794). The majority of the patients are over fifty years of age, and women are slightly more often affected than men. A duodenal or gastric ulcer may be present as well. Gall stones, diverticulitis coli and hiatus hernia are not uncommonly associated (Saint's triad, Chap. 46). An aspiration pneumonitis may develop.

Hiatus Hernia in Infants

Hiatus hernia with œsophagitis occurs in infants. The outstanding clinical feature is effortless vomiting of small amounts, often blood-tinged, starting soon after birth.

Radiography.—In order to demonstrate a sliding hiatus hernia radiologically, technique is important. During the course of a routine barium meal the patient is turned into a semi-prone position on the right side and the table tilted into a 20° Trendelenburg position. In the case of a true hiatus hernia, barium will regurgitate into the hernia without the aid of any additional pressure (fig. 831). If there is no regurgitation from the hernia into the œsophagus, there are usually no symptoms and no treatment is necessary.

FIG. 831.—A large sliding hiatus hernia.

Œsophagoscopy reveals varying degrees of inflammation of the lower end of the œsophagus (p. 790). The most valid sign of a hiatus hernia is reflux

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig, Germany (see footnote p. 75).

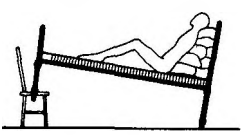
of gastric juice through the cardia, best seen during œsophagoscopy under surface anæsthesia. Furthermore the cardia will open on inspiration, whereas it normally closes and descends.

Differential Diagnosis.—Now that the prone head down position is routine in all barium meals, hiatus hernia and reflux are a not infrequent finding. Above the age of 60 years about 50 per cent. of all patients coming for a barium meal examination have a demonstrable hiatus hernia at some time or other (Airth). One must therefore be vigilant lest cholelithiasis, peptic ulcer, or diverticulitis be overlooked in a patient with an incidental hiatus hernia.

Factors which influence Treatment

In all cases medical treatment should be tried and faithfully followed. Many cases do not need surgery, and it is unwise to advise operation in the absence of definite indications. Operation undertaken in patients who are obese or who have chronic bronchitis often gives unsatisfactory results.

Medical Treatment.—The principles to be followed are:



1. The patient must sleep in a semi-recumbent position propped up with pillows and with the lower end of the bed raised on a chair. Five out of six patients benefit from lying on the left side at night.
2. He must avoid heavy work, lifting weights and excessive bending.
3. Six small, non-bulky meals should be taken daily, instead of three; food should be masticated thoroughly, and eaten slowly. The maintenance of the upright position after meals is sometimes of benefit.
4. Mucaine and Antacids such as Nulacin may control symptoms.
5. If the patient is obese, reduction of weight is very important.
6. Correction of anæmia.

Especially when the patient is old or follows a sedentary occupation, these simple measures may bring about and maintain such a remarkable improvement that the question of operation can be postponed indefinitely. In other circumstances operation should be advised.

Medical Treatment in Infancy.—The first injunction is to nurse the baby in an upright position, and the second is to thicken the feeds, giving semi-solid diet as soon as possible. The most practical method of maintaining the sitting position is by an almost legless chair made of plaster of Paris, which can be used both in the cot and the perambulator. Some children lose all symptoms when they start to walk; some of the remainder will require surgery.

Indications for Surgery

1. Severe symptoms which cannot be controlled medically or which interfere with patients' occupation.
2. Complications such as stenosis, hæmorrhage and œsophagitis.
3. Associated lesions such as gallstones, ovarian cysts and pyloric ulcers which should be dealt with in their own right.

Operative Treatment.—There are two approaches in common use—abdominal or thoracic. The guiding principle of surgical repair is to ensure a length of at least 4 cm. of intra-abdominal œsophagus and to prevent this segment from returning to the chest. The positive intra-abdominal pressure of inspiration closes this segment of gullet thus preventing the reflux of gastric juice into the thoracic œsophagus (Keen).

Graham Robertson Airth, Contemporary. Radiologist, Southmead Hospital, Bristol, England.
Gerald Keen, Contemporary. Thoracic Surgeon, Royal Infirmary, Bristol, England.

The choice between the abdominal and thoracic operation is vexed. If the case is not complicated by œsophagitis it may be approached from below. This approach makes any gall-bladder and duodenal surgery much easier; it is not anything like such a difficult procedure and avoids intercostal neuralgia which so often follows the thoracic approach. If severe shortening of the œsophagus is present the thoracic approach should be used.

Abdominal Approach—Fundo-plication. Nissen—almost by accident—found that wrapping the fundus of the stomach around the lower 5 cm of the œsophagus produced a very effective valve. A left upper abdominal incision is made in the line of the left margin of the rectus abdominis and the left lobe of the liver is mobilised to expose the cardia. A transverse incision at the hiatus is then made to expose the lower œsophagus and the crura. It is important to clear the abdominal œsophagus for 5 to 7·5 cm both behind the œsophagus and along the upper part of the lesser curve. The fundus of the stomach is freed, its anterior aspect is brought round behind the œsophagus and sutured anteriorly (fig. 832). Splenectomy may be required. Some believe that vagotomy with a drainage operation (p. 832) or more recently Highly Selective Vagotomy (p. 833) is advisable; this view is not widely shared. The operation is best carried out with a large bore stomach tube in position in order to prevent narrowing of the œsophagus. Lesions of the stomach, duodenum, gall bladder and colon may be dealt with at the same time. Late recurrence is reported to be uncommon and the persistence of symptoms to be of the order of 10%.

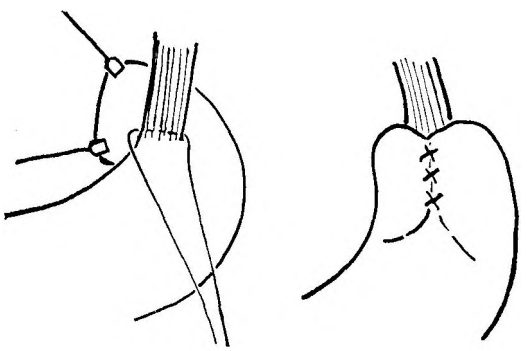


FIG. 832.—(*a*) Mobilisation of the gastric fundus with tissue forceps and a rubber catheter round the œsophagus. (*b*) Plication of the gastric fundus.

Thoracic Approach.—Nowadays this approach parallel to the left 8th rib is reserved for cases requiring extensive mobilisation of the œsophagus when perio-œsophagitis has caused severe shortening.

Treatment of Complications. Stricture and Penetrating Ulcer.—These complications result from the reflux of gastric and duodenal contents into the œsophagus, i.e. acid, the enzyme pepsin, bile acids, lysolecithin, pancreatic enzymes and partly digested food. Their treatment therefore is concerned with the control of reflux and dilatation of the stricture, or excision of it and the penetrating ulcer if dilatation is impossible.

Methods of Control of Reflux.—(1) Abdominal or transthoracic repair of the sliding hiatus hernia (see above). (2) Nissen Fundoplication (see above). (3) Belsey-Collis Gastroplasty. A plastic procedure on the fundus of the stomach at which part of it is fashioned into a tube to lengthen the œsophagus and the remaining part of the fundus wrapped round the tube and the lower end of the œsophagus.

Methods of Dilation of Œsophageal Stricture.—(1) Rigid œsophagoscopy and passing graded gum elastic bougies or stretching of the stricture with a hydrostatic bag. (2) Fibre-optic œsophagoscopy and inserting a guide wire through the stricture over which flexible dilators are passed.

Undilatable Œsophageal Stricture.—The stenosed portion is resected and continuity restored by œsophago-gastrostomy or by interposition of a jejunal or colonic conduit. More recently Thal's patch has been gaining in popularity (Pringle). The operation is done through a left thoracotomy incision (fig. 833).

Very Elderly and Frail Patients.—These are unsuitable for curative surgery and have to be palliated by regular dilation. Medication with antacids, cimetidine, pyrogastrone or preparations containing alginic acid may help. Anticholinergic drugs are contraindicated because they may reduce the resting tone of the lower

Rudolph Nissen, Contemporary. Formerly Professor of Surgery, Istanbul, Turkey, and Basle, Switzerland.
Ronald Belsey, Contemporary. Consultant Thoracic Surgeon, Frenchay Hospital, Bristol, England.
John Leigh Collis, Contemporary. Emeritus Professor of Thoracic Surgery, Queen Elizabeth Hospital, Birmingham, England.
Alan P. Thal, Contemporary. Surgeon, Kansas City, Kansas, U.S.A.
Rupert Pringle, Contemporary. Consultant Surgeon, Dundee, Scotland.

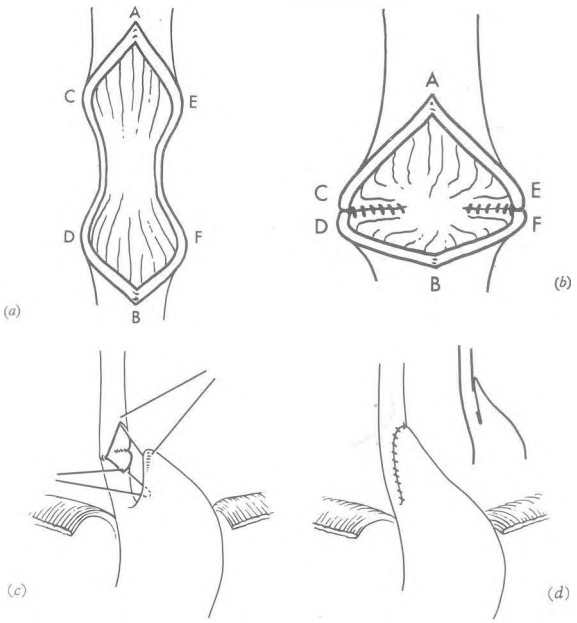


FIG. 833.—Thal's patch. (*a*) Longitudinal incision (A–B) through stricture. (*b*) Enlargement of lumen by approximating C to D, E to F. (*c*) Onlay patch of gastric fundus to œsophageal defect. (*d*) The completed patch. (Inset shows longitudinal section through patch.) (*Reproduced by permission of the Royal College of Surgeons of Edinburgh.*)

œsophageal sphincter and hence reduce the volume of alkaline saliva in the œsophagus which has a buffering effect on gastric acid reflux.

Hæmorrhage.—Although chronic œsophagitis can cause severe iron deficiency anæmia and is an indication for control of reflux, severe hæmorrhage from a penetrating ulcer may necessitate excision.

Para-œsophageal ('Rolling') Hernia.—This is a true hernia into which the greater curvature of the stomach (figs. 827 and 835) or, very rarely, the whole stomach itself ascends into a preformed sac lying in the mediastinum (fig. 827). Acute dilatation or volvulus of the intrathoracic stomach may result in serious symptoms or death and such herniæ are best repaired early.

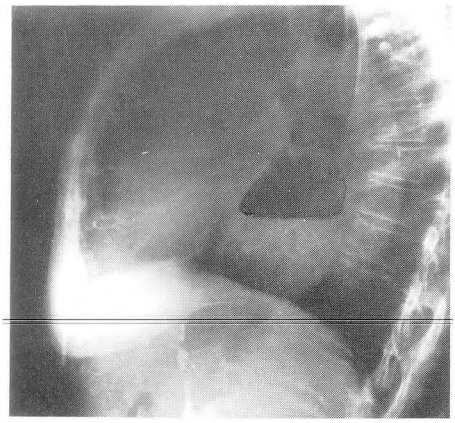


FIG. 834.—Para-œsophageal hernia. A fluid level is seen in the chest.

FIG. 835.—Barium study of this case.

Clinical Features.—Regurgitant œsophagitis and peri-œsophagitis are not features of this condition since the cardia remains in its normal position (fig. 827). The gastro-œsophageal angle is not disturbed and the competence of the cardia is not impaired. Symptoms therefore rarely appear until the hernia is large (fig. 827), and they are variable: (*a*) intermittent dysphagia; (*b*) cardiac symptoms due to pressure on the heart; (*c*) occasionally bouts of hiccough form irritation of the phrenic nerve. These symptoms are most frequently due to a recurrent gastric volvulus (p. 856).

Operation.—The muscular fibres of the crus are so attenuated by stretching that the operation described for sliding hernia is usually inapplicable.

For a right-sided hernia the thoracic approach is chosen, because the liver prevents a satisfactory abdominal exposure of the hernial site. In the case of the left side (much more common) the abdominal approach is chosen. The upper abdomen having been opened, a stomach tube is passed by the anæsthetist and all gaseous and fluid stomach contents are aspirated before reduction of the hernia is undertaken. Reduction having been affected, the hernial sac is usually excised. A very large adherent sac may have to be detached from the stomach, but the intrathoracic portion is left *in situ*.

The hernial orifice is repaired by rows of overlapping sutures (Harrington).

Mixed Hiatus Hernia.—This is a combination of sliding hernia and para-œsophageal hernia (fig. 828).

Clinical Features.—These depend on the degree of angulation of the cardia and the bulk of the para-œsophageal component. If angulation is disturbed and the paraœsophageal component is small, the main symptoms are those of reflux œsophagitis. Alternatively, normal angulation will be associated more with symptoms of a para-œsophageal hernia.

Treatment.—Operative treatment is strongly indicated. For the repair of the hernia some prefer the transthoracic route and others the abdominal route as described on p. 798.

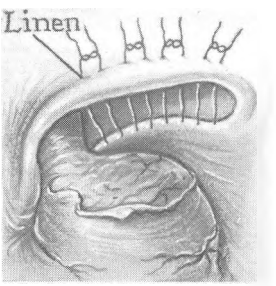


FIG. 836.—Repair of a para-œsophageal hernia. First layer of sutures. (*After S. W. Harrington.*)

CHRONIC ŒSOPHAGEAL OBSTRUCTION

The causes of chronic œsophageal obstruction can be classified as follows:

Congenital—see p. 790.

Acquired

- Pressure from without
  - Thyroid swelling
  - Pharyngeal pouch (p. 692)
  - Aortic aneurysm
  - Mediastinal
    - Neoplasm
    - Abscess
  - 'Rolling' Hiatal Hernia (p. 800)
- Causes in the wall
  - Stricture
  - Diverticulum
  - Neoplasm
  - Spasm
    - Plummer-Vinson syndrome
    - Achalasia
    - Diffuse spasm
- Causes in the lumen
  - Foreign body

LOCALISED MUSCULAR SPASM

There are two important clinical entities associated with muscular conditions of the œsophagus. One effects the pharyngo-œsophageal junction an is known as the Plummer-Vinson syndrome. The other affects the extreme lower end of the œsophagus and is called cardiospasm, or achalasia.

Stuart William Harrington, Contemporary. Emeritus Professor of Surgery, Mayo Graduate School of Medicine, Rochester, Minnesota, U.S.A.
Henry Stanley Plummer, 1874–1937. Physician, Mayo Clinic, Rochester, Minnesota, U.S.A.
Porter Paisley Vinson, 1890–1959. Physician, Mayo Clinic, Rochester, Minnesota, U.S.A.

THE PLUMMER-VINSON SYNDROME

This syndrome was first described by Paterson and Kelly in 1919. It is however commonly called after Plummer and Vinson who described it again in 1921. The patient is nearly always a middle-aged woman who comes complaining of difficulty or inability to swallow. Severe retching spells are wont to occur and classically the patient fears choking. Provided the clinician is aware of the existence of the syndrome, thoughtful examination nearly always provides clues to the diagnosis, but not all the following are necessarily in evidence in a given case.

The tongue is usually devoid of papillæ, smooth and pale, but rarely inflamed or sore.

The lips and corners of the mouth are often cracked, giving the mouth a pursed appearance.

FIG. 837.—Spoon-shaped finger-nail.

The finger-nails are brittle, and tend to be spoon-shaped (koilonychia) (fig. 837).

The spleen is enlarged in the same ratio as in other iron-deficiency anæmias.

The bone marrow is devoid of stainable iron stores.

The Blood.—Hypochromic[1] anæmia is always present, the serum iron levels being particularly low. Usually dysphagia precedes the anæmia, which is due to lack of iron and other requirements of a balanced diet.

Achlorhydria is often present, as is usually the case in iron-deficient anæmia.

Dysphagia is due to spasm of the circular muscle fibres at the extreme upper portion of the œsophagus. This is associated with the formation of webs (fig. 838). The mucous membrane is hyperkeratotic in places, and desquamated in others; it is extremely friable and easily traumatised by the passage of an œsophagoscope. In long-standing cases the orifice looks like a mere pin-hole. This lesion is definitely a precarcinomatous condition.

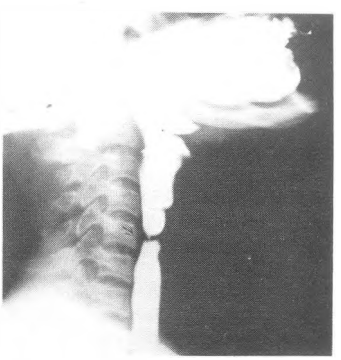


FIG. 838.—Œsophageal web. (*H. Talib, F.R.C.S., Baghdad*)

Treatment.—The dysphagia yields readily to dilatation of the stricture through an œsophagoscope. As always, gentleness must be used; even so, in cases of some standing, bleeding will occur. The administration of tab. ferrous sulphate, 0·3 g t.d.s., together with vitamins, is indicated. Some patients require intramuscular or intravenous iron. Sometimes blood transfusion is necessary. Hyperalimentation with a liquid diet through a gastric aspiration tube helps to bring about regeneration of the desquamated epithelium of the mucous membrane of the œsophagus. Once the anæmia is under control and the patient can swallow an adequate diet, rapid improvement occurs and usually is maintained.

ACHALASIA OF THE ŒSOPHAGUS (*syn.* 'CARDIOSPASM')

Ætiology.—Achalasia is a disease of the œsophagus, the exact cause of which still remains unsolved.

It consists essentially of a failure of integration of the parasympathetic impulses so that œsophageal peristalsis is disorganised, and there is failure of relaxation of the cardia. These result in a *functional* obstruction. An epidemic variety has been described in Breslau and was associated with Vitamin B_1 deficiency on the one hand and trypanosomiasis on the other (Chagas' disease). Stress and emotional upsets are also associated with achalasia.

The œsophagus consists of a dilated tortuous sac above and a narrow neck below. The elements of Auerbach's plexus are defective though ganglia are present (cf. Hirschsprung's disease (Chap. 49) where they are completely absent). There is marked

[1] Hypochromic anæmia is so-called because the red blood-corpuscles contain less hæmoglobin than normal.

Donald Rose Paterson, 1862–1939. Surgeon, Ear, Nose and Throat Department, Royal Infirmary, Cardiff, Wales.
Adam Brown Kelly, 1865–1941. Surgeon, Ear, Nose and Throat Department, Victoria Infirmary, Glasgow, Scotland.
Carlos Chagas, 1879–1934. Professor of Tropical Medicine, Rio de Janeiro, Brazil.

stasis and a retention œsophagitis due to the presence of a foul smelling fluid. Like stasis anywhere, it predisposes to diverticula and it is pre-cancerous.

Clinical Features.—Usually achalasia of the œsophagus occurs in women about forty years of age, but it can occur at any time of adult life and in both sexes. The history is one of progressive dysphagia; there are several special features.

The onset is insidious and, more often than not, the patient seeks relief only after the symptoms have been present for many years. Although the patient says she vomits, on closer interrogation it becomes apparent that there is regurgitation of food, often several hours after the meal. In advanced cases mucus and froth are brought up in considerable quantities. There may be retrosternal discomfort, fœtid flatulence and aspiration pneumonitis. A toxic 'rheumatoid' arthritis may occur.

As a result of the obstruction the patient fails to obtain sufficient nourishment, and consequently remains in a state of continual ill-health, rendering normal activities impossible.

The *radiographic findings* are most important. Characteristic features are:

1. A smooth pencil-shaped narrowing of the lower segment.
2. Dilatation and later tortuosity and recumbency of the lower œsophagus (fig. 839).
3. Lack of a gas bubble in the stomach.
4. Inco-ordinated peristalsis which is hypersensitive to acetyl-choline (when Carbachol 0·25 mg is injected intramuscularly abnormal peristaltic contractions occur).

Œsophagoscopy.—Once the instrument has passed the cricoid cartilage it appears to enter a gaping cave partially filled with dirty water, which laps to and fro with respiratory movement. When the fluid has been aspirated the cardiac orifice is located with difficulty, owing to its contracted state.

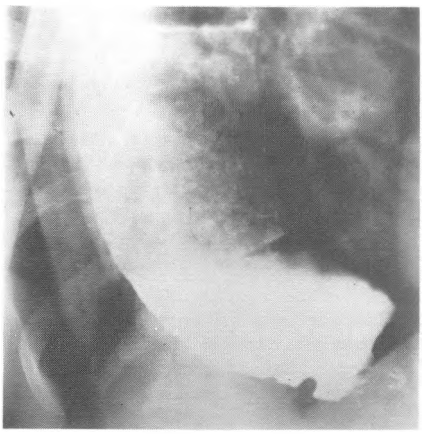


FIG. 839.—Typical appearance after barium swallow in a case of achalasia showing dilatation and recumbency.

Treatment.—The essential is the disruption of the constricting fibres of the cardia either from without by Heller's operation or from within by Plummer's bag.

Operative treatment becomes necessary in at least 20 per cent. of cases.

Heller's Operation (*Œsophagocardiomyotomy*).—Aspiration of the dilated œsophagus is commenced twenty-four hours before operation during which time the patient is on a water diet. The approach may be thoracic or abdominal but the essential procedure is the same. In the abdominal operation a long high left paramedian incision is made. The left lobe of the liver is retracted to the right and the triangular ligament, thus rendered taut, is severed. The stomach is retracted downwards so as to display the abdominal portion of the œsophagus, the peritoneum overlying which is incised, permitting the lower end of the œsophagus to be liberated circumferentially. A soft rubber catheter is passed around the cardia to facilitate retraction. When the lower end of the œsophagus has been mobilised sufficiently, a longitudinal incision is

Ernst Heller, Surgeon, St. George's Hospital, Leipzig, Germany.

made extending 5 cm proximal and 5 cm distal to the constricted portion. The incision is deepened to the mucosa throughout the length of the incision (fig. 819). After its completion, the mucous membrane bulges, as occurs in Ramstedt's operation (p. 819). The rubber sling is removed and the abdomen is closed. The advantage of the thoracic operation is that the myotomy can be carried up much higher on the œsophagus. This is necessary in about 10% of cases.

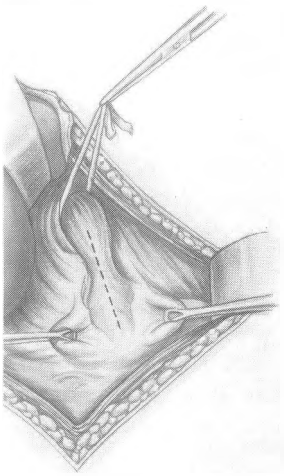


FIG. 840.—Heller's operation, showing the length and position of the incision dividing the muscular coats. (*After P. Thorek, Chicago, U.S.A.*)

In 80 per cent. of cases the results of Heller's operation are most satisfactory; in the remainder, symptoms due to reflux œsophagitis develop and mar the benefits conferred by the operation. This can be much reduced by a formal repair of the hiatus at the time of operation (Collis).

Plummer's Hydrostatic Bag.—A silk thread with a shot on the end is swallowed, and when the shot has entered the stomach, suitable bougies are passed by the 'rail-road' method. Once the narrow neck has been dilated sufficiently, the hydrostatic bag (fig. 841) is inserted. Plummer's bag often succeeds where dilatation fails, because the hydrostatic bag can be distended to a transverse diameter of 5 cm. This ruptures the circular muscle fibres of 'the neck'.

Diffuse spasm (Presbyœsophagus).—In this condition multiple areas of spasm (fig. 842) may be seen throughout the œsophagus ('corkscrew œsophagus'). Like scleroderma, however, it does not proceed to the massive dilatation seen in achalasia. 85% of cases are men over 60 (Flavell).

The condition presents as mild dysphagia with pain on swallowing felt mainly in the root of the neck but may be discovered accidentally during barium meal examination. The patient is usually of a nervous disposition. Treatment is by dilatation, although there is evidence that cholinergic antagonists and cholinesterase inhibitors may ultimately prove useful.

SCLERODERMA

Œsophageal manifestations of this distressing systemic disease are not uncommon, although the peripheral cutaneous, vascular and arthritic changes are more frequently seen.

The disease has a male:female ratio of 1:2 and is of the collagen variety.

Dysphagia is the commonest gastrointestinal symptom, although other

FIG. 841.—Plummer's hydrostatic bag *in situ*.

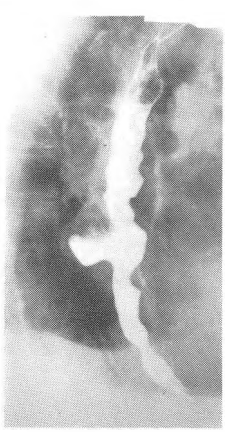


FIG. 842.—Diffuse spasm. A diverticulum is present in the mid-œsophagus.

FIG. 843.—Scleroderma of the œsophagus.

Geoffrey Flavell, Contemporary. Surgeon, The London Hospital, England.

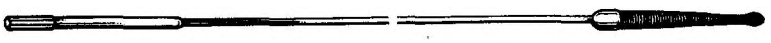
regions of the gastrointestinal tract may be involved and indeed, intestinal obstruction may occur.

Widespread infiltration of the mucosa of the viscus with fibrous tissue leads to ineffectual contraction and eventually to a suspension of peristalsis and shortening particularly in its distal half (fig. 843). Areas of spasm may occur proximal to this. In its fully developed state surgical replacement of the lower œsophagus may be required.

BENIGN STRICTURES

Compared with obstruction due to carcinoma, simple strictures are rare in this country. As set out in the table on p. 801, the causes of benign stricture are numerous. When consequent upon swallowing corrosive poisons, the strictures are usually multiple, the densest being at the level of the crossing of the left bronchus. The symptom of benign strictures is increasing dysphagia, and the diagnosis is established by a barium swallow X-ray and by œsophagoscopy. *A Schatzki's ring* is a kind of mucosal stenosis situated just above the cardia.

Treatment.—In the established condition treatment consists in dilatation with bougies (see below). In the first instance this should always be carried out under vision; in most cases it is advisable to continue to dilate the stricture at regular intervals under the vision afforded by an œsophagoscope. When the stricture has a very small



Chevalier Jackson's carrot-shaped œsophageal bougie.

lumen a special guide is passed, to the end of which a larger bougie is attached by a screw. By these means nearly all simple strictures can be dilated sufficiently to permit bougies to be passed in the usual manner, but treatment must be continued at regular intervals suited to the individual case for the remainder of the patient's life.

In cases where a bougie cannot be passed from above or threaded along a swallowed string weighted by a small shot, retrograde bouginage can be attempted through a gastrotomy. If this measure is successful, bougies can be passed afterwards in the usual way. In extreme cases, where dilatation of a dense stricture is impossible, colon replacement of the strictured segment is advisable (p. 808).

In acute corrosion the use of cortisone has been found valuable.

BENIGN TUMOURS

Simple tumours of the œsophagus are exceedingly rare. Usually they are discovered as a space-occupying lesion in a radiograph following a barium swallow, ordered on account of dysphagia. The diagnosis is confirmed by œsophagoscopy with, perhaps, biopsy. Papilloma of the œsophagus is usually solitary, and occurs in the upper third of the organ. Diathermy excision through an œsophagoscope is a satisfactory method of treatment. A polyp with a narrow stalk can be removed with a snare. Occasionally a leiomyoma or a submucous lipoma causing symptoms of dysphagia has been removed successfully by the transthoracic approach. The most difficult benign neoplasm of the œsophagus to treat is a cavernous hæmangioma which, as might be expected, usually gives rise to hæmatemesis. Injection of a sclerosing agent is sometimes successful; in others, partial œsophagectomy is required.

CARCINOMA OF THE ŒSOPHAGUS

Most cases are diagnosed only after œsophageal obstruction has supervened and the tumour has progressed beyond the anatomical limits of the organ. By this time it has usually involved the regional, if not distant, lymph nodes. In spite of modern methods of treatment, only 2 or 3 per cent. of patients with this disease survive for five years after the diagnosis has been made. In selected cases 9 per cent, 5-year survival has been reported.

The œsophagus is a common site for a carcinoma in men but is exceeded in

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Chevalier Jackson, 1865–1958. Professor of Bronchoscopy and Œsophagoscopy, Jefferson Medical College, Philadelphia, Pennsylvania, U.S.A.

incidence, by the bronchus, stomach, colon, prostate and pancreas. In Japan, carcinoma of the œsophagus occurs even more frequently, and at a younger age, than in Western races. It may be primary or secondary. The latter is becoming more common with the increased incidence of bronchial carcinoma.

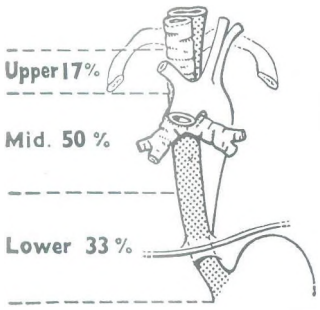

FIG. 844. — The relative frequency of squamous-celled carcinoma in various portions of the œsophagus.

Pathology.—The lesion may be squamous celled or an adenocarcinoma. The latter occurs essentially at the lower end from the lower 3 cm lined by columnar epithelium (fig. 816), or may be secondary to carcinoma of the stomach. The sites of predilection for the squamous-celled growth are the three areas of constriction (fig. 817). The incidence is shown in fig. 844.

Macroscopically three types can be recognised:

1. An annular stenosing lesion usually found at the cardia.
2. An epitheliomatous ulcer with everted edges.
3. A fungating cauliflower-like friable mass.

Spread: (*a*) *Direct.*—This is the main method of spread and the most important to the surgeon. It is both transverse and longitudinal in direction and erodes the muscular wall to invade the important structures of the neck and posterior mediastinum, as well as the lungs and trachea. It may perforate and lead to mediastinitis; rarely it causes massive fatal bleeding from the aorta.

(*b*) *Lymphatic.*—Submucosal lymphatic permeation may lead to satellite nodules away from the main tumour. Similarly, embolic spread to surrounding lymph nodes occurs. From the cervical œsophagus the spread is to the lymph nodes of the supraclavicular triangles. From the thoracic œsophagus metastases pass to the para-œsophageal and tracheo-bronchial lymph nodes, with downward extension to the subdiaphragmatic nodes. In the case of the abdominal œsophagus, spread is to the lymph nodes along the lesser curvature of the stomach, and thence to those around the cœliac axis. In the main, lymphatic metastases occur in a *downward* direction.

(*c*) *By the blood-stream* is exceptional; occasionally metastases occur in the liver or the lungs.

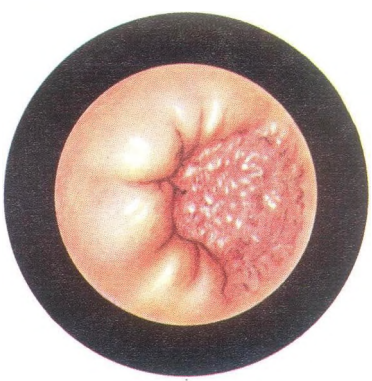

FIG. 845.—Carcinoma of the œsophagus as seen through an œsophagoscope. (*A. Lawrence Abel, F.R.C.S., London.*)

Clinical Features.—Men between forty-five and seventy are the usual victims, their average age being sixty-three. About 28 per cent. of the patients are women, and in them the neoplasm is situated most frequently in the upper third of the œsophagus; their average age is in the late fifties.

The leading symptom—indeed, often the only symptom—of this fell disease is dysphagia. Sometimes, owing to sloughing of a portion of the growth, for a time swallowing becomes easier, but as a rule the difficulty is

steadily progressive. Only 40 per cent. of patients report within three months; too often the patient procrastinates seeking advice until he can no longer swallow milk pudding. By this time the growth must have encircled at least three-quarters of the lumen of the œsophagus and, as might be expected, loss of weight is obvious. Regurgitation (œsophageal pseudo-vomiting) is a fairly common symptom. The regurgitated material is alkaline, mixed with saliva, and possibly streaked with blood. In growths situated in the lower part of the œsophagus, anorexia is sometimes a feature. Pain, if it occurs, is usually a late manifestation, but it is not in itself a contraindication to an exploratory operation.

Radiography after barium emulsion has been swallowed should be carried out in every case of dysphagia. By this investigation the presence of a carcinoma of the œsophagus (fig. 846), or, more usually, the stenosis produced by the neoplasm ('rat tail'), is displayed (fig. 847). Nevertheless, complete reliance must not be placed on the radiological findings, particularly if the examination is negative.

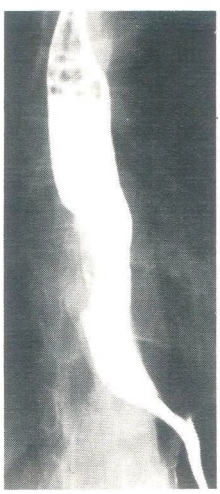

FIG. 846. — Early carcinoma of the œsophagus.

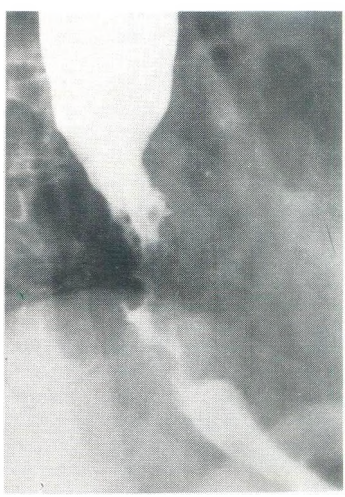

FIG. 847.—'Rat tail' appearance, in advanced carcinoma.

Œsophagoscopy is usually diagnostic. A piece of the tumour must always be removed for histology.

Bronchoscopy should be undertaken when there is a cough with expectoration of large quantities of purulent sputum which suggests involvement of the bronchial tree, and possibly a fistula.

Exfoliative Cytology.—Lavage of the œsophagus and examination of the fluid for malignant cells has led to the discovery of an early carcinoma when radiology and œsophagoscopy were negative.

Treatment.—Dysphagia is a most distressing symptom and somehow it must be relieved. Gastrostomy is an unsatisfactory procedure and is mentioned only to be condemned. Treatment is by operative resection, radiotherapy or by some palliative procedure.

Pre-operative Treatment.—Hourly feeds, as much as the patient can take, of fortified milk (2 eggs, 60 g sugar, 1 teaspoonful salt, 60 g dried milk, and 30 g of butter to 1·0 l of milk) are given. Breathing exercises and, when necessary, postural

drainage are carried out. The patient's teeth, if present, are scrutinised as hygiene of the mouth is most important. Suitable preparations of iron and vitamins are prescribed. Antibiotic therapy is commenced three days before the operation.

Operation.—It is only after the region of the growth has been displayed and the extent of its fixity to surrounding structures ascertained that it is possible to tell if partial œsophagectomy is practicable. As a result of exploration, about 50 per cent. of growths will be found to be irremovable, in which case a short-circuiting operation is performed. If the growth is operable, the methods of procedure are as follows:

(*a*) **Growth at the Cardiac Orifice.**—Through a left thoraco-abdominal approach a block dissection of the stomach, together with the spleen, omenta, and tail of the pancreas, is undertaken. Restoration of the continuity is by œsophagojejunostomy, employing a Roux-en-Y anastomosis.

(*b*) **Growth situated in the Lower Third of the Thoracic Œsophagus.**—The operative surgery for such a growth has swung away from the left thoracoabdominal exposure to that used for a **growth in the middle third,** namely an abdominal and right thoracic approach. This trend has been due to the difficulty in securing a sufficient length of growth-free œsophagus for an anastomosis below the arch of the aorta. An abdominal incision is made first, and the greater and lesser curvatures of the stomach fully mobilised from cardia to pylorus, preserving the right gastric and gastro-epiploic vessels. The cardia is freed from its hiatus at this stage and a pyloroplasty is performed. The patient is then turned on to his left side, and the œsophagus approached via a right-thoracotomy at the level of the fifth rib. The vena-azygos is the only structure requiring division. The œsophagus is then fully mobilised after which the stomach is drawn up into the chest. The œsophageal resection extends from 6 cm above the growth right down to the cardia to include a cuff of gastric wall. This gastric defect is closed in two layers, and the highest point of the fundus anastomosed to the cut end of the œsophagus (fig. 848). Debilitated patients may benefit from a two-stage procedure several days apart.

(*c*) **Growth lies in the Thorax above the Arch of the Aorta.**—See Post-Cricoid carcinoma (p. 699).

Other Methods of bridging the Defect.—The great disadvantage of utilising the stomach as a substitute for the œsophagus is reflux œsophagitis above the anastomotic line. For this reason other methods are being used. The best of these is colon replacement. An isolated segment of the transverse or the right colon, with its blood supply intact, is brought up to connect the stump of the œsophagus to the stomach. Jejunal loops are not favoured. Leaks after œsophago-gastric anastomosis seem fewer.

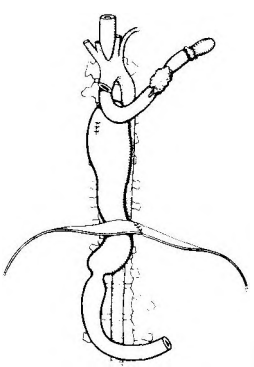

FIG. 848. — Resection of the œsophagus for a growth in its middle third, utilising the mobilised stomach for restoring the continuity of the alimentary tract (semi-diagrammatic).

Radiotherapy.—In carcinoma of the upper and mid-œsophagus radiation frequently restores the ability to swallow, although this benefit lasts only a few months. Recent results with penetrating radiation are more encouraging and an increasing number of long-term survivals are being obtained.

Growths of the Upper Third.—The magnitude and severity of extirpation of the œsophagus in this region, and the radio-sensitivity of most of the neoplasms, render radiotherapy the method of choice.

Growths of the Mid Third.—The ^{60}cobalt teletherapy unit emits gamma radiation equivalent to the most powerful super-voltage X-ray machine, and a rotational device has enabled a more uniform dose to be delivered to the growth. In a number of instances, as a result of this treatment, the tumour disappears; however, in spite of improvement in swallowing, the majority of patients soon die of metastases.

Growths of the Lower Third.—Frequently the neoplasm is columnar-celled and less radio-resistant and figures of from 10–15% five-year survival rate are reported.

The results of radiotherapy plus operation appear to be little better than either alone. McKeown, however, found about 30% of his patients surviving after 5 years after excision of a middle-third growth and slightly less living for 3 years after lower-third growth. The operative mortality was about 25% in growths above the lower third.

PALLIATIVE PROCEDURES

When Thoracotomy reveals that the Growth is Irremovable.—If the neoplasm is situated at the cardia, the œsophagus can be divided above the growth, the lower end closed, and œsophagojejunostomy-en-Y (Roux) performed above the obstruction. When the neoplasm is higher in the thoracic œsophagus, after similar exclusion, the previously mobilised stomach can be anastomosed to the œsophagus above.

When Inoperability is decided upon at a Clinical Examination.—Inoperability is so frequent and the results of heroic surgery so disappointing that a simple palliative procedure assumes an added importance. The introduction of a tube not only allows the patient to take sufficient nourishment by mouth, but relieves him of his one distressing symptom: dysphagia. It also protects him from recurrent aspiration pneumonitis. Neither of these is avoided by a gastrostomy.

The Souttar tube, made of German silver and pushed blindly into position after dilatation, has been replaced by tubes that are *pulled* into position. The Celestin tube based on physiological considerations consists of a 25-cm shaft carrying an oval barrel-shaped funnel about the size of a bolus. It is pulled into position by a detachable pilot-bougie. The later is navigated through the growth under direct vision, no dilatation being required. The tip of the bougie is recovered in the stomach via high gastrotomy. The endo-œsophageal tube is then plugged on securely to the bougie and traction applied steadily to the latter so as to draw the tube through the tumour. The tube must be finally left in a comfortable vertical position, even if this means that the funnel lies several inches above the tumour (fig. 849); otherwise ulceration may result. It is then tethered to the lesser curve and any excess cut off. After the operation the patient may be allowed a fairly free diet provided it is chewed very thoroughly, and frequently washed down with aerated water. The tube may also be used in patients receiving high-voltage irradiation, or as a preoperative measure.

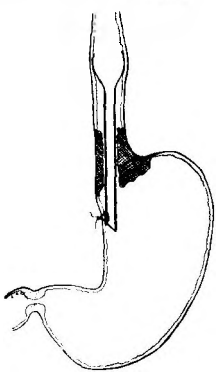

FIG. 849.—Method of fixation of tube.

Terminal Complications.—Unresected, the growth causes death in one of the following ways:

1. Progressive cachexia and dehydration.
2. Pneumonia from perforation into some part of the bronchial tree.
3. Mediastinitis from perforation into the posterior mediastinum.
4. Erosion of the aorta (rare).

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Sir Henry Sessions Souttar, 1875–1964. Surgeon, The London Hospital.
Louis Roger Celestin, Contemporary. Surgeon, Frenchay Hospital, Bristol, England.

CHAPTER 43

THE STOMACH AND DUODENUM

SURGICAL ANATOMY

Blood Supply

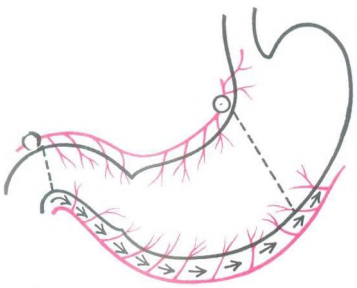


FIG. 850.—The main arteries of the stomach. Circles denote where the R. and L. gastric are divided. Arrows denote place of section of gastro-epiploic arteries. Dotted lines show where stomach is divided in partial gastrectomy.

Arterial Supply (fig. 850).—(*a*) The left gastric artery, the smallest branch of the coeliac axis, runs towards the cardiac orifice and thence along the lesser curvature, from left to right, to join (*b*) the right gastric artery, which arises from the hepatic artery and pursues a course from right to left along the lesser curvature. (*c*) The gastro-duodenal artery is the largest branch of the hepatic artery. It passes behind the first part of the duodenum, where it bifurcates into the superior pancreatico-duodenal artery and (*d*) the right gastro-epiploic artery. (*e*) The left gastro-epiploic artery is the largest branch of the splenic artery. (*f*) Vasa brevia (short gastric arteries) are five to seven small vessels that spring from the splenic artery towards its termination, and are distributed to the fundus of the stomach. An important feature of the gastric circulation is the large number of arterio-venous shunts in the submucosa of the lesser curve. These are opened up by vagotomy.

Veins.—Those corresponding to the right and left gastric arteries terminate in the portal vein. Those corresponding to the left gastro-epiploic artery and vasa brevia join the splenic vein, while the right gastro-epiploic vein empties into the superior mesenteric vein. In the living, the veins of Mayo (fig. 851), are helpful landmarks in distinguishing the pyloric canal from the first part of the duodenum.

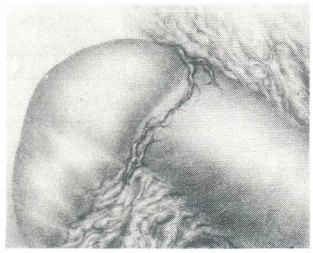


FIG. 851.—The pre-pyloric vein of Mayo.

Lymphatics are described on p. 853 in connection with malignant neoplasms of the stomach with which they are so intimately concerned.

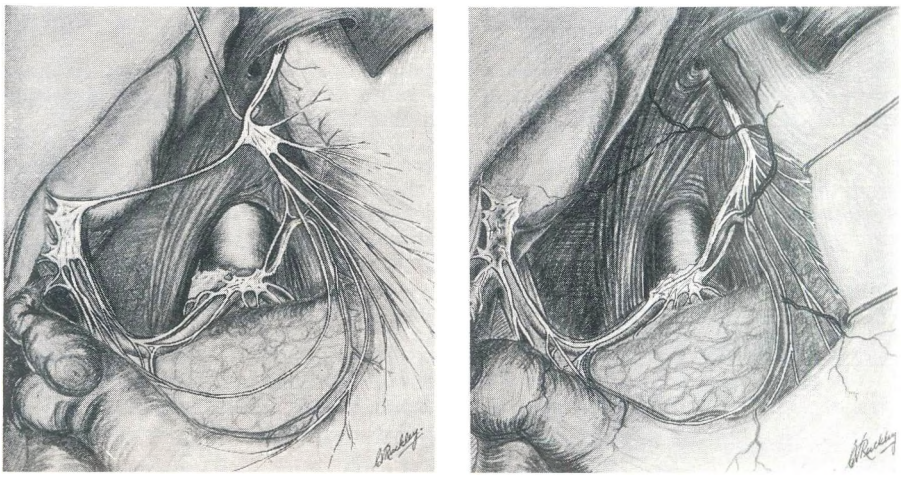
Innervation.—The vagus nerves are the motor nerves of the stomach. Fig. 852 shows the general disposition of the nerves, and their branches. Familiarity with the common distribution and main variations of the nerves is important owing to the swing from total to highly selective vagotomy (parietal cell vagotomy) operations for duodenal ulcer (p. 833).

Histology

Gastric Epithelial Cells lining the stomach are of the columnar type. They are filled with mucigenous granules and are responsible for the lubrication of contents. About a third of them regenerate by mitosis daily. They do not respond to acid secretagogues.

Parietal Cells.—These occur only in the body of the stomach and lie in the gastric crypts. Electron-microscopic studies show many inter-cellular canaliculi which communicate with the crypt lumen. They are responsible for the secretion of isotonic HCl to a pH of 0·9. In a normal adult male the parietal cell population is of the order of 1 billion whilst in about two-thirds of duodenal ulcer cases this may

William James Mayo, 1861–1939. Surgeon, Mayo Clinic, Rochester, Minnesota, U.S.A. The pre-pyloric vein should be called the Vein of Latarget as it was first described by André Latarget (1876–1947), Professor of Anatomy, Lyons, France.



A B

FIG. 852.—A, anterior and B, posterior vagus nerve. Showing distribution to stomach, cœliac, and subhepatic plexuses (*C. V. Ruckley, F.R.C.S., Edinburgh*). The anterior and posterior nerves of Latarjet are seen crossing the pancreas on their way to supply the pylorus. The sympathetic innervation of the organ may carry a proportion of pain transmitting fibres.

be nearly doubled. In the Zollinger-Ellison syndrome (Chap. 47) a four-fold increase has been reported. In gastric ulcer the count is low.

Chief Cells.—These lie mainly in the proximal part of the gastric crypts in the fundic mucosa and are responsible for the secretion of pepsinogen.

Endocrine Cells.—The mid-zone of the gastric mucosa contains G-cells, which synthesise gastrin. G-cells are found mainly in the gastric antrum, and their distribution differs from that of parietal cells on which gastrin acts. The number of G-cells may be increased in duodenal (but not gastric) ulcer patients, and is greatly increased in the condition known as G-cell hyperplasia (Polak).

PHYSIOLOGY OF THE STOMACH AND DUODENUM

The important physiological functions of the stomach and duodenum are secretory and motor. The stomach acts as a reservoir from which food, ingested over a period of minutes, is released over a period of hours after undergoing acid and peptic digestion into the duodenum. In the duodenum, the mixed food and gastric secretions, now known as ' chyme ', are brought to neutral pH by duodenal and pancreatic secretion of bicarbonate, and the osmolarity of the chyme is equilibrated with plasma. In addition, various nutrients stimulate the release from endocrine cells in the duodenal mucosa of polypeptides which modulate the secretory and motor activity of the digestive tract. The rate at which chyme is delivered from the stomach is dependent upon the osmolarity and the fat and caloric content of the chyme through receptors in the proximal duodenal mucosa (Hunt).

Secretion of Gastric Juice

The gastric juice consists mainly of pepsin, intrinsic factors, ions and other organic solutes in dilute hydrochloric acid. Conventionally, three phases of secretion of gastric juice are recognised.

1. **Cephalic.**—This is mediated by vagal activity, both from psychic arousal as demonstrated by Pavlov, and reflexly from antral stimulation (Uvnäs).

2. **Gastric.**—Gastrins are polypeptides differing in secretogenic potency and molecular size, but identical in action (Gregory), which are released from antral G-cells in response to mechanical distension of the antrum, and to meat proteins. After release from the G-cell, gastrin reaches the parietal cell *via* the systemic circulation. Secretion of acid is controlled by negative feedback; when intragastric

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Jack Naylor Hunt, Contemporary. Professor of Physiology, Houston, Texas, U.S.A.
Börje Karl Magnus Uvnäs, Contemporary. Professor of Physiology, University of Stockholm, Sweden.
Roderic Alfred Gregory, Contemporary. Holt Professor of Physiology, University of Liverpool.

pH falls below 3, due to acid secretion, the release of gastrin is inhibited. Because of the buffering capacity of a mixed meal, acid output is high for half an hour after eating, but falls thereafter. The role of histamine, long known as a potent stimulus of gastric acid secretion, has been a matter of controversy, but it now seems likely that gastrin acts on the parietal cell through the release of histamine.

3. **Intestinal.**—Prolongation of acid secretion for some hours after feeding was originally ascribed to ' intestinal factors '; but, since a mixed meal is not emptied from the stomach for several hours, it is probably due, for the most part, to prolonged gastrin release from mechanical stimulation of the antrum. The effect of chyme in the intestine on gastric secretion is largely inhibitory in order to preserve the neutral pH of the duodenum. Duodenal acidification provokes secretin release which inhibits gastric acid secretion (Grossman), and other intestinal polypeptides are probably implicated in this regulation; such polypeptides are termed ' gastrones '.

Pepsin Secretion

The secretion of pepsin is usually coupled to H+ secretion, but may be varied by cholinergic stimuli and by polypeptide hormones.

Gastric Mucus

The gastric mucosa is covered and lubricated by this viscid secretion, which is composed of mycopolysaccharides. As with pepsin, its secretion may be evoked by vagal stimulation on feeding. Its pH is alkaline, and it has considerable buffering capacity (up to 80 mmol/l, Hollander).

Gastric mucus forms a physiological barrier which protects the mucosa from mechanical damage and the effects of gastric acid. Davenport has suggested that this barrier is broken in cases of gastric ulcer, leading to back diffusion of H+ ion into mucosal cells; this would account in part for the apparently reduced secretion of acid in such patients, although the reduced number of parietal cells in such patients may produce hyposecretion. The barrier may be damaged by bile acids refluxed through the pylorus and by drugs such as salicylates and alcohol. Irritation of the gastric mucosa may lead to excess mucus production.

Duodenal Exocrine Secretion

Although the duodenum is a relatively short tube, its surface area is extensive, due to the arrangement of the mucosa in transverse folds, the valvulæ conniventes (' winking ' valves). In the first and second part of the duodenum, an alkaline secretion is produced by Brunner's glands into the crypts of Lieberkühn: the volume of secretion is related to the amount of acid delivered through the pylorus. This alkaline secretion is largely responsible for correcting the pH of chyme to neutrality.

Gastric and Duodenal Endocrine Secretion

Both in variety and quantity of secreted polypeptides, the alimentary tract may be justly termed the largest endocrine organ in the body. Although a gastrointestinal hormone, secretin, was the first hormone to be described in physiological terms by Bayliss and Starling, the majority of gastrointestinal polypeptide hormones have been described in the last decade, following advances in immunoassay, cytochemistry and protein chemistry. While the physiology of these substances is still uncertain, there are some facts which are generally agreed.

Structure of the Polypeptides

The majority of the polypeptides may be divided into two groups, on the basis of their structure. *Gastrin* and *cholecystokinin-pancreozymin* (CCK-PZ) have structural and functional affinities, with similar terminal amino-acid chains; both polypeptides occur in man in several variants of different peptide chain lengths. *Secretin*, *enteroglucagon* and *gastric inhibitory peptide* (GIP) have numerous structural affinities. Some polypeptides, such as *motilin*, appear to be unique in structure.

Localisation

The secretory cells of the polypeptide hormones are scattered among the epithelial cells of the gastrointestinal mucosa and are not aggregated in discrete glands. Gastrin is largely confined to the gastric antrum; most of the others are secreted in the small

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Horace Davenport, Contemporary. Professor of Physiology, University of Michigan, Ann Arbor, Michigan, U.S.A.
Johann Conrad Brunner, 1653–1729. Anatomist, Basle, Switzerland.
Johann Nathanael Lieberkühn, 1711–1756. German Anatomist.
William Maddock Bayliss, 1860–1924. Physiologist, University College Hospital, London.
Ernest Henry Starling, 1866–1927. Physiologist, University College Hospital, London.

intestine, and principally in the duodenum. All secretory cells are adjacent to the lumen of the digestive tract, and luminal stimuli provoking polypeptide release (such as peptones releasing gastrin) have been identified in some cases. Polypeptides are also found in myenteric nerves, and the existence of ' peptidergic ' nerves has been postulated (Polak). Some polypeptides normally found in the gut have also been identified within neurones in the central nervous system.

Function

Most of the polypeptides seem to be implicated in the control of digestive exocrine secretion, either exciting or inhibiting secretion. Gastrin provokes gastric acid secretion, secretin and CCK-PZ induce pancreatic secretion, while secretin inhibits gastric acid secretion.

A second role for the gastrointestinal polypeptides is in the control of gastrointestinal motor activity. CCK-PZ is the agent which mediates gall-bladder contraction, while gastrin stimulates gastric motor activity. Many other motor effects have been described, but, again, no clear picture has yet emerged.

The proximity of some endocrine cells to their exocrine target cells has led to the suggestion that *paracrine* effects (passage of polypeptide from secretory cell to target cell by local spread) may occur, but this is difficult to prove, and proof is as yet lacking. It is also possible that the function of some polypeptides is as *neurotransmitters*.

Gastroduodenal Motor Activity

The complex sequence of digestive and absorptive processes acting on ingested nutrients requires the orderly propulsion of luminal content through different regions at different rates; this is accomplished by neurohumoral regulation of the contractile activity of the smooth muscle which completely invests the digestive tract.

The motor activity of the stomach and duodenum is rhythmic, and is controlled by waves of electrical depolarization, known as *slow waves*. Slow waves are generated at proximal pacemaking sites in the stomach and duodenum, and spread aborally through the longitudinal smooth muscle fibres at speeds of several centimetres per

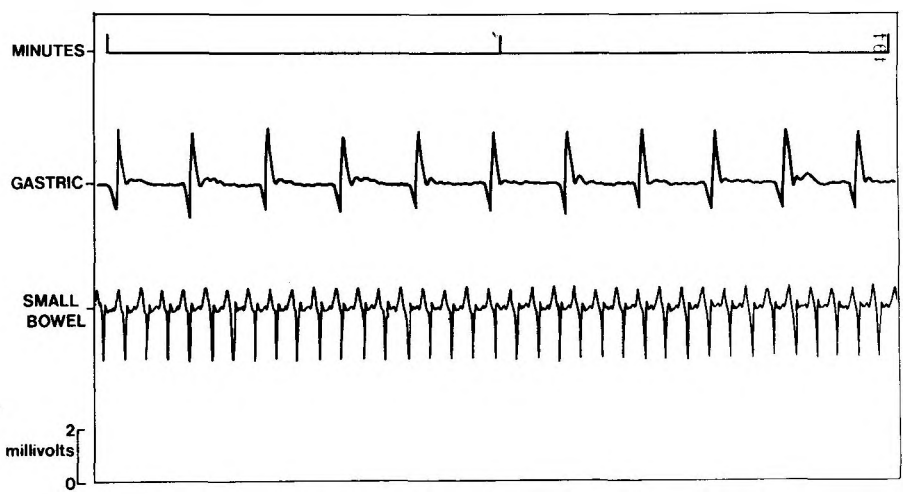


FIG. 853.—Pacemaking activity recorded at a single site in the canine stomach (upper trace) and duodenum (lower trace).

second. The gastric pacemaker, in the gastric fundus, emits three slow waves per minute which descend to, but do not pass, the pylorus. Duodenal slow waves are generated by a pacemaker immediately distal to the pylorus (Hermon-Taylor) at a rate of ten per minute. These rhythmic slow waves, sometimes known as the *basic electrical rhythm*, resemble the electrocardiogram in nature and function: slow wave recording from the serosal surface of stomach and duodenum show rhythmic changes similar to, but slower than the E.C.G. (fig. 853).

John Hermon-Taylor, Contemporary. Professor of Surgery, St. George's Hospital, London.

Since smooth muscle contraction at any site can occur only during the passage of a slow wave, pacemaker activity controls the *timing* of motor activity, and also the *direction*, as the slow waves travel caudally. But (unlike the E.C.G.) gastric and duodenal slow waves do *not* invariably initiate contraction during their passage. Simultaneous recording of duodenal electrical activity and intraluminal pressure change (fig. 854) shows regular slow wave activity, but pressure changes only occur with slow waves which show associated action potentials (fig. 854). Intrinsic and

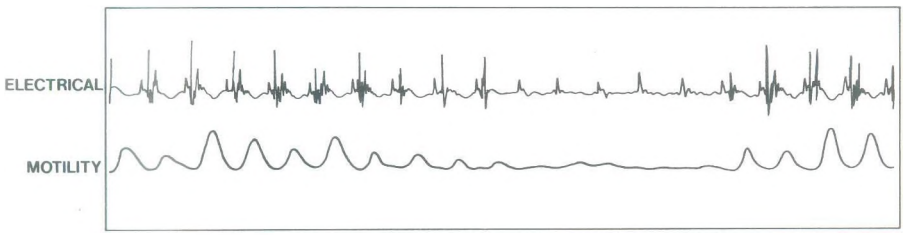


FIG. 854.—Simultaneous recording of myoelectric activity from a serosal electrode in the canine small intestine (upper trace) and intraluminal pressure adjacent to the site of the electrode. Regular pacemaking (small deflections) is visible, but pressure changes only occur when action potentials (large deflections) follow the pacemaking wave.

extrinsic neural and humoral changes, as yet not fully explained, determine whether or not a slow wave will produce contraction; it is these factors, which integrate the motor activity of stomach and small intestine with the processes of digestion and absorption.

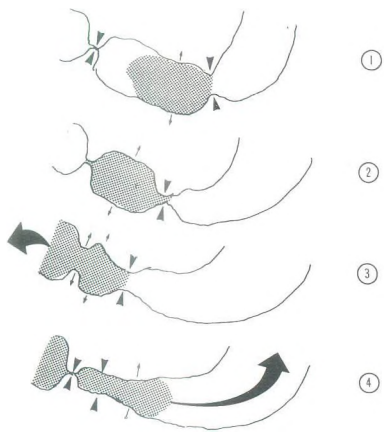
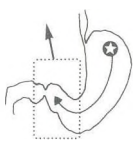


FIG. 855.—Sequential stages of gastric emptying. The pacemaker site and direction of flow wave propagation are shown at the right; the caudal propagation of contraction is shown in sequence from 1 to 4. Gastric contents are propelled into the antrum by the wave of contraction coupled to the slow wave (1, 2); as the antrum contracts some, content is expelled through the lax (3) as antral pressure rises. When the contraction wave reaches the pylorus (4), it closes, and antral pressure leads to retropulsion of gastric contents.

antral pump



Gastric Emptying

The mixing and slow emptying of a mixed meal from the stomach is accomplished by means of the 'antral pump'. Gastrin release stimulates gastric motor activity by a slight increase in slow wave frequency, and a marked increase in the associated contractile activity. Caudal contraction waves propel contents into the gastric antrum (fig. 855): as the antrum fills, the pylorus, which has no resting tone, opens to allow the escape of some chyme. When the contraction wave reaches the pylorus, it closes,

and the continuing antral contraction produces retropulsion of antral contents into the gastric corpus. Thus each contraction wave produces both the escape of a small quantity of chyme and the mixing of the remainder; in this way, the capacity of the duodenum to restore the pH of chyme to neutrality is not overcome. The volume lost through the pylorus with each contraction will depend upon the viscosity and the amount of solid in the gastric contents; fluids are emptied more rapidly.

Fasting *vs.* Feeding

There is a striking difference between the overall pattern of gastroduodenal motor activity during fasting, and following feeding. During fasting, a few minutes of active motor activity is followed by an hour or more of absent motor activity; this cycle of alternating brief activity and prolonged inertia is repeated indefinitely in the absence of food. This pattern is not merely cyclical; it is also migratory. The brief phases of motor activity occur synchronously in stomach and proximal duodenum; as they decline, the activity ' front ' passes caudally to the distal duodenum, and so on

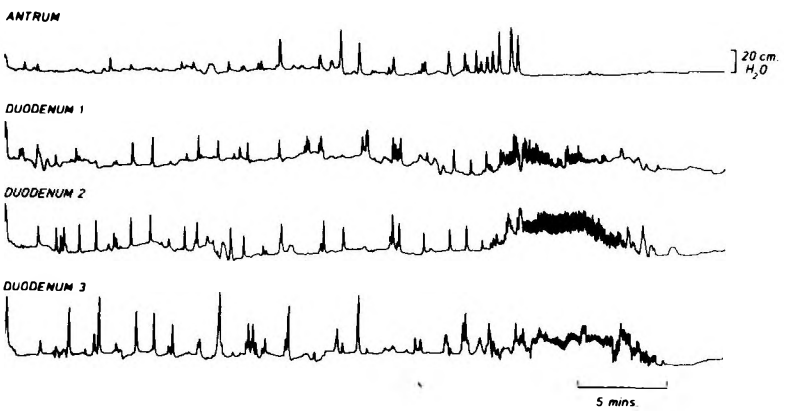


FIG. 856.—The onset of a human migrating complex in a healthy fasted subject registered by simultaneous recording from four pressure sensors equi-spaced between antrum and jejunum. At each site, pressure activity terminates in a burst of phasic contraction at 10/minute: this is the activity ' front ' (see text).

along the length of the small intestine. By contrast with the slow waves, the migration speed of these fronts is slow; passage from the stomach to the terminal ileum takes 1–2 hours, and the appearance of a new gastroduodenal front coincides with arrival of the previous front at the terminal ileum (fig. 856). These migratory complexes were described in the first decade of the 20th century as ' hunger contractions '; more recently, the migrating complex has been termed the intestinal ' housekeeper ' (Code) serving to propel fasting secretions and debris. Again in contrast to pacemaking activity, the ' clock ' which controls the migration of fasting fronts lies outside the digestive tract, possibly in the cœliac plexus. The duodenum is important in the integration of this activity; there is evidence that one of the duodenal hormones, *motilin*, is implicated in this fasting control.

The transit of the ' housekeeping ' front would be too fast to allow digestion and absorption of food to be complete; on feeding, the ' housekeeper ' is immediately abolished, to be replaced at all levels of the intestine by intermittent contractile activity, permitting agitation and mixing of the chyme without rapid passage. When the stomach is empty, the fasting pattern resumes. The effects of feeding on the pattern of gastric motor activity can be simulated by the administration of gastrin (Code); thus it seems that gastrin release has an important motor function. It seems probable that other gastrointestinal polypeptide hormones will be shown to be implicated in the regulation of motor activity, and it may be that further study will lead to clarification of some of the common but ill-defined ' functional bowel syndromes ' as disorders of neurohumoral regulation.

Charles F. Code, Contemporary. Associate Director, Center for Ulcer Research, Los Angeles, California, U.S.A.

CURRENT TESTS OF GASTRIC SECRETION

(1) Night Fasting Secretion
(2) Maximal Acid Output (Pentagastrin)
(3) Insulin Test (Hollander)

(1) **Night Fasting Secretion** (Dragstedt).—This is a method of assessing the inter-digestive or resting secretion of the stomach. Continuous or intermittent low-pressure gastric suction is carried out through an indwelling gastric tube previously screened into position. Normally about 400 ml. secretion is aspirated in the twelve-hour period from 9 p.m. to 9 a.m. Volumes above this level are suggestive of duodenal ulceration, and in Zollinger-Ellison syndrome cases (Chap. 47), volumes in excess of a litre occur. The estimation of free HCl content is valuable as an index of vagal activity (Table 1). This view has recently been challenged by Baron who found it to give variable results.

TABLE 1

Free HCl in mmol in twelve hours' night secretion

Normal	10– 20
Gastric Ulcer	5– 15
Duodenal Ulcer	40– 80
Duodenal Ulcer with Gastric Ulcer	40– 60
Zollinger-Ellison Syndrome	100–300

TABLE 2

Average HCl response in mmol Free Acid/hr

	Basal	*Maximal*
Gastric Ulcer	2	14·6
Duodenal Ulcer	6	37·5
Anastomotic Ulcer	7·7	31
Zollinger-Ellison Syndrome	22	

(2) **Maximal Acid Output** (Pentagastrin).—Basal secretion is measured by aspiration of the fasting stomach for 1 control hour. Then the pentagastrin test is performed. Gastric acid secretion may be maximally stimulated using pentagastrin (Peptavlon I.C.I.). Side effects are negligible. 6μg/kg body weight is administered s.c., or i.m. and 15 minute samples are collected during the subsequent hour. (See Table 2.) The total number of mmol/hr secreted is then calculated and is taken to be directly related to the size of the parietal cell mass. Ranges found in different conditions are shown in Table 2.

Only limited reliance may be placed on such tests if they are not carried out by experienced investigators. It can be taken, however, that a patient cannot have *less* acid than is aspirated. He *may* have more. If the technique is faulty this will not be realised.

The tests are of value in the diagnosis of stomal ulcer or the Zollinger-Ellison syndrome (Chap. 47). In duodenal ulcer they may be a useful guide to which operation is more suited to the patient, e.g. where low, vagotomy and drainage—where high, 50 mmol and above—vagotomy and antrectomy or partial gastrectomy.

Achlorhydria, by definition, occurs when a stomach cannot produce juice with a pH of less than 7·1 even after maximal stimulation. It occurs in 18% of patients with gastric carcinoma (Baron).

(3) **Insulin Test** (Hollander).—Since acid production following hypoglycæmia is attributed to direct vagal action on the parietal cell mass, insulin given to a patient who has had a vagotomy performed should result in no increase in acid production. This test is thus valuable post-operatively to verify the completeness of vagal section. To a fasting patient 2 units/10 kg soluble insulin are given intravenously, and serial quarter-hourly gastric aspirations performed for the next four hours. Blood-sugar levels are also measured at frequent intervals to ensure that the level falls below 35 mg/100 ml (2 mmol/l)—the level necessary to guarantee adequate vagal stimulation.

Early response.—A rise in concentration of 20 mmol per litre above the basal level in the first hour suggests incomplete vagotomy. If the basal samples were anacid, a concentration of 10 mmol per litre is regarded as positive.

Delayed response.—A rise in the concentration of acid secreted may occur between the first and fourth hours. This is thought to be due to vagal gastrin release. It is abolished by antrectomy. It suggests a less than complete vagotomy.

An early negative Hollander test may become positive some years later. The importance of this finding is not yet clear.

In addition, the glucose tolerance curve, an X-ray of the pituitary fossa, the serum

Lester Reynold Dragstedt, Contemporary. Research Professor of Surgery, University of Florida, Gainsville, U.S.A.
Jeremy Hugh Baron, Contemporary. Consulting Physician, St. Charles Hospital, London.

calcium and phosphorus levels, and the urinary 17-ketosteroids may help to elucidate the difficult case with an endocrine background (multiple adenoma syndrome).

Estimation of circulating gastrin.—Radio-immunoassay techniques are available. The principles of assay are:

A specific antigen is raised against gastrin. The biological sample competes with radioactive isotope-labelled hormone for combination with the antigen. The gastrin in the sample quantitatively inhibits the antigen binding with labelled hormone. The degree of inhibition gives a measure of gastrin in the sample. The findings have proved useful in diagnosis of the Zollinger-Ellison syndrome (Chap. 47).

Atrophy of Gastric Mucosa.—Atrophy may be due to duodenal regurgitation or it may be due to antibodies in the blood specific for the cytoplasm of the parietal cells. Such antibodies are found in pernicious anæmia in which there is also an antibody against intrinsic factor.

CURRENT TESTS OF DUODENAL SECRETION

No standard test of duodenal secretion exists, but monitoring of pH in the proximal duodenum may reveal an excessive fall in pH after a meal due to acid hypersecretion associated with duodenal ulcer.

CURRENT TESTS OF GASTRODUODENAL MOTOR ACTIVITY

Gastric emptying of a semi-solid meal can be monitored in a number of ways; the most reliable is the use of a gamma camera to follow the emptying of a meal of scrambled eggs labelled with Technetium. This may be of value in assessing the results of surgery on the gastric outlet.

HYPERTROPHIC PYLORIC STENOSIS OF INFANTS

Ætiology.—The cause of the condition, which occurs 3 or 4 times in every 1,000 births, is unknown. Several theories have been propounded, the most acceptable being that the condition is due to an achalasia (primary failure of the pylorus to relax). In a small proportion (about 7 per cent.) of cases the abnormality appears in certain families with greater frequency than can be accounted for by coincidence. In these families one parent bears the scar of an operation for hypertrophic stenosis in infancy, and in 50 per cent. of cases it is the mother, which, when the much higher incidence in the male is taken into consideration, is remarkable. In these families a brother is affected similarly in 10 per cent. and a sister in 2 per cent. of cases.

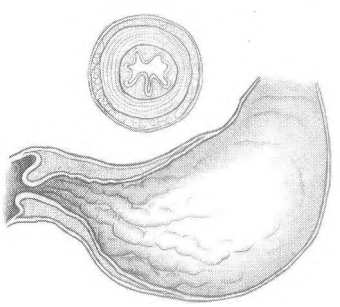


FIG. 857.—Pyloric stenosis of infants. Longitudinal and transverse sections of the stomach to show the enormous muscular hypertrophy. Note the abrupt termination.

Pathology.—The musculature of the pyloric antrum is always hypertrophied. Proceeding distally, the hypertrophy increases, to reach its zenith in the circular fibres of the pylorus. Here the muscle layer is so thick that the mucosa is compressed, and often the lumen admits only a fine probe. As estimated at operation, the size of the pyloric lump is proportional to the duration of the symptoms. The hypertrophy terminates abruptly, the duodenum being normal.

Clinical Features.—Characteristically first-born male infants are most commonly affected.

The peak incidence for the onset of symptoms is between the third and sixth weeks of life; however, the condition can commence before that time, and as late as the seventh week. The pillars upon which the diagnosis rests are:

Vomiting is the presenting symptom in all cases and within two or three

days it becomes forcible and projectile. Bile is not present in the vomit. Immediately after vomiting the baby is often very hungry.

Visible Peristalsis.—After the child has been fed, peristaltic waves may be seen passing from left to right across the upper abdomen (fig. 858). A good light is essential for this; the abdomen should be watched throughout a feed until vomiting occurs.

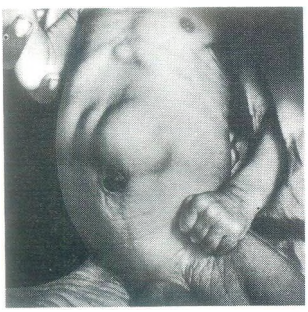


FIG. 858.—Visible peristalsis in hypertrophic pyloric stenosis: it is unusual for the waves to be so obvious. (*Alan Kark, F.R.C.S., London.*)

The Presence of a Lump.—The palpation of the hypertrophied pylorus is the most essential step in reaching a diagnosis. The surgeon should sit on the left side of the child and palpate under the liver with a warm hand. It may be helpful to examine the child more than once. The lump is most easily felt when the child is given a feed.

Constipation is usually present, and when a stool is passed it is small and dry resembling those of a rabbit. It is important to ask the mother about napkins; if the child is dehydrated, they are not wet and the case is correspondingly more urgent.

Loss of Weight.—One of the most striking signs of infants suffering from hypertrophic pyloric stenosis is loss of weight. Moreover, it is not long before the infant begins to look emaciated and dehydrated.

Often a change from one type of feeding to another brings about a remission. Consequently a series of changes in diet are sometimes made before the diagnosis is established, by which time the infant's condition may be pitiable.

In premature infants, in whom the condition is not uncommon, the symptomatology is often paradoxical. There is anorexia instead of a voracious appetite; the vomiting is regurgitant rather than projectile, and so frequently is peristalsis normally visible in these attenuated subjects that its significance is liable to be disregarded. None the less, amidst this sea of bewilderment one diagnostic rock remains—a hypertrophied pylorus can be felt through the poorly-developed abdominal wall with comparative ease.

Radiography.—The diagnosis should be made on a careful clinical examination and an X-ray is rarely necessary. To be diagnostic, a barium meal should show persistent narrowing and elongation of the pyloric canal.

Differential Diagnosis.—Conditions from which hypertrophic pyloric stenosis must be differentiated are (*a*) intracranial hæmorrhage; (*b*) duodenal atresia; (*c*) high intestinal obstruction, e.g. volvulus neonatorum (Chap. 50). In (*b*) and (*c*) bile will be present in the vomitus.

Treatment.—The majority of pædiatricians believe that surgery is the treatment of choice: the condition is immediately and completely corrected and the child is well within twenty-four hours. Babies who are admitted in a dehydrated condition should receive sufficient half-strength dextrose-saline solution, given subcutaneously with hyaluronidase, to restore the fluid balance. This results in a remarkable improvement in the general condition:

the sunken eyes and cheeks and depressed fontanelle fill out; the dry skin and mucous membrane become moist, and the output of urine increases.

Medical Treatment

In cases complicated by infection, especially of the mouth, medical treatment is advisable because of the increased risk of post-operative gastro-enteritis, even if operation would otherwise be indicated.

In subacute cases the patient often reaches the age of two months before the symptoms become obvious and if, as a result of medical treatment, the gastric residue decreases in amount, a cure by non-operative means can be anticipated.

Eumydrin (atropine methylnitrate) 1:10,000 of water, freshly made, is given half an hour before each feed, beginning with 0·5 to 1 ml. and increasing to 2·5 ml. to control the symptoms. Toxic symptoms (erythema and hyperpyrexia), though less common than when atropine is employed, do occur with Eumydrin[1], and are signs that the dose of the drug should be decreased or its use discontinued. Small, frequent feeds of milk (if possible the mother's milk), diluted with 5% dextrose, are given. Eumydrin must never be given until a firm diagnosis has been made.

Surgical Treatment.—After correction of dehydration, operation is performed without delay. The recovery rate approaches 100 per cent in early cases.

Ramstedt's Operation.—*Preliminary Preparation.*—The stomach is washed out with saline several times; finally, one hour before operation. Immediately before operation gastric aspiration must be performed, and continued throughout the operation. The prevention of chilling is of great importance. To this end the temperature of the operating theatre should be high (27°C) and the infant's body is encased in wool, the upper abdomen alone being accessible.

Operation.—Local anæsthesia is advisable unless the child is in good condition and the services of an anæsthetist skilled in anæsthesia of infants are available. The abdomen is opened by a grid-iron[2] incision in the upper right quadrant of the abdomen. The hypertrophied pylorus is delivered and rotated so that its superior surface comes into view; thus the least vascular portion (fig. 859) can be selected for the incision. In order to ascertain the distal limit of the hypertrophied pylorus the surgeon invaginates the duodenum with his index finger. The incision is made through the serosa only from this point along the whole length of the lump (fig. 860). Because the hypertrophied pylorus is the consistency of an

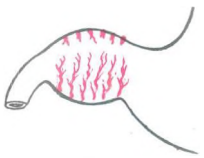


FIG. 859. — The upper part of the hypertrophied pylorus is comparatively bloodless.

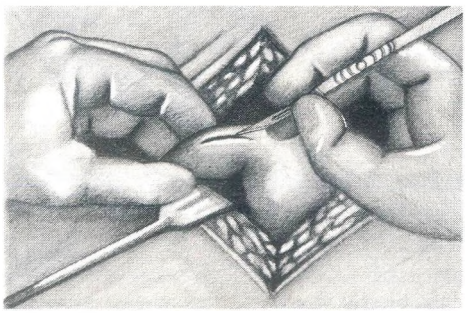


FIG. 860.—Ramstedt's operation.

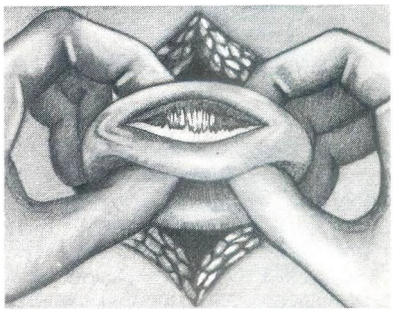


FIG. 861.—Showing the hypertrophied muscle divided and the mucous membrane bulging into the incision.

[1] Eumydrin was introduced as a therapeutic improvement on atropine in the treatment of congenital pyloric stenosis by Svensgaard in 1935.

[2] Transverse through the skin and the anterior rectus sheath, and splitting the rectus muscle vertically.

Elisabeth Svensgaard, 1894–1952. Physician, Rigshospitalet, Copenhagen.
Wilhelm Conrad Ramstedt, 1867–1963. Surgeon, Rafael Clinic, Munster, Germany. Performed his first pyloromyotomy in 1911. He discovered in church records that his grandfather had misspelt the family name with two 'm's, so in 1920 he reverted to the original spelling.

unripe pear, splitting of muscle coats can be accomplished with a blunt dissector. On separating the edges with artery forceps, the pyloric mucosa bulges into the cleft which has been made in the muscle (fig. 861). Great care is taken not to penetrate the mucosa, an accident which is liable to occur while dividing the most distal part of the constricting fibres which are in the vicinity of the duodenal 'fornix' (fig. 857). In order to be sure that there is no perforation, some air is squeezed from the stomach into the duodenum. If a perforation has occurred, it is closed by a wisp of omentum held in place by three or four interrupted sutures of fine silk (fig. 862). The free entry of air into the duodenum indicates adequate division of the muscle. Hæmostasis must be meticulous. The abdominal incision is repaired. It is important to remove the wool from the infant's body as soon as the operation is over, or hyperthermia may occur.

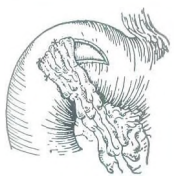


FIG. 862.—Patching a perforation of the mucous membrane. (*After C. E. Welch, Boston, Mass.*)

After-treatment.—A small feed of not more than 5 ml should be given one hour after operation, and after that the feeds can be rapidly increased. The child should be feeding normally in forty-eight hours and if there is no vomiting at this time, it is the practice in many hospitals to send the child home and bring it back for removal of stitches. If the mucosa was accidentally opened at operation, it is wise not to feed the child orally for forty-eight hours.

Complications:

(*a*) *Post-operative pyrexia* is rather common and is not necessarily of serious consequence. If protracted, tepid sponging is advisable.

(*b*) *Gastro-enteritis* is minimised by absolute cleanliness of feeding utensils, segregation and sending the child home as soon as convenient.

(*c*) *Disruption of the wound* is also rare, and is more liable to occur in emaciated subjects. The use of the grid-iron incision in these cases has reduced the incidence of this complication.

Hypertrophic pyloric stenosis of adults is a definite entity and is often mistaken for carcinoma of the pylorus. When a diagnosis can be made with certainty, pyloroplasty or a gastro-jejunostomy (p. 833) gives excellent results; when doubt exists, a Billroth I gastrectomy is done. Microscopical examination of the specimen shows fibrosis of the myenteric plexus (contrary to the corresponding condition in the infant).

CONGENITAL ATRESIA OF THE DUODENUM

There is stenosis, usually complete, across the duodenum (fig. 863). This occurs at the point of fusion of the fore- and mid-gut, and consequently lies in the neighbourhood of the ampulla of Vater. It is frequently accompanied by other congenital defects. The infant vomits *from birth*, and daily rapidly loses weight. In contradistinction to congenital pyloric stenosis *the vomit contains bile*. Laparotomy should be undertaken without delay. Duodeno-jejunostomy is the best procedure when, as is usually the case, the obstruction is in the second or third part of the duodenum (Ladd). In these cases the stoma is difficult to keep open and it is wise to introduce a fine plastic tube through a gastrostomy opening, through the stomach across the stoma and down into the jejunum for a few inches. This should be retained for several days for feeding purposes.

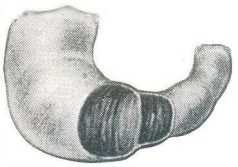


FIG. 863.—Congenital septal duodenal obstruction at the commencement of the third part of the duodenum. The gut above is enormously dilated. (*After W. E. Ladd.*)

PEPTIC ULCERS

There are two main types—gastric and duodenal. Either may present in an acute or chronic form.

Christian Albert Theodor Billroth, 1829–1894. Professor of Surgery, Vienna.
Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg, Germany.
William Edwards Ladd, 1880–1967. Professor of Child Surgery, Harvard University, Boston, Massachusetts, U.S.A.

Acute Peptic Ulcers

Ætiology.—Acute gastric ulcers are thought to be due to disruption of the gastric mucosal barrier. They appear as multiple erosions and the gastric mucus presents as large white globules instead of the usual opalescent fluid. About half the patients give a history of ingestion of aspirin or butazolidine. Classically they present with hæmorrhage.

Pathology.—Acute peptic ulcers are frequently multiple—in 75 per cent. of cases more than three of these lesions are present. They can occur in any part of the stomach, but in the duodenum they are almost confined to the first part. These ulcers are oval or circular in shape, and vary in size from 1 to 2 mm in diameter (when they are called erosions) to 1 or more cm in diameter. They are shallow, punched out, and seldom invade the muscular coats. When healing occurs, acute peptic ulcers are unlikely to leave scars.

Clinical Features.—Acute peptic ulcers probably occur with great frequency. They give rise to short-lived attacks of dyspepsia which are not diagnosed, and the ulcer heals. Usually they are recognised only when they cause hæmatemesis. Occasionally an acute ulcer, particularly when it is situated on the anterior wall of the duodenum, perforates. By gastroscopy, it has been ascertained that acute peptic ulcers can be the cause of hæmatemesis at all ages and in both sexes. Such lesions have been seen to progress to chronic ulceration (Tanner).

Treatment.—If possible, the cause must be removed. Under medical treatment acute peptic ulcers tend to heal rapidly. Blood transfusion may be required for hæmatemesis. Dietetic irregularities must be corrected in order to prevent recurrence or chronicity.

Stress Ulcers.—These constitute a special group of acute ulcers. They occur most frequently in patients in Intensive Therapy Units. There is usually a history of hypotension from hæmorrhage, endotoxin shock or cardiac infarction. Sometimes they are seen after cerebral trauma or neurosurgical operations (Cushing's ulcer) but it has been held that these are not true stress ulcers. After major burns two types of ulcer occur (Curling's ulcer). Within the first 48 hours multiple acute erosions may develop anywhere in the body and fundus of the stomach (14% of cases). The antrum and duodenum are unaffected. These are thought to be true stress ulcers. Later, during convalescence acute duodenal ulcers may occur (9%), which sometimes become chronic. These are probably not stress ulcers.

As to ætiology, there may be mucosal ischæmia due to vascular shunting, as well as reflux of duodenal contents into the stomach. The output of gastric mucosubstances is also increased and there is shedding of the surface epithelial cells all of which predisposes to ulceration. Patients on steroids may develop similar lesions, sometimes referred to as *steroid ulcers*.

CHRONIC PEPTIC ULCER

Ætiology.—We do not know why some acute ulcers become chronic. Many theories have been put forward but none is a satisfactory explanation for all the various forms of the lesion.

Incidence.—Peptic ulcer, rare before the age of sixteen, becomes more frequent as middle age approaches, and in England nearly 10 per cent. of men aged forty-five to fifty-four years are thus afflicted. Occupation has some bearing on the condition; thus significantly high incidences are found among doctors, foremen and business executives, while significantly low incidences occur among agricultural workers and sedentary workers, e.g. clerks, civil servants and draughtsmen (Doll). The popular belief that bus-drivers are especially liable to this condition has not been substantiated. In many parts of the world, e.g. U.S.A., Scandinavia, this disease is as rife as it is in Great Britain. Conversely, the incidence among Africans and Asiatics living in their native lands is comparatively low, except in the newly emerging nations where the local population are assuming increasing responsibility. Duodenal

Norman Cecil Tanner, Contemporary. Formerly Surgeon, St. James's Hospital, Balham, London.
Harvey Cushing, 1869–1939. Professor of Surgery, Harvard University, Boston, Mass., U.S.A.
Thomas Blizard Curling, 1811–1888. Surgeon, The London Hospital. Described this ulcer in 1842.
Sir William Richard Shaboe Doll, Contemporary. Formerly Regius Professor of Medicine, Oxford, England.

ulcer is found four times more commonly than gastric ulcer in patients under the age of thirty-five years, but after forty-five years of age it is only one to two times more common. In Scotland the ratio of duodenal to gastric ulcer is higher than in England, while in India the ratio of duodenal to gastric ulcers is 30:1.

All over the world the incidence of peptic ulcer in women is much lower than in men, and in women the two types of ulcer are about equal. Chronic gastric and duodenal ulcers not infrequently co-exist. In such instances the duodenal ulcer appears first.

Gastric Ulcer.—Chronic gastric ulcer is associated with either normal or hyposecretion, atrophic gastritis, and usually occurs in a later age group. It is a disease of the labouring classes rather than those subject to stress and responsibility. Frequently the patient has dental trouble.

Duodenal Ulcer.—Most patients, although by no means all, have gastric hypersecretion of acid. Duodenal ulcer patients tend to have a larger than normal parietal cell mass.

(*a*) *Genetic and Blood Groups.*—There is definite evidence that chronic ulcers occur in families. Moreover, persons of blood group O are about three times more likely to develop a peptic ulcer than are persons of other blood groups. It seems possible that the ABO genes may modify the size of the parietal cell mass.

(*b*) *Neurogenic Theory.*—Stimulation of the vagus results in gastric hypersecretion and hypermotility. Stress and anxiety are undoubtedly a leading cause of duodenal ulcer and these probably exert their effect via the vagus.

(*c*) *Accessory Causes.*—Inadequate mastication, alcohol, irregular meals, excessive smoking, and vitamin deficiency have, at one time or another, been blamed. It has been conclusively shown that smoking delays healing in gastric ulcers.

(*d*) *Endocrine.*—The effects of emotional, as well as physical stress are hormonally transmitted to the stomach via the pituitary-adreno-cortical axis (fig. 684). There are specific endocrine disorders which may be associated with severe or intractable ulceration. These include (i) the Zollinger-Ellison syndrome, where a non-beta cell tumour secreting 'gastrin' occurs in the pancreas (Chap. 47); (ii) the multiple adenoma syndrome, where adenomas occur in the pituitary, adrenal, pancreatic, and parathyroid glands (p. 647); (iii) hyperparathyroidism (p. 647).

Pathology.—Ulcers, whether gastric or duodenal, tend to occur in alkaline mucosa. The ulcer-bearing area is shown (shaded) in fig. 864. This

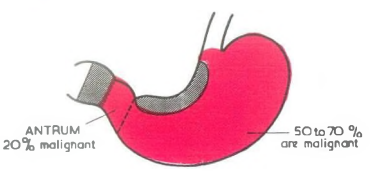


FIG. 864.—*Shaded Area.*—The common area for benign ulcers in stomach and duodenum. *Red Area.*—In this area ulcers carry a high risk of malignancy.

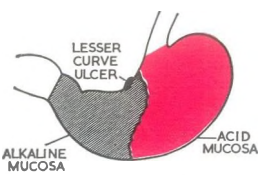


FIG. 865.—Area of gastritis (alkaline) in gastric ulcer. Red area = acid mucosa.

area is much smaller than the area of atrophic gastritis (fig. 865) which is the precursor of gastric ulcer. The ulcer itself occurs in the alkaline mucosa of the atrophic area adjacent to the acid-secreting fundic mucosa. A chronic peptic ulcer invades the muscular coats, which it tends to penetrate. When a

gastric or duodenal ulcer heals, the site is covered by a mucosal scar. Fibrosis, the result of recurrent ulceration, causes deformities, including pyloric stenosis and hour-glass contracture.

Chronic Gastric Ulcer is usually larger than a chronic duodenal ulcer. It varies in size, but in a well-established case it will admit the tip of a finger. The floor of a chronic gastric ulcer is situated in the muscular coats of the stomach, and as time goes on the ulcer occupying the posterior wall becomes adherent to, and later invades (chronic perforation), the pancreas. In the same way a chronic ulcer situated on the antero-superior aspect of the stomach can penetrate the liver, while a saddle-shaped ulcer situated on the lesser curvature can, and often does, penetrate both the liver and the pancreas.

Chronic Duodenal Ulcer.—The ulcer is nearly always situated in the first part of the duodenum, and sometimes two ' kissing ' ulcers are present: one on the anterior surface and one on the posterior surface of the first inch of the duodenum. An anterior ulcer may perforate, a posterior one carries the risk of secondary hæmorrhage by erosion of a large vessel.

Microscopical Examination.—There is nearly always greater destruction of the muscular coat than of the mucosa. The base of the ulcer is covered by a thin layer of granulation tissue. The arteries in the neighbourhood show evidence of endarteritis obliterans. Often there are no nerves in the floor of the ulcer but always many in the edge. The terminations of these nerves are bulbous, akin to those in an amputation stump (Kinsella). At the margin of the ulcer there may be epithelial proliferation, and downgrowths of glandular tissue are apt to be found, which are sometimes misinterpreted as indicating a carcinomatous change (Newcomb).

Do Peptic Ulcers become malignant?—A chronic duodenal ulcer never becomes carcinomatous. On the other hand, a chronic gastric ulcer may become malignant, but how frequently this change takes place is a matter of great difference of opinion (see above). It would seem probable that it does not exceed 0·5 per cent. Even **giant ulcers** (those with a crater of more than 2·5 cm. in diameter) are seldom carcinomatous.

Pathology at Operation.—A chronic peptic ulcer may present as a white scar under the peritoneal coat. Delicate vascular adhesions, salmon pink and fluffy in appearance, can often be observed in the immediate neighbourhood of the peritoneal aspect of the ulcer. At other times the ulcer must be sought for by palpation; induration, frequently extensive in the case of a gastric ulcer, is centred over the mucosal lesion. When the ulcer is situated in the duodenum, the surrounding induration is not so evident, but if the ulcer is situated on the posterior wall it may be possible to feel the crater with the tip of the finger. A useful method of confirming the presence of a peptic ulcer, particularly one situated on the anterior wall of the duodenum, is to rub the peritoneal surface gently with a swab; the peritoneum overlying the ulcer becomes speckled, as though sprinkled with cayenne pepper (fig. 866), a characteristic phenomenon due to minute petechial hæmorrhages.

If there is any doubt about the presence of an ulcer, a longitudinal gastrotomy incision should be made and the mucosal area inspected. Ulcers in the pyloric channel (p. 840), and around the cardia, may be missed if this is not done.

Victor John Kinsella, Contemporary. Consulting Surgeon, St. Vincent's Hospital, Sydney, Australia.
Wilfrid Davidson Newcomb, 1889–1971. Professor of Pathology, St. Mary's Hospital, London.

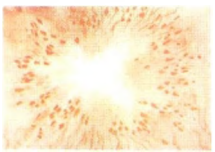


FIG. 866.—Petechial hæmorrhages around the peritoneal aspect of a chronic peptic ulcer. These become more obvious after gently rubbing the surface with gauze.

THE CLINICAL FEATURES OF GASTRIC AND DUODENAL ULCERS CONTRASTED

It is useful to record the patient's history under seven headings.

Chronic Gastric Ulcer.—The patient is usually beyond middle age, and by reason of a restricted diet is often thin. In many instances the patient appears anæmic, and this is often confirmed by a hæmoglobin estimation. On careful enquiry certain features of the dyspepsia become manifest. Typically there is:

1. *Periodicity.*—The attacks last for several weeks, and are followed by intervals of freedom from two to six months. The attacks are more in evidence in the spring and autumn and should be recorded.

2. *Pain* is epigastric, and may occur immediately, or any time up to two hours, after food. Pain may radiate through to the back, is relieved by lying down flat and practically never occurs at night.

3. *Vomiting.*—In over 50 per cent. of cases vomiting is a notable symptom. It relieves the pain, and may be self-induced.

In the 'pyloric channel' ulcer vomiting may be the predominant or only symptom and the barium meal may be negative. Endoscopy is essential in order to make the diagnosis. Pyloric channel deformity may be the precursor of a lesser curve gastric ulcer.

4. *Hæmatemesis and Melæna.*—At some time or other 30 per cent. of patients with gastric ulcers suffer from bleeding from the ulcer. The ratio of hæmatemesis to melæna is about 60:40.

5. *Appetite* is good, but the sufferer is afraid to eat.

6. *Diet.*—The patient learns to avoid fried foods, stews, curries, and twice cooked meat. Milk, eggs, and fish are the staple diet.

7. *Weight.*—Usually by the time the surgeon is consulted there has been some loss of weight.

On examination there is frequently deep tenderness in the mid-line of the epigastrium a few inches above the umbilicus.

Chronic duodenal ulcer can occur at any time during adult life, but is commonest between the ages of twenty-five and fifty. It is more common in men, who appear otherwise healthy.

1. *Periodicity* is usually well marked, and classically the attacks come on in the spring and in the autumn and are precipitated by 'work, worry, or weather'. These attacks usually last from two to six weeks, with intervals of freedom from one to six months.

2. *Pain* is severe, and may double the patient up. It usually occurs one to two and a half hours after food. As it is often relieved by food, the pain is known as 'hunger pain', and, classically, the patient carries biscuits, which he eats at frequent intervals to prevent this gastric torment. The pain, which is also relieved by alkalis, often awakens the patient round about 2 a.m. but is usually absent at the normal hour of rising.

Berkeley George Andrew Moynihan (Lord Moynihan of Leeds), 1865–1936. Professor of Clinical Surgery, Leeds, England, was the first to describe 'hunger pain'.

3. *Vomiting* is rare in duodenal ulceration unless it is self-induced or stenosis has occurred. Regurgitation of burning fluid into the mouth (' water-brash ') together with pain deep to the sternum (' heart-burn ') due to reflux œsophagitis are common complaints (1 : 10).

4. *Hæmatemesis and melæna*, which occur in the ratio of 40 : 60, but sometimes together, are rather more frequent than in the case of gastric ulcer.

5. *Appetite* is exceptionally good, but the patient sometimes refrains from eating solid food during the attacks.

6. *Diet.*—In contradistinction to patients with a chronic gastric ulcer, those suffering from duodenal ulcer who have not been ordered a special diet seldom display much dietetic discrimination, although some of the more intelligent find it advisable to avoid fried food.

7. *Weight.*—Usually there is no loss of weight; indeed, the patient often becomes plump (partly because of the ingestion of milk to relieve pain).

On examination it is not unusual to find localised deep tenderness in the right hypochondrium.

After about two years the course of the disease becomes steady. There is 1 per cent. risk of perforation and 3 per cent. risk of massive bleeding—1 in 4 require surgical intervention in four to five years. An ulcer that starts with bleeding or perforation is likely to do it again.

Summary

	Gastric Ulcer	*Duodenal Ulcer*
Periodicity	Present	Well marked
Pain	Soon after eating, but not when lying down	Two hours after food Night pain
Vomiting	Considerable vomiting	No vomiting
Hæmorrhage	Hæmatemesis more frequent than melæna	Melæna more frequent than hæmatemesis
Appetite	Afraid to eat	Good
Diet	Lives on milk and fish	Eats almost anything
Weight	Loses weight	No loss in weight

SPECIAL METHODS OF INVESTIGATION

Barium Meal.—The radiological findings are often conclusive. In a lesser curve *gastric ulcer* a niche will be seen projecting from the usually smooth outline (fig. 867). The stomach is often hypotonic and J-shaped and hangs low in the pelvis. In *antral ulcer* the appearance of the niche is similar (fig. 868). *Anastomotic ulcers* can also be shown (fig. 869) although only about 30% can be demonstrated. In *duodenal ulcers* diagnosis depends on demonstrating an ulcer crater filled with barium—this is positive evidence of an ' active ' ulcer (fig. 871). In longstanding ulcers so much scar tissue is present that it is often impossible to show an ulcer crater with a persistent flake of barium. Where, however, deformity is *constant* the inference is that there has been a chronic cicatricial process due to an ulcer. In such a case the folds of scar tissue converge on the ulcer site, and if this ' *rugal convergence* ' can be shown, there is a very strong suspicion that an ulcer is present. It is important to recognise the shape of the normal duodenal cap when distended with barium (fig. 870). The appearances of pyloric stenosis (fig. 889) and hour-glass deformity (fig. 890) are characteristic.

Blood Studies.—A hæmoglobin estimation may show evidence of chronic blood loss. A raised erythrocyte sedimentation rate in a gastric ulcer suggests malignancy.

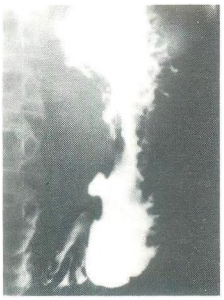
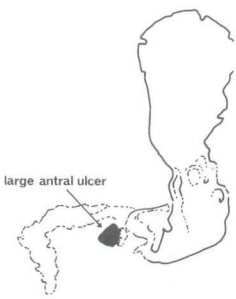


FIG. 867.—Typical gastric ulcer on the lesser curve.

FIG. 868.—Antral ulcer.



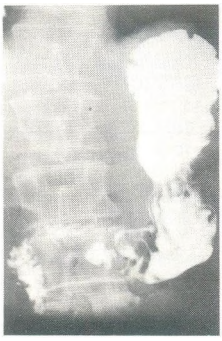


FIG. 869.—Anastomotic ulcer.

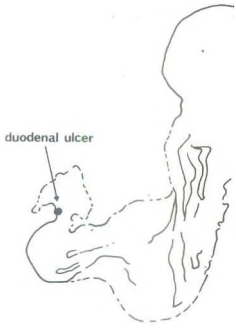


FIG. 871.—Duodenal ulcer.

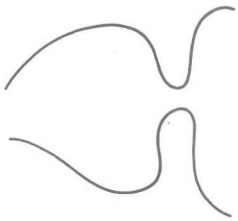


FIG. 870. — Normal duodenal cap filled with barium.

Stool Studies.—Examination for occult[1] blood is important and should be done routinely in such cases on the specimen of fæces on the finger-stall at rectal examination. If the patient has recently ingested meat or aspirin, or has used a stiff tooth brush, a false positive may be obtained.

Studies of Gastric Function.—These are described in the section on gastric physiology on p. 811.

Gastroduodenoscopy requires special training and considerable experience but affords valuable information. By its employment the whole of the interior of the stomach can be adequately scrutinised by the eye of the gastroscopist (fig. 872).

FIG. 872.—Flexible Lo-Presti œsophago-gastric fibrescope with terminal lens, biopsy channel and camera. (*A.M.S.C. and M.S.A. Ltd.*)

The fibrescope is an instrument in which glass fibre provides the lens system. It is thus flexible and the technical difficulties associated with the introduction of earlier gastroscopes are eased. It can be used to inspect the duodenal bulb (Hirschowitz) and to see beyond a gastro-duodenal anastomosis.

Following the development of the gastrocamera in Japan there is now a wide range of endoscopes which allow biopsies to be taken under direct vision. Photographs may be taken by still or cinecamera and television methods.

Indications.—Gastroduodenoscopy is valuable in the diagnosis of shallow gastric ulcers that do not show on radiography, in checking the results of medical treatment in cases of chronic gastric ulcer, in the differential diagnosis between a chronic peptic ulcer (fig. 873) and a carcinoma (fig. 874), in the diagnosis of a small gastric neoplasm,

FIG. 873.—Gastroscopic view of the lesser curvature showing a gastric ulcer above the entrance to the antrum. (*W. W. Davey, F.R.C.S.*)

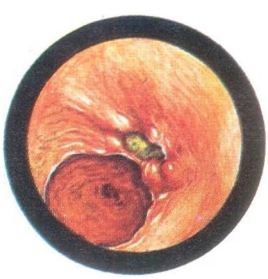


FIG. 874.—Gastroscopy — early carcinomatous change in a chronic gastric ulcer in a patient undergoing medical treatment. (*Hermon Taylor, F.R.C.S., London.*)

[1] Occult = hidden.

Basil I. Hirschowitz, Contemporary. Professor of Medicine, University of Alabama, Birmingham, U.S.A.

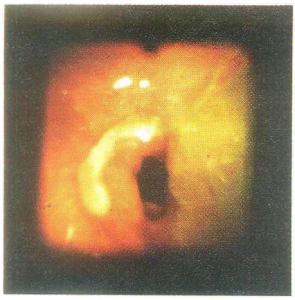


FIG. 875.—Benign prepyloric ulcer.

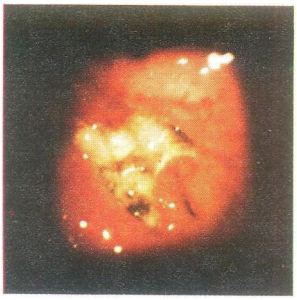


FIG. 876.—Neoplastic ulcer involving the pylorus.

in the detection of certain forms of gastritis, in the examination of the stoma in cases of suspected gastrojejunal ulcer, and in the diagnosis of duodenal ulcer.

TREATMENT OF CHRONIC PEPTIC ULCER

CONSERVATIVE MANAGEMENT

All are agreed that in the absence of complications threatening life, the treatment of a proven chronic gastric or duodenal ulcer should in the first instance be conservative (medical). There is no doubt that the patient benefits from close and continuing collaboration between surgeon and physician, and that this collaboration is improved when each has a clear understanding of both conservative and surgical therapy. The management of the two conditions is similar in some respects, but there are important differences.

Aims of Conservative Treatment

1. The aim of treatment for *gastric ulcer* is the *healing of the ulcer*; in the absence of a clear understanding of the pathogenesis of the lesion, the regime consists of the elimination of possible aggravating factors, and the creation of a bland intragastric milieu.

2. By contrast, the aim of treatment for *duodenal ulcer* is the *relief of symptoms*; this is often, but not always, synonymous with healing; typical symptoms may persist in the absence of a detectable lesion, while a frank ulcer may be virtually asymptomatic. Since gastric acid is the main provocative factor in duodenal ulcer disease, the control of acid secretion is the essential therapy. The introduction of drugs which selectively and specifically block acid secretion has increased the effectiveness of conservative therapy without, so far as can be ascertained, altering the natural history of the chronic disorder.

Chronic Gastric Ulcer.—Prior to the institution of treatment, accurate diagnosis is essential. Endoscopic inspection and biopsy of the ulcer must be performed; in general, the size of the lesion is best assessed radiologically. Once the lesion has been confirmed as benign, symptomatic treatment is instituted. Although secretion of acid may be subnormal, regular antacids between regular meals may be helpful. Provocative agents should be avoided, including chemical irritants such as aspirin, corticosteroids, phenylbutazone, and alcohol. Cigarette smoking should be stopped as it is known to delay gastric healing. There is no specific ulcer diet, but foods which provoke symptoms should be avoided. The status of specific therapy is uncertain, but carbenoxolone appears to accelerate ulcer healing; because it promotes fluid retention, it should be used with caution in the elderly.

Six weeks after the institution of treatment, objective evidence of healing must be obtained. A barium meal is the simplest test, as comparison with the original films gives a good estimate of partial healing even when resolution is incomplete. If there is no clear evidence of healing, four-quadrant endoscopic biopsy is essential, and if doubt persists, a Billroth I gastrectomy is indicated.

Chronic Duodenal Ulcer.—Even though the ætiology of duodenal ulcer in re-

lation to acid secretion remains a matter of controversy, there is no doubt that (1) active duodenal ulcer disease is often associated with increased gastric acid secretion and (2) that the pain of duodenal ulcer arises from the contact of acid with the lesion. Thus, for duodenal ulcer, the control of gastric acid secretion is logical treatment. It has to be remembered that spontaneous healing of duodenal ulcers within 6 weeks is usual, and the aim of treatment is the abolition of pain during the healing phase as well as avoiding possible perpetuation of the ulcer by acid. Traditional doses of antacid are helpful in the immediate relief of symptoms, but fall far short of the doses required to buffer secreted acid. Large doses of antacid (such as 120 ml daily of an aluminium and magnesium hydroxide mixture) will significantly neutralise gastric acid but are not very acceptable, partly because of a high incidence of diarrhœa on this regime.

H2-Receptor Antagonists—Cimetidine.—At the parietal cell level, acid secretion is mediated by histamine acting on H2 histamine receptors. Recently, drugs have been developed which specifically block these receptors, without other significant effects. The first such drug to be introduced is cimetidine (Black), and its effect is virtual abolition of resting (or basal) acid secretion and, in normal therapeutic dosage, reduction by half of meal or gastrin stimulated acid secretion. The timing of doses is related to the timing of maximum risk to the lesion. Acid secretion is stimulated by a meal, but the meal itself will buffer the acid for about one hour after food. After that time, continuing secretion induces a fall in gastric pH, and it is then that the pH in the duodenal bulb falls. Although the fall in pH will cut off acid secretion by negative feedback, duodenal ulcer patients in particular tend to have defective auto-regulation, with prolongation of the acid secretion after the buffer capacity of the meal is exhausted (Malagelada). It is thus logical to give cimetidine tablets after each meal to give maximal plasma levels 1–2 hours after food, and also before sleep, to ensure the reduction of nocturnal fasting secretion.

Aspects of Management

General and Specific.—The diagnosis is made on the basis of X-ray findings (of which deformity of the duodenal bulb is the most characteristic) and the presence of symptoms. Endoscopic confirmation of an ulcer crater is not indicated unless the diagnosis remains in doubt. The same general lines of treatment follow as for gastric ulcer in terms of dietary modification and avoidance of irritants. There is no evidence that smoking delays healing of duodenal ulcer, but reduction or cessation of cigarette smoking often alleviates persistent symptoms. Specific treatment consists of cimetidine 200 mg after meals and 400 mg at night for 4–6 weeks. A further period of 400 mg at night only may be helpful if some symptoms persist.

Relapses.—Most relapses in case of chronic duodenal ulcer disease will respond to the standard regime. As there is no evidence that H2-receptor antagonists alter the natural history of the disease, relapses may occur as expected, and two or three courses of treatment per year (' interval therapy ') may be necessary. Prolonged treatment (' maintenance therapy ') with a nocturnal dose only will keep symptoms at bay, and apparently prevent relapse. Since there is evidence that prolonged treatment may mask the symptoms of recurrent ulceration, complications of ulcer can occur on such a regime.

Prolonged Therapy.—In the present state of knowledge, there must be doubts about the wisdom of prolonged maintenance therapy; apart from the possibility of unwanted side-effects,[1] it is not always acceptable to patients, and it is also expensive. Where symptoms can only be controlled by such measures, assessment of the patient for elective surgery is advocated.

Warning.—Since duodenal ulcer disease may be lifelong, physicians must beware diagnostic complacency. Gallstone disease presents with similar symptoms, and ' ulcer ' symptoms which do not respond to antacids or H2-receptor antagonists in a chronic duodenal ulcer patient should always raise the suspicion that there is other pathology, and lead to appropriate investigations. In uncomplicated cases, where symptoms rapidly respond to treatment, repeated radiology and endoscopy is neither helpful nor necessary.

[1] Gynæcomastia and reduction in sperm count have been reported.

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J. R. Malagelada, Contemporary. Gastroenterologist, Mayo Clinic.

SURGICAL TREATMENT

Indications for Operation.—Where healing has not progressed under medical treatment or where there is any doubt about the histology, operation is indicated. Less frequently, the ulcer may be in the antrum or in the prepyloric region. Antral ulcers have a 10% and prepyloric ulcers a 20% chance of being malignant when they are first diagnosed radiologically. These ulcers therefore must be regarded with considerable suspicion, and endoscopy with four quadrant biopsy is mandatory. Biopsy, however, is often difficult to achieve from ulcers in this region and requires the services of a highly skilled endoscopist. Where such a person is not available, it is wise to submit these patients to operation, because of the high risk of malignancy. Medical treatment unless covered by biopsy evidence, is unjustified in such cases.

It is wise to let the duodenal ulcer patient ' earn ' his operation. If surgery is done early in the disease before he has had much pain or a complication, he will be likely to resent some of the sequelæ of surgery. In general, the following facts suggest the need for surgery:

(*a*) Intractable pain, or recurrence of pain with frequent loss of work, especially after previous perforation or hæmorrhage.

(*b*) Complications, e.g. pyloric stenosis, hour-glass deformity, perforation, or bleeding.

(*c*) In general, ulcers which have been present for five years are unlikely to heal (Watkinson). It has been suggested that the risks of operation approximate to the risks of having a peptic ulcer for five years.

Assessment for operation.—Since the advisability of operation, the type of operation, and additional operative procedures may be affected by the results of assessing the patient, it is important to note :

1. The age, sex and general condition of the patient, including the cardiovascular, respiratory and renal systems.

2. The presence of pyloric stenosis, bleeding, combined gastric and duodenal ulcers, hiatus hernia, gallstones and other gastrointestinal disorders.

Aims of operations.—Surgical treatment for peptic ulcers aims to cure the ulcers with the maximum effectiveness, the minimum danger and side effects. The variety of operations performed testifies to the lack of agreement among surgeons. Because duodenal and gastric ulcers are considered to be different disease entities, the aims of operation differ for each type of ulcer.

Gastric ulcer.—Gastric ulcer is not associated with high levels of gastric acid secretion.

1. Orthodox surgical treatment aims at excision of the ulcer-bearing area and the stomach distal to the ulcer,

2. Improving the emptying of the stomach.

3. Prevention of complications.

4. To ensure that malignant ulcers are removed and avoid the risk of such a change in benign lesions.

Duodenal ulcer.—1. Operation aims to reduce the acid contact with the

Geoffrey Watkinson, Contemporary. Physician, Western Infirmary, Glasgow, Scotland.

ulcer by diverting acid through a stoma as in gastroenterostomy, by removing part of the parietal cell mass as in gastrectomy, or by reducing stimuli to acid secretion by vagotomy and by antrectomy.

2. Correction of ill effects of the ulcer, such as pyloric stenosis, hæmorrhage, gastric ulcer.

3. Prevention of complications.

Mortality.—The average national mortality for gastrectomy is 2 per cent., but this figure is greater in the elderly. The mortality after vagotomy with a simple drainage procedure is 0·75 per cent. Gastrectomy is a more difficult operation and usually takes longer.

The sequelæ of gastrectomy, both immediate and remote, are now well established (p. 844). We are less sure of the remote sequelæ of truncal vagotomy.

Operations

For gastric surgery, the upper abdomen may be opened by a right paramedian incision, but if a vagotomy is contemplated, a left paramedian or mid-line approach is preferable. Having verified the diagnosis and ensured the absence of other pathology, the ensuing procedure differs according to the actual operation to be carried out.

Operations for gastric ulcer.—The classical operation is the Billroth I partial gastrectomy.

The stomach is mobilised by division of the main arteries as shown in fig. 850. The distal part, including the ulcer, is resected. The cut edge of the remnant is partially closed, leaving a stoma at the lower end which matches the lumen of the duodenum. The duodenum may be mobilised by Kocher's method, with division of the peritoneum around its convex border, allowing union of the stomach and duodenum without tension (fig. 877). The stoma should be as large as possible.

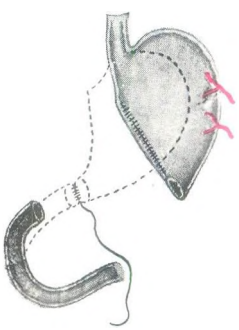


FIG. 877. — Billroth I operation. Dotted lines show rotation of the stomach, mobilisation of the duodenum, and the anastomosis in progress.

Selection of operations for gastric ulcer.

1. Billroth I partial gastrectomy is followed by recurrent ulcer in less than 1%. The resected ulcer is available for histological examination to confirm that it is benign.

2. Polya gastrectomy with resection of the stomach below a high benign gastric ulcer invariably results in healing of the ulcer. The operative difficulty, and consequent risk, is reduced. The post-operative nutritional state of the patient is better following a conservative resection.

3. Vagotomy and pyloroplasty has gained support as a conservative operation for benign gastric ulcer. If necessary, biopsy of the ulcer, with frozen section histology, should confirm the benign nature of the ulcer. The theoretical basis for employing this operation is hotly discussed. In both 2 and 3 an ulcer cancer may be missed.

Operations for Duodenal Ulcer

Historical

In January 1881, Billroth of Vienna performed the first successful gastrectomy, and in September of the same year Wolfler introduced the operation of gastroenterostomy. The Billroth operations consisted of gastric resection followed by gastro-duodenal anastomosis (Billroth I technique).

Four years later, in 1885, the Billroth II operation was introduced as a two-stage

Theoder Kocher, 1841–1917. Professor of Surgery, Berne, Switzerland.
Anton Wolfler, 1850–1917. Professor of Surgery, Prague, Czechoslovakia.

procedure, more by accident than design. A preliminary gastro-enterostomy was done on a gravely ill patient with a pyloric carcinoma. Contrary to expectations, the patient improved, and the stomach was resected, distal to the anastomosis. It soon became evident that the use of a gastro-jejunal anastomosis after gastric resection could be safer and easier than the Billroth I procedure. Now there are some forty modifications in technique of the Billroth II procedure—the main variations involving the use of an antecolic or retrocolic anastomosis, the direction of peristalsis in the jejunal loop, and the use of a full-width stoma or a restricted stoma with a 'valve'.

I. Polya Gastrectomy (Billroth II type)

The lesser sac is opened through the gastro-hepatic omentum and the left hand introduced behind the stomach. Holding the stomach in this manner, the blood vessels and omentum along the greater curvature are divided and ligated. It is safer to do this between the gastro-epiploic artery and the greater curve. It is important at this stage to see that damage is not done to the spleen itself. The dissection is carried on to the first part of the duodenum below, and up to include the lower short gastric vessels above. The right gastric artery is divided. The duodenum is then divided and closed by at least two layers of inverting sutures. Great care is needed to avoid damage to the common bile duct. With the stomach held up, the left gastric artery is now divided. The stomach is turned over to the left and a clamp is applied to permit removal of about 60 per cent. of the stomach. End-to-side gastro-jejunal anastomosis is now made, so that the afferent loop is as short as possible. This is achieved by a retrocolic anastomosis at the duodeno-jejunal flexure. The closure of the mesocolic leaf around the anastomosis is of paramount importance. There are still many surgeons who advocate an antecolic anastomosis. The addition of 'valves' offers no increased benefit. If the surgeon is not absolutely satisfied with the duodenal closure, a soft drainage tube should be put down to, but not touching, the duodenal stump (see p. 843).

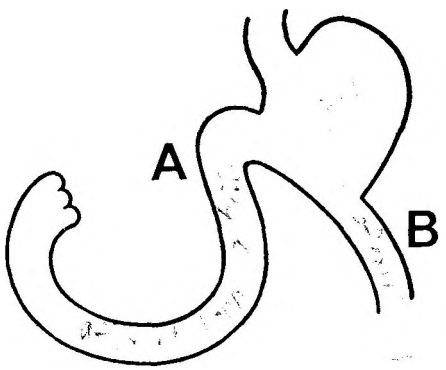


FIG. 878.—Polya type of gastrectomy. The duodenum is closed, and the first piece of jejunum is joined to the stomach remnant. A = afferent 'loop', B = efferent 'loop'.

II. Vagotomy (Vagus Nerve Section) with Gastric Drainage.—Vagotomy has the twofold effect of reducing hypermotility and hypersecretion of the stomach. Unfortunately it also causes gastric retention. It is therefore essential to perform some sort of 'drainage procedure' such as pyloroplasty or gastro-jejunostomy to correct this. Vagotomy moreover may be followed by severe episodic attacks of diarrhœa. This is less following (1) selective rather than total vagotomy and (2) drainage by pyloroplasty rather than by gastro-jejunostomy; but in any case vagotomy should be avoided in patients who suffer from diarrhœa.

The main varieties of vagotomy are as follows:

(*a*) *Total Abdominal Vagotomy* (fig. 879).—The abdomen is opened by a left upper paramedian incision, the diagnosis is confirmed, and the other abdominal organs are examined. The left lobe of the liver is mobilised by dividing the left triangular ligament close to its hepatic attachment. The peritoneum to the left of the œsophagus is incised and the right index finger encircles the œsophagus in order (*a*) to pass the soft rubber tube which is used for traction purposes, and (*b*) to identify the position of the posterior vagus trunk, which is thicker than the anterior nerve trunks, and lies almost entirely free from the œsophagus. With the œsophagus taut it is possible to make a thorough search for and divide all the anterior vagal fibres. Two centimetres of the posterior vagus are resected.

(*b*) *Selective Vagotomy* (fig. 879).—The operation is not widely used.

Eugen Alexander Polya, 1876–1944. Surgeon, St. Stephen's Hospital, Budapest, Hungary.

Both of the above operations are associated with gastric stasis and pylorospasm following the vagal denervation and hence a gastric drainage procedure must be carried out.

(*c*) *Highly selective vagotomy* (or parietal cell vagotomy) (fig. 879) is now increasing in popularity. This preserves the innervation of the pylorus and hence a drainage procedure is unnecessary. In this the anterior and posterior nerves of Latarjet are identified (fig. 852). Their terminal fibres reach the antral wall about 5–7 cm above the pylorus. They are preserved and with them the innervation of the pylorus. The stomach is now pulled to the left and its lesser curve laid bare from just above this point of entry of these nerves right up to the cardia. The main vagal trunks are now identified, drawn aside and the lower 5–7 cm of œsophagus is cleared circumferentially of nerve fibrils and vessels. The operation has not yet been widely adopted perhaps because of the anatomical difficulties of identifying these small vagal filaments.

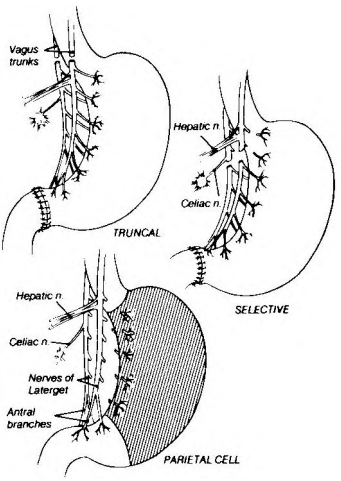


FIG. 879.—Diagrammatic representation of the three types of vagotomy. 1. Truncal or total intra-abdominal vagotomy. 2. Selective vagotomy. 3. Parietal cell or highly selective vagotomy. (*Dr. Paul H. Jordan, Houston, Texas.*)

GASTRIC DRAINAGE PROCEDURES

There are three main types:

(1) **Gastro-jejunostomy.**—The essential feature of this procedure is to establish an anastomosis at the most dependent part of the stomach, which is the greater curve on the posterior wall. A short-loop posterior operation with a vertical stoma extending to the greater curvature (fig. 880) is the type of operation that gives the best results. In this operation the stoma should be larger than in gastrectomy. Dragstedt and Tanner advocate an anterior anastomosis made in the pre-pyloric area. The risk of recurrent ulcer is less, and if it does occur, it is easier to deal with.

(2) **Pyloroplasty.**—The aim here is to produce free drainage by division of the pyloric ring. This can be done by two methods, that of Heineke-Mikulicz or that of Finney (figs. 881 and 882).

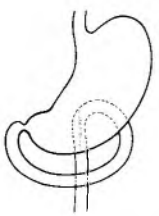


FIG. 880.—The posterior, vertical, short-loop retrocolic gastro-jejunostomy of Mayo.

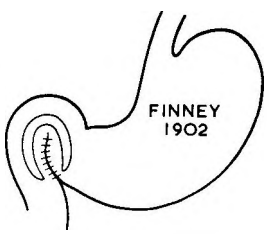


FIG. 881.—Finney pyloroplasty. The adjacent antrum and duodenum is approximated and an inverted U incision is sutured to give a wide gastro-duodenal anastomosis.

FIG. 882.—Heineke-Mikulicz pyloroplasty. A longitudinal incision of 6 cm. is closed transversely by a layer of interrupted silk sutures, reinforced by a large omental patch.

Walther Hermann Heineke, 1834–1901. Surgeon, Erlangen, Germany.
Johann von Mikulicz-Radecki, 1850–1905. Professor of Surgery, Breslau, Germany (now Wroclaw, Poland).
John Miller Turpin Finney, 1863–1942. Surgeon, Johns Hopkins Hospital, Baltimore, Maryland, U.S.A.

(3) **Antrectomy.**—In addition to acting as a drainage procedure, antrectomy also abolishes the hormonal phase of gastric secretion. A gastrojejunal anastomosis is made. The operation is combined with truncal vagotomy. Care must be taken that all of the antral mucosa is removed; hence in practice, a hemigastrectomy is done.

SELECTION OF OPERATIONS FOR DUODENAL ULCER

The mortality rates and recurrent ulcer rates following different operations for duodenal ulcer are set out in fig. 883. Simple operations carry a low mortality, but a high rate of recurrence. More complicated operations carry a higher risk to life

	Mortality	*Recurrence*
Gastrojejunostomy	Less than 1%	40%
Vagotomy + Gastrojejunostomy Vagotomy + Pyloroplasty	Less than 1%	5%
Highly Selective Vagotomy	Less than 1%	10%
2/3 Gastrectomy	2%	1–4%
Vagotomy + Antrectomy (hemigastrectomy)	2%	1%

FIG. 883.—Mortality and recurrent ulcer rates following operations for duodenal ulcer.

but are more effective in curing peptic ulceration. The operative risks are increased in elderly patients, in patients with concomitant disease, in those operated upon in emergency conditions because of bleeding. In contrast, all elective operations carry low risks in young, fit patients.

Gastrectomy.—Must be recognised to be an ablative procedure and is not much practised nowadays for chronic duodenal ulcer, the long-term sequelæ being regarded as too heavy a price to pay for a relatively benign condition.

Truncal Vagotomy plus a drainage operation is at present the most widely used procedure, but here the problem of postvagotomy diarrhœa, which is significant in up to 8% of patients and may be present in a lesser form in up to 30%, is a serious drawback (p. 847).

Selective Vagotomy.—Used very infrequently.

Highly Selective Vagotomy. Data on the long-term results are not yet available. After it, the Hollander test for incompleteness of vagotomy becomes positive within a year in over half. It must not be carried out if the pylorus is narrowed by scarring. Some advocate simple dilatation for this as part of the procedure. Although tedious to do, the patients seem to tolerate the operation easily and nothing is resected. If its results prove comparable to the more extensive operations described above, it seems likely to prove the operation of choice for chronic duodenal ulcer. There is evidence, however, that the recurrent ulcer rate may be up to 10%.

Vagotomy and Antrectomy.—Is regarded by some as a reasonable alternative and less mutilating than gastrectomy itself, but the mortality rate for this procedure (fig. 883) is the same although the long-term sequelæ are less.

Gastro-jejunostomy.—It would be remiss not to mention the great value of this operation as the *sole* procedure for the treatment of duodenal ulcer. It has been practised for the whole of this century and has given great benefit to hosts of patients. Its mortality is low, but unfortunately the ulcer-recurrence rate following this has steadily increased with time, e.g. it may be as much as 40 per cent. within twenty-five years after operation. It still has a value, however, in the elderly, especially women, with pyloric stenosis in whom the gastric acidity remains low after ten days thorough gastric lavage. Women do not do well after gastrectomy of the Polya type and rarely, if ever, should a woman be submitted to this operation for simple duodenal ulceration.

LOCAL COMPLICATIONS OF CHRONIC PEPTIC ULCER

Acute — Perforation; Hæmatemesis and/or melæna.

Intermediate.—Residual abscess.

Chronic — Stenosis — Pyloric stenosis; 'Tea-pot' deformity; Hour-glass contracture.
Penetration into neighbouring viscera, notably the pancreas.
Carcinoma (Gastric ulcer).

PERFORATED PEPTIC ULCER

Sex.—The ratio is 19 men to 1 woman (Illingworth).

Age.—The highest incidence is between forty-five and fifty-five years.

Most often a peptic ulcer that perforates is situated on the anterior surface of the duodenum; much less frequently it is situated on the anterior surface of the stomach, usually near the lesser curvature or the pyloric antrum. Rarely an ulcer on the posterior wall of the stomach perforates into the lesser sac. In 80 per cent. of cases there is a history—often a long history—of peptic ulceration. In 20 per cent. there is no such history; it is a 'silent' chronic ulcer that perforates, especially in those patients who are being treated with cortisone. Usually the symptoms of perforation occur with dramatic suddenness.

The gastric or duodenal contents escape through the perforation into the general peritoneal cavity, resulting in peritoneal irritation (peritonism). At that moment the victim cries out in agony, and, at any rate if the perforation is a large one and the stomach is full, he is riveted temporarily to the spot where the perforation felled him. The peritoneum reacts to this chemical irritation by secreting peritoneal fluid copiously and this gives relief of pain for a short time. This *stage of reaction* lasts from three to six hours, and is followed by diffuse bacterial peritonitis. Following a perforation, gastric secretion is abolished and it is this loss of the acid barrier which permits bacterial invasion to occur.

Clinical Features

1. *Massive perforation.*—In the early stage of peritoneal irritation, the patient is pale, anxious, and loath to move. The temperature may be subnormal but the pulse is raised to 80–90. The abdomen is held still, moving little or not at all with respiration (fig. 884). The whole abdomen is tender with board-like rigidity. It is dull to percussion. Sufficient gas may have escaped to reduce liver dullness in the mid-axillary line. Pelvic tenderness may be elicited on rectal examination.

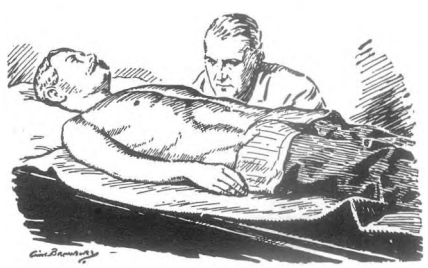


FIG. 884.—A sketch of Mr. Hamilton Bailey watching for abdominal movement on respiration. In cases of perforated peptic ulcer abdominal movement is restricted or absent.

Sir Charles Frederic William Illingworth, Contemporary. Emeritus Regius Professor of Surgery, Glasgow, Scotland.
Hamilton Bailey, 1894–1961. Surgeon, Royal Northern Hospital, London.

After three to six hours, the pain, tenderness, and rigidity may lessen. The temperature rises to normal or higher. However, the pulse remains high, the bowel sounds are absent. This temporary improvement has rightly been called the ' period of illusion '.

After six hours, the stage of diffuse peritonitis develops, accompanied by silent abdominal distention. Enough free fluid may have collected to be clinically detectable. The rising pulse rate marks the progressive deterioration in the patient's condition with each hour that passes without operative treatment.

2. *Subacute perforation.*—The pain may be less severe, less generalised with definite tenderness but equivocal guarding and rigidity when the perforation is small, and occurs in a robust, phlegmatic patient. Bowel sounds frequently persist. The small amount of fluid may track down the paracolic gutter, producing pain and tenderness in the right iliac fossa, simulating appendicitis. It is important to establish the site of onset of the pain.

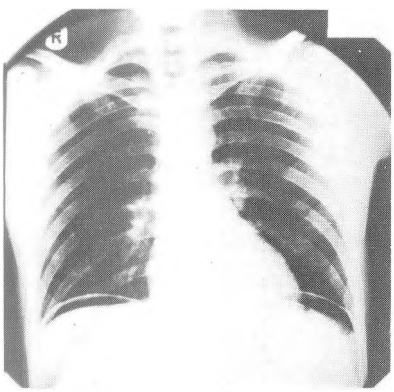


FIG. 885.—Plain X-ray of a perforated duodenal ulcer showing gas beneath the diaphragm. (*Dr. R. Vecht, Bristol.*)

Diagnostic aids.—A plain X-ray of the abdomen with the patient erect reveals a translucent area beneath the right cupola of the diaphragm in 70% of cases (fig. 885). Bile-stained fluid may be aspirated from the peritoneal cavity; it is alkaline to litmus.

Treatment.—Morphine should not be given until written permission for operation has been obtained. Operation, as soon as the general condition permits, is usually the best course. Laparotomy is performed and the typical œdema with a curious punched-out ulcer of the anterior wall of the juxtapyloric region is found. The perforation is closed with interrupted sutures reinforced with an omental patch. In the case of a gastric ulcer a biopsy *must* be taken as there is a risk of malignancy. In large perforations it is advisable to reinforce the suture line with a patch of omentum. *Thorough peritoneal toilet is essential.* With a mechanical sucker fluid and food debris are removed from the peritoneal cavity. Drainage of the peritoneal cavity is employed.

The immediate after-treatment consists of continuous gastric aspiration supplemented by intravenous fluid. Antibiotic therapy and breathing exercises are important in the elderly, and in cases where operation is more than six hours after perforation.

Pyloroplasty with vagotomy may be carried out only if the patient would merit elective surgery for the ulcer before the perforation occurred, if the perforation is recent, if the patient is fit, if the surgeon is experienced and backed by a fully trained supporting team (Kirk).

Follow-up of Patients after Perforation.—As might be expected, there is a transient remission of symptoms due to the rest in bed and the careful dietetic supervision during convalescence. Elderly patients and those of any

Raymond Maurice Kirk, Contemporary. Surgeon, Royal Free Hospital, London.

age with a short dyspeptic history are likely to remain symptom-free after successful treatment of a perforation (Illingworth). Nevertheless, within one year 40 per cent. of patients relapse, and within five years 70 per cent. On this account, those who survive perforation should be followed up as out-patients, so that if symptoms suggesting renewed activity of the ulcer occur, timely treatment can be instituted. Operation is advisable after three months.

Residual Abscess.—This may occur after perforation in any of the sub-phrenic spaces (Chap. 48) or in the pelvis.

HÆMATEMESIS AND MELÆNA

Ætiology.—

Chronic peptic ulcer	65% of cases.
Acute peptic ulcer (p. 821)	30% „ „
Multiple erosions	
Œsophageal varices (Chap. 45)	5% of cases.
Carcinoma of the stomach (p. 849)	
Mallory-Weiss syndrome	
Peptic ulcer in a Meckel's diverticulum (Chap. 49)	
Purpura (Chap. 44)	
Hæmophilia (p. 80)	
Pernicious and other anæmias	

(Compiled mainly from statistics by Sir Francis Avery Jones, London.)

Special Types.—Chronic Peptic Ulcer.—Slight bleeding due to trauma from solid food occurs frequently from all chronic peptic ulcers; such bleeding is demonstrated by finding traces of blood during gastric analysis and occult blood in the stools. With advancing age and the associated arteriosclerosis there is an increasing risk of severe hæmorrhage due to the erosion of an artery in the base of the ulcer. Occasionally the artery is of considerable size, viz. the splenic or gastro-duodenal artery; more usually it is a branch of one of these vessels. Once a sclerotic artery has been eroded, even if it is sealed by clot, it is liable to bleed again (fig. 886). Even when a large vessel is eroded, death seldom results from the initial hæmorrhage. Far more frequently a large hæmorrhage is heralded by two or three smaller ones on consecutive days, as in other cases of secondary hæmorrhage.

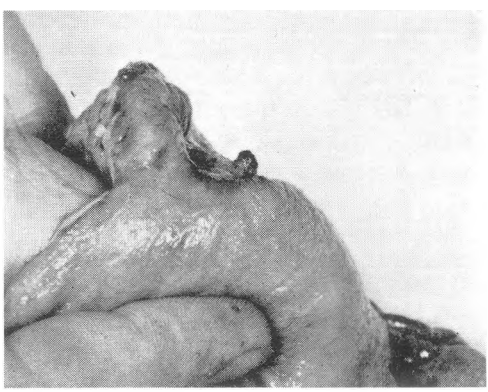


FIG. 886.—A gastric ulcer with an eroded and aneurysmal artery temporarily sealed by clot. *(Norman C. Tanner, F.R.C.S., London.)*

Acute Peptic Ulcer (p. 821).—This may be solitary or present as multiple erosions all over the stomach. The diagnosis is established by gastroscopy. It should be treated conservatively and all drugs withdrawn. Cimetidine (300 mg i.v. 2–4 times daily if renal function is satisfactory) may control the bleeding. It is particularly useful in stress ulcers.

Mallory-Weiss Syndrome.—This condition has specific features—the patient, usually a man over fifty, has a prolonged vomiting bout, often after imbibing alcohol. Having vomited gastric contents, he suddenly starts to vomit blood profusely and persistently, and becomes exsanguinated. As a result of the straining and retching a longitudinal tear of the mucosa just below the cardia occurs and gives rise to the sudden onset of hæmatemesis (fig. 887). The violent vomiting is sometimes due to

Sir Francis Avery Jones, Contemporary. Physician, Central Middlesex Hospital, London.
George Kenneth Mallory, Contemporary. Professor of Pathology, Boston University, Boston, Mass., U.S.A.
Soma Weiss, 1899–1942. Hersey Professor of Physic, Harvard University, Boston, Mass., U.S.A.

migraine or vertigo. The diagnosis can be made on the history and is easily confirmed by gastroscopy.

In 90% of cases the condition responds to sedation, blood transfusion and if necessary, pitressin. In the remainder, laparotomy is done and the linear tear exposed through a gastrotomy incision. It can then be sutured.

FIG. 887.—A healing mucosal tear extending across the œsophagogastric junction. (*Dr. Michael Atkinson, Nottingham, and the Editor and Publishers of 'Gut'.*)

Clinical Features in General.—Initial features are usually faintness, sweating, and pallor; occasionally the patient collapses. Soon afterwards there is hæmatemesis, which is effortless vomiting of coffee-ground material or bright-red blood. Later, black tarry stools (melæna) or red clotted blood may be passed per rectum.

Treatment.—*On admission* the collapsed patient is laid flat, with one pillow under the head, and the foot of the bed is raised. He is accorded the usual number of blankets for the time of year, and not heated artificially in any way. If restlessness is in evidence, morphine 15 mg. is given intravenously and repeated in four hours if necessary. Pending arrangements for blood transfusion, a plasma infusion is given. The important consideration is to replenish the circulation without overloading it, and a drip blood transfusion proportional to the estimated blood loss should be given. A pulse chart, if necessary half-hourly, is compiled, and a careful watch is kept on the blood pressure and central venous pressure (p. 73).

Very soon after a severe hæmorrhage the hæmoglobin concentration is often unchanged, and therefore no reliance should be placed upon it at this critical time. After three hours, estimations, repeated at frequent intervals, provide helpful information.

As a result of replenishing the circulation, improvement often occurs Conversely, signs that bleeding has very seriously depleted the blood volume and is probably continuing are a cold nose, increasing pallor, increasing pulse-rate, beads of sweat on the forehead, and clammy palms of the hands (p. 70).

Management:

Conservative (Medical) or Surgical Regimen?—This decision should be made by joint consultation and co-operation between the physician and surgeon. They will take into consideration the following factors:

Response to Treatment.—Above all, if there is recurrent or profuse bleeding while the patient is under adequate conservative treatment, the call for surgery is compelling.

Age.—Seventy per cent. of cases of bleeding are over the age of forty-five. After this age operation is increasingly necessary.

Chronicity of Ulcer.—The patient with a short history is more likely to respond to conservative treatment than one whose history suggests a deep penetrating ulcer. Sometimes, on account of cerebral anoxia, the patient's history is unreliable, when it will be necessary to obtain all possible information from the relatives. If he has been treated at another hospital, no time should be lost in contacting that hospital because all-important X-ray evidence may be forthcoming.

Ingestion of Drugs.—The patient must be closely questioned about the ingestion of aspirin, butazolidine, or cortisone. If so, withdrawal of these drugs may suffice.

Condition of Arterial Tree.—The presence of arterio-sclerosis as evidenced by the radial artery, fundal and electrocardiographic changes suggest the likelihood of recurrent bleeding.

Ancillary Aids.—As far as possible the exact cause of the bleeding should be made before operation.

Œsophagogastroscopy using the fibreoptic-type of endoscope is the ideal means of doing this. The patient's pharynx is sprayed with a local anæsthetic and sedation is achieved with Valium or other appropriate drugs. The instrument is then passed and the œsophagus examined. It is usually possible to exclude bleeding from varices in this way. If these are not seen, the instrument is passed into the stomach and the blood aspirated through its lumen. Should multiple clots be present and the site of bleeding not obvious a plastic nasogastric tube of large diameter should be inserted and the gastric contents aspirated. This is continued until a good view of the stomach can be obtained. Acute ulceration, erosions or the Mallory-Weiss syndrome (p. 837) may be seen. If blood is seen coming back through the pylorus, it is highly likely that a duodenal ulcer is the cause of the bleeding.

Conservative Treatment.—The essentials are:

Blood Transfusion.—When the estimated blood loss has been replenished, a drip transfusion is continued at the rate of 30 drops a minute for as long as is deemed necessary. The aim always is to render and maintain the reading of the hæmoglobin level at least above 75 per cent. of normal. In addition, frequent pulse-rate, C.V.P. and blood-pressure readings are continued for a minimum three days after the apparent cessation of hæmorrhage (p. 73).

Morphine.—Sufficient intramuscular morphine is prescribed to keep the patient just drowsy.

Intragastric milk drip is a valuable adjunct for the first forty-eight hours. It is discontinued when no further bleeding is manifest on gastric aspiration.

Diet.—Two-hourly 200-ml. feeds of milk, egg and milk, alternating with Benger's Food or Ovaltine, are given.[1] After three days milky porridge is allowed. It is essential that an accurate fluid balance-sheet be kept. After a week the patient is given the routine treatment for peptic ulcer (p. 828).

Prevention of Pulmonary Complications.—These patients are prone to develop respiratory infection, and not a few of them have bronchitis, with or without emphysema. Regular chest physiotherapy and prophylactic antibiotics are essential.

Operative Treatment.—A decision should be made within 48 hours of the commencement of bleeding, particularly if the patient rebleeds. Ex-

[1] Einar Meulengracht, Emeritus Professor of Medicine, Copenhagen, introduced in 1934 early liberal purée feeding in these cases. Before that time absolute starvation by mouth for several days was the unwavering rule.

perience has shown that when operation is delayed beyond that time the mortality rises steeply.

Before the patient is anæsthetised it is essential to ascertain that the blood transfusion is capable of rapid acceleration. In this connection, sometimes it is advisable, in order to provide a stream of blood equivalent to that of the splenic or the gastro-duodenal artery, to set up a second transfusion into a second vein. The aim of the operation is to stop the bleeding. *If an ulcer is found*, a partial gastrectomy is performed including removal of the ulcer if gastric. Alternatively, vagotomy and a wide pyloroplasty may be carried out together with direct suture of the bleeding vessel in the ulcer base if it is duodenal.

When no ulcer is found on external examination of the stomach, a wide gastrotomy, extended to gastroduodenotomy, should be made to expose occult ulcers, erosions, vascular 'raspberry' malformations (Gius), and cardiac tears (p. 837). Whenever possible, bleeding should be controlled by suture, diathermy and local excision-biopsy with suture. A 'blind' gastrectomy should be avoided but, if deemed necessary, the Billroth I type is favoured. Should no cause be found, examination of the rest of the gastrointestinal tract, and of structures which may bleed into the gut, must be meticulously performed.

CHRONIC COMPLICATIONS

1. **Pyloric stenosis** is usually the result of cicatrisation from a duodenal or juxta-pyloric ulcer. Not infrequently it is due to a carcinoma situated at or near the pylorus. Classically, pyloric stenosis occurs in women with a slow silent onset, but when there is a long standing history of ulcer the following modifications are evident:

FIG. 888.—Pyloric stenosis. Wave of peristalsis passing from left to right. Note that outline of stomach extends well below umbilicus.

Periodicity is lost.

Pain and *fullness* become more pronounced towards evening. The patients can usually eat breakfast, a very small lunch, and nothing thereafter because they feel full.

Vomiting.—Very large foul and frothy vomits are characteristic. They usually occur once a day, commonly in the evening. Classically, the patient recognises currants or other undigested particulate matter eaten one or more days previously. Vomiting usually gives considerable relief.

On Examination.—In thin subjects the outline of the enlarged stomach can sometimes be observed. Visible peristaltic waves passing from left to right (fig. 888) are characteristic. A succussion splash is heard. These patients may be mentally confused as a result of alkalosis.

Barium Meal.—The stomach is large and low (fig. 889), it is never empty, and the barium is mixed with food residue. There is much delay in its evacuation.

Treatment.—Operation in this condition carries a high mortality unless the patient is carefully prepared. Preliminary treatment by gastric lavage with saline through a wide-bore tube for 4–5 days, a high-protein fluid diet, and correction of electrolytes and vitamin deficiencies is necessary. Potassium and chlorides are usually deficient. A normal output of urine indicates adequate hydration (p. 88). Where chronic peptic ulcer is the cause, partial (hemi-)gastrectomy is usually the best procedure. A Polya type operation is preferred. Because of the stenosis, the risk of leakage from the duodenal stump is negligible. In elderly frail patients a gastrojejunostomy will

John Armes Gius, Contemporary. Professor of Surgery, University of Iowa, Iowa City, Iowa, U.S.A.

suffice. Should the condition be due to carcinoma, radical gastrectomy will be required (p. 854).

2. **Hour-glass stomach** occurs almost exclusively in women, is usually silent, and is due to cicatricial contracture around a saddle-shaped lesser-curve ulcer. In extreme cases the stomach is divided into two compartments, united by a channel which barely admits a pencil (fig. 890). The condition is sometimes associated with pyloric stenosis.

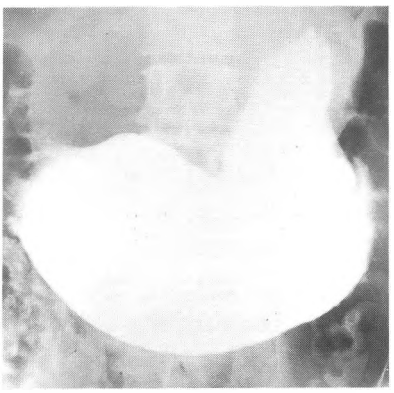


FIG. 889.—Typical X-ray appearance of pyloric stenosis.

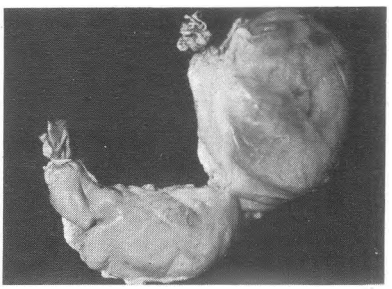


FIG. 890.—Hour-glass stomach.

History.—*Periodicity* is lost. The symptoms have become practically constant. *Vomiting* is more frequent, and gives no relief to the discomfort.
The *appetite* becomes poor.
Weight.—Loss may be so great that carcinoma is suspected.
Barium meal is often characteristic. We have known of cases of hour-glass stomach being reported as pyloric stenosis, owing to failure of the second pouch to fill. True hour-glass stomach must be distinguished from gastric spasm of the hour-glass type, which is sometimes associated with an uncomplicated ulcer on the lesser curvature.
Gastroscopy.—The gastroscope enters the upper compartment of the stomach, and the narrow, scarred channel leading to the lower compartment is usually seen.
Treatment.—Billroth I gastrectomy, with removal of the second pouch and the isthmus, is the best treatment.

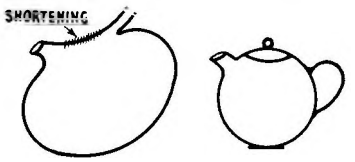
3. **Penetration of pancreas** may occur with posteriorly disposed gastric and duodenal ulcers.

History.—The main change is that pain is referred to the back and may be aggravated by stooping or exercise and *relieved by lying down.* The back pain may be so prominent that the patient is referred in the first instance to an orthopædic surgeon.
Treatment.—Recognition of this feature of the ulcer is an indication for surgery, not only for relief of pain but also because of the possibility of torrential hæmorrhage.

4. **Malignant Change.**—This only occurs in gastric ulcers and is rare in lesser curve ulcers proximal to the angulus (p. 823). Endoscopy and biopsy are essential if surgery is not to be offered.

History and Barium Meal.—This often gives no guide to the seriousness of the condition. The important point is a constant awareness of the possibility of malignancy *de novo* at all times. Over 20 per cent. of all antral ulcers seen on X-ray examination for the first time are malignant, or have been malignant, from the start.

5. **'Tea-pot' Stomach.**—Cicatrisation around a long-standing gastric ulcer often causes shortening of the lesser curvature, thus producing the 'tea-pot' deformity of the stomach, viz.—→



'Tea-pot' stomach (Davey), called in the U.S.A. 'handbag' stomach, is not uncommon. Unless the deformity is known to the radiologist it is likely to be diagnosed as a congenital abnormality or as the result of a carcinoma. The pylorus being no longer in an advantageous position for complete emptying of the stomach, these patients may present with symptoms and signs of pyloric stenosis.

COMPLICATIONS AFTER GASTRIC OPERATIONS

(1) Complications following any operation (e.g. pulmonary, cardiac, thrombotic).

(2) *Specific* complications of gastric operations.

A. **Early**	B. **Remote**
1. Hæmorrhage from anastomotic line.	6. Recurrent ulcer.
2. Paralytic ileus.	7. Gastro-jejuno-colic fistula.
3. Stomal obstruction.	8. Post-gastrectomy syndromes.
4. Duodenal fistula.	9. Intestinal obstruction.
5. Acute post-operative pancreatitis.	10. Pulmonary tuberculosis.
	11. Anaemia.
	12. Decalcifying bone disease.
	13. Carcinoma in remnant.
	14. Gallstones.

1. **Hæmorrhage from Anastomotic Line.**—If it does occur, morphia should be given together with a slow blood transfusion. Adrenaline (1 ml. (1:1000) solution hourly) by mouth may be helpful. If the hæmorrhage is *severe* and *persistent*, the abdomen should be re-opened, continuous gastric lavage started and the anastomosis oversewn with continuous through-and-through catgut. The stomach is not opened. With control of the hæmorrhage, the washouts become clear. If this measure fails, the stomach must be opened and the cause of the bleeding found.

2. **Paralytic Ileus.**—Is treated by 'drip and suction' (Chap. 50.)

3. **Stomal Obstruction.**—This includes two different conditions: (*a*) Due to œdema of the actual stoma or of adjacent small bowel. This occurs more frequently after a gastro-duodenal anastomosis—hence the need for a large stoma in this operation. It is important to persevere with gastric aspiration and intravenous therapy maintaining correct electrolyte balance, especially of potassium. After a Polya gastrectomy, the stoma should be open and the patient absorbing fluids within 3 days. The Billroth I operation stoma often takes 5 days to open.

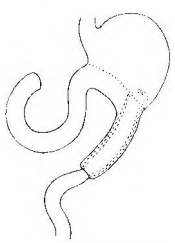


FIG. 891.—Jejuno-gastric intussusception following Polya gastrectomy. (*After P. F. Early, F.R.C.S.*)

(*b*) Due to the occurrence of *retrograde jejuno-gastric intussusception* (fig. 891). This, of course, only occurs after a gastro-jejunal anastomosis and may appear immediately or after many weeks or months. The stomach completely fails to empty and a filling defect can be clearly seen on X-ray with a small amount of barium. The intussusception may reduce following the barium or gastric saline lavage or the adoption of the sitting or standing position. If it does not respond to these measures (and it usually does in a few hours), an operation must be done to reduce the intussusception by gentle downward traction. An attempt may be made to anchor the loop by stitching or entero-anastomosis.

William Wilkin Davey, Contemporary. Formerly Professor of Surgery, Ibadan, Nigeria.

4. **Duodenal fistula** (**'Blow-out'**) is an infrequent, but very serious, complication of Polya partial gastrectomy with a maximal incidence about the fourth day. It accounts for 60% of the deaths after gastrectomy, and occurs where there has been difficulty in closing the duodenum so that avascular necrosis occurs. Another cause is increased tension within the afferent jejunal loop.

Prevention.—There are two important points in technique:

(*a*) Careful duodenal closure with meticulous turn-in. A crushing clamp should not be used.

(*b*) Construction of the stoma in such a way that afferent loop contents have free egress. Multi-lumen tubes have been used to drain the afferent loop, but unless they are stitched into position, the tube is usually rejected. A tube drain down to the duodenal stump, or even into the duodenum (formal fistula) are worthwhile safeguards if trouble is anticipated. When a blow-out occurs the duodenal sutures give way and, if drainage was not provided at the time of the operation, there is intense thoraco-abdominal pain, which is not infrequently mistaken for acute basal pneumonia with pleurisy. Unless prompt action is taken, diffuse peritonitis ensues, and death is the usual sequel.

(*c*) Avoid operating on a hot duodenum, i.e. where the ulcer is active and inflamed. Choose another operation, e.g. vagotomy and gastrojejunostomy.

Treatment consists in providing free drainage down to the duodenum with peritoneal toilet by suction. When drainage of the periduodenal tissues was not provided at the time of the gastrectomy, a small subcostal incision is made down to the duodenum. In either event, sump suction drainage (fig. 1027) is instituted until, and even after, the track of an external duodenal fistula has become defined. Some advise temporary jejunostomy at the same time. The duodenal discharge can be collected and returned into the jejunostomy together with the necessary nutritive fluids (p. 858). The fistula usually closes spontaneously.

5. **Acute post-operative pancreatitis** is rare. The condition carries a high mortality rate. Treatment is along the usual lines (Chap. 47).

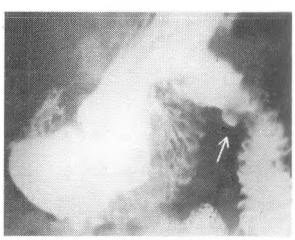


FIG. 892.—Jejunal ulcer following gastro-jejunostomy. (*The late Dr. K. J. Yeo, London.*)

6. **Recurrent Ulcer.**—This includes the true anastomotic ulcer (gastro-jejunal, gastro-duodenal or jejunal ulcer) or a gastric ulcer in the remnant. True anastomotic ulceration has an incidence of 3 per cent. after a Polya gastrectomy, 5 to 7 per cent. after vagotomy and gastro-enterostomy, and up to 40 per cent. following gastro-enterostomy (fig. 892). If any part of the antrum is left *in situ* after gastrectomy, the recurrence rate may rise to 30 per cent. The symptoms which appear usually within two years after operation consist of severe persistent pain, 'boring' in type, which is worse within a few minutes of taking food. The pain passes down the left side of the abdomen, sometimes into the perineum, but may be felt in the lower left chest especially following an ante-colic anastomosis. It is not relieved by antacids or milk. Bleeding manifest as hæmatemesis, melaena, occult blood, or secondary anæmia is common and perforation may occur. Only 30 per cent. are seen by radiology. Gastroscopy tends to be more helpful. A maximal acid output of 25 mEq/l (25 mmol/l) is, however, very suggestive of stomal ulceration. Diagnostic laparotomy may be justified if symptoms are severe.

Management.—Conservative treatment is ineffectual, or of only temporary benefit; re-operation is usually necessary.

(a) **Following Polya gastrectomy.**—An anastomotic ulcer may be obvious or require the opening of the anterior suture line. If the stoma is adequate and acid hypersecretion is present, vagotomy will cure the patient. If low acid secretion is found, revision gastrectomy with a fresh anastomosis to correct stasis, is advisable.

(b) **Following Billroth I gastrectomy.**—Anastomotic ulcers should be treated by vagotomy. Recurrent gastric ulcer should be treated by higher gastrectomy, including the ulcer in the resected specimen. A Polya type operation is preferred.

(c) **Following gastro-enterostomy.**—Provided the stoma has not been narrowed by scarring, vagotomy will effect a cure.

(d) **Following Vagotomy and a drainage operation.**—The Hollander Test (p. 812) should reveal incomplete nerve section. A search must be made for an intact vagal trunk in order to divide it. The drainage operation should not be changed but it must be functioning adequately. If an intact vagus nerve cannot be found, the operation should be converted to Polya gastrectomy. If, however, a gastric ulcer is present, Billroth I conversion should be carried out, resecting the ulcer with the specimen.

(e) In all patients having anastomotic ulcers and gastric hypersecretion, a search must be made for an ulcerogenic pancreatic islet-cell tumour.

7. **Gastro-jejuno-colic fistula**—is a complication of gastro-jejunal ulcer, and more often follows simple gastro-enterostomy than partial gastrectomy. The ulcer penetrates and erodes the transverse colon. Usually the symptoms of anastomotic ulcer disappear soon after the fistula develops, but in their place the unfortunate patient is troubled with severe diarrhœa after every meal, and eructates foul gas. Exceptionally the patient vomits fragments of formed fæces. Loss of weight and strength, dehydration, and anæmia complete the picture. It is important to stress that this final picture may be of extremely rapid onset so that the patient becomes desperately ill within two to three weeks. Severe malabsorption is shown by the presence of cachexia, hypoproteinæmic œdema, and steatorrhœa. The chief factor producing the rapid deterioration of the patient is the fouling of the jejunum by colonic contents with resulting disturbance of the vital absorptive mechanisms. It is *not* that the gastric contents pass directly into the colon. The diagnosis is established by a barium enema; in more than half the patients a barium meal may fail to reveal a fistula (fig. 893). *The sudden onset of severe diarrhœa in a patient who has a gastro-enterostomy must always raise the serious possibility of this condition.*

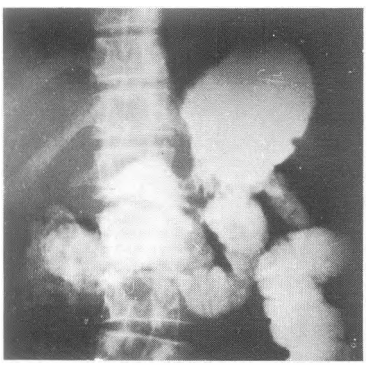


FIG. 893.—Gastrocolic fistula.
(Courtesy Dr. Vachhani).

Treatment.—This consists of a resection of the fistula with repair of the colon and jejunum, together with either vagotomy or a high partial gastrectomy. This can be one of the most difficult procedures that any surgeon may be called upon to do.

Some of these patients are extremely ill, and may require resuscitative measures, oral antibiotics and intravenous feeding before operation is attempted.

8. Post-gastrectomy Syndromes

These complications are directly attributable to altered alimentary function. The proper understanding of all these syndromes depends on a thorough knowledge of gastric and intestinal physiology.

Briefly, the main role of the stomach is mechanical breakdown of food with limited proteolysis and, more important, the slow regular delivery of chyme into the small bowel. Chemical breakdown of nutrient (digestion) is accomplished almost entirely

within the lumen of the small intestine by pancreatic juice and by mucosal surface enzymes, as is assimilation into the body (absorption). In health, there is co-ordination of function within the small bowel so that the transit of digesta is related to the rates of digestion and absorption. Normally, water and crystalloid solutes (sugars, amino acids) are absorbed in the proximal small bowel, and fats (triglycerides) in the mid bowel, leaving a considerable margin of safety. Selective absorption sites exist in the terminal ileum for bile acids and for vitamin B_{12}.

The effect of gastrectomy may be to distort normal physiological function, with consequent effects on transit, digestion, and absorption. Two effects of gastrectomy may be of particular importance:

(1) The loss of pyloric regulation may permit the rapid entry of nutrients into the duodenum, with an increased volume flow, acid load, and solute load. One consequence of this may be adverse alteration of the optimum conditions for digestive enzyme activity; another may be altered intraluminal osmolarity.

(2) The establishment of a gastro-jejunal anastomosis bypassing the duodenum means that important receptor sites are not stimulated by the presence of food, with adverse consequences on hormone release, and the neural regulation of motor activity. In addition, mixing of pancreaticobiliary secretions with food will be less efficient. These effects are probably more important than the simple loss of absorptive surface.

Gastrectomy may cause reduced secretion of HCl, pepsin, intrinsic factor and pancreatic enzymes, inadequate mixing of food with enzymes and bile, reduced absorption of protein, fat and iron, rapid absorption of glucose, increased intestinal motility, and creation of a blind loop (i.e. the afferent loop, fig. 894).

The effects of some of those changes may appear a few weeks after operation, especially the post-cibal syndromes. It may be several years, however, before the absorption defects produce apparent nutritional disturbances.

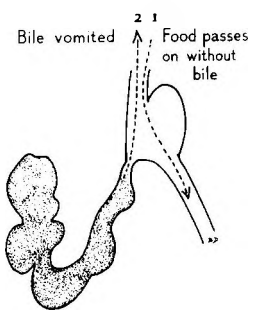


FIG. 894.—The afferent loop syndrome. The food (1) passes out of the gastric remnant before the bile (2) is ejected. *(After Charles Wells, F.R.C.S.)*

The various **Post-gastrectomy Syndromes** therefore may be classified as follows:

A. Post-cibal Syndromes

(i) Early post-cibal syndrome (*syn.* early dumping).

(ii) Late post-cibal syndrome (*syn.* hypoglycæmia or late dumping).

(iii) Bilious vomiting.

B. Nutritional Syndromes

(i) Weight loss.
(ii) Steatorrhœa.
(iii) Diarrhœa.
(iv) Iron-deficiency anæmia.
(v) Megaloblastic anæmia.
(vi) Vitamin B deficiency.
(vii) Calcium deficiency.
(viii) Gross malabsorption states.

By and large the higher the resection, the greater the risk of post gastrectomy troubles. In addition, these syndromes are best prevented if the normal duodenal pathway is maintained—and not by-passed.

Post-cibal Syndromes—Early and Late

In general, these occur more frequently in women than in men, and are seen following the Polya operation rather than the Billroth I. Time relation to meals permits classification into early and late varieties; the main features of these are summarised in the following table:

	Early	*Late*
1. Incidence	5–12 per cent.	5 per cent.
2. Relation to meals	Immediately afterwards	During second hour afterwards
3. Duration of attack	Thirty to forty minutes	Thirty to forty minutes
4. Long-term	Severe cases indefinitely	Two to five years
5. Relief by	Lying down	More food, glucose
6. Aggravated by	More food	Exercise
7. Precipitating factor	Bulk of food, especially if wet	Carbohydrates
8. Chief symptoms	Epigastric fullness Sweating Sensation of warmth Tachycardia Occasionally colic, diarrhœa	Tremor Faintness Epigastric emptiness Nausea
9. Occurrence	Commonest after Polya	After any type of gastrectomy

The Early Post-cibal Syndrome.—This syndrome, consisting of abdominal and vasomotor symptoms, is seen in the majority of patients during convalescence, and persists in 5–12 per cent. A variety of causes may be involved—rapid emptying of the stomach with increased small bowel activity, distension of the afferent loop, and a fall of blood volume (of the order of 7 per cent.), especially in patients susceptible to such changes. According to Le Quesne, there is a primary disorder of carbohydrate metabolism. Following the ingestion of glucose, there is initial transient hyperglycæmia. This suppresses further absorption of glucose, which is retained in the intestine, resulting in fluid shift from the blood to the lumen of the intestine. Both increased intestinal activity and fall of blood volume may occur.

Treatment.—Time must be allowed to elapse for the syndrome to subside naturally. Meals should be small in bulk and dry. Milk and carbohydrate meals should be avoided. Belladonna or codeine may reduce intestinal activity. An oral hypoglycæmic agent before food has a beneficial action. It is important to maintain the hæmoglobin level as near 100 per cent as practicable. When the syndrome persists, especially when associated with nutritional defects, conversion of a Polya anastomosis to a Billroth I is the operation of choice.

Late Post-cibal Syndrome.—This is not a common or a serious cause of symptoms. It is due to a low blood sugar, occurs two hours after meals, and may be seen in some 5 per cent. of patients. After the initial hyperglycæmia there is a rapid fall of blood sugar to 50 mg/100 ml (2·8 mmol/l) or so. This rapid fall is probably due to increased insulin sensitivity. The syndrome is readily corrected by taking food, especially glucose

Bilious Vomiting.—This symptom is usually an isolated feature but may be associated with dumping. It occurs in 10 to 15 per cent. of patients and consists of vomiting of bile unmixed with food. The vomiting is intermittent rather than after every meal. In the Polya operation the cause is probably transient obstruction of the afferent loop. Following the Billroth operation it is more likely to be due to incompetence of the cardia. This symptom can be eliminated almost entirely by a retrocolic anastomosis with the shortest possible afferent loop, i.e. into the fourth part of the duodenum if this is possible. If surgery is necessary to correct the vomiting, conversion to a Billroth I should be done if there are other post-cibal symptoms.

Nutritional Disturbances

Weight Loss.—This is a well recognised feature, occurring in some 50 per cent. of patients. It is the rule after total gastrectomy, common after the Polya operation, and infrequent after the Billroth I. It is due to reduced intake of food, and diminished absorption, and is particularly liable to occur in patients with postcibal symptoms. It is not necessarily serious, and indeed may have a beneficial effect. There is a reduced incidence of coronary disease following gastrectomy.

Leslie Philip Le Quesne, Contemporary. Professor of Surgery, Middlesex Hospital, London.

It is essential in these patients to exclude reactivation of previous tuberculous disease.

Steatorrhœa.—This has a similar incidence to weight loss, and is due to poor mixing of food and enzymes, reduced pancreatic output, and inactivation of pancreatic enzymes in the afferent loop. If it appears slowly after operation, the cause may be latent intestinal disease such as jejunal atrophy or the development of a cul-de-sac phenomenon (i.e. blind loop, fig. 1087) in the afferent loop.

Diarrhœa.—Most patients say that their bowel habits are much improved after gastrectomy, but some 5 per cent. have diarrhœa, either episodic or persistent. It may be related to intestinal hurry or to steatorrhœa. Diphenoxylate or loperamide may be helpful in managing this problem.

Iron-deficiency Anæmia.—This is a progressive condition after gastrectomy, and may be seen in some 40 per cent. of patients, particularly when the duodenum is by-passed. If occult blood loss is excluded, daily supplements of iron are essential.

Megaloblastic Anæmia.—Although invariable several years after total gastrectomy, it is rare following partial gastrectomy. It may occur as an isolated lesion due to gastric mucosal atrophy, or as part of a gross malabsorption defect (*vide infra*). There is evidence of reduced serum vitamin B_{12} levels in some 25 to 50 per cent. of patients five to ten years after partial gastrectomy. Intramuscular vitamin B_{12} (Cyanocobalamin) 100 μg. weekly is given until the blood is normal; thereafter a maintenance dose each month is necessary.

Vitamin B Deficiency.—This occurs in roughly 10 per cent. of patients, and is manifest by angular stomatitis, glossitis, or peripheral neuritis. A regular supplement is the best method of prevention.

Calcium Deficiency.—Reduced acidity in the proximal intestine may interfere with calcium absorption, and some 40 per cent. of patients may become deficient. Bone changes may occur in some 5 per cent. and although such changes are usually seen associated with steatorrhœa, they may occur independently.

Gross Malabsorption States.—Very rarely, in less than 1 per cent. of patients, a severe malabsorption syndrome may develop. Conversion to a Billroth I is usually very effective.

Post-vagotomy Syndromes

Effects of Vagotomy.—In addition to its gastric secretory effects, total abdominal vagotomy may produce undesirable side effects. The loss of gastric motility produces gastric distension, with delayed emptying, resulting in a persistent gastric splash, nausea, loss of appetite, foul eructations, vomiting and abdominal pain. Gastric ulceration may occur. The effect of vagotomy on the rest of the bowel is variable. About 30% of patients have an increase in bowel habits; of these a very small number suffer from variable diarrhœa which at its worst is urgent, episodic, uncontrollable and watery.

Vagotomy and inadequate drainage operation.—When no drainage operation accompanies vagotomy, gastric emptying may be inadequate, partly resulting from duodenal ulcer scarring, partly resulting from the reduced gastric motility following vagotomy. It is not possible to estimate the size of the pyloroduodenal canal by inspection from outside; moreover the site of narrowing is inconstant. Pyloroplasty in which the duodenotomy does not extend beyond any narrowing is similarly inadequate. A small, badly sited gastroenterostomy stoma may not allow the stomach to empty. Symptoms of gastric retention follow, and can be relieved by performing an adequate drainage operation.

Vagotomy and adequate drainage operation.—In spite of apparently adequate complementary surgery following vagotomy, some patients have symptoms of gastric retention after operation. Failure of gastric emptying may continue for up to 3 weeks, even though the stoma is patent and bile can be aspirated from the stomach. This functional disability slowly resolves. However, post-prandial fullness, regurgitation of bitter fluid into the mouth, and abdominal colic are experienced by some patients for longer periods.

The post-operative effects may be linked not to the vagotomy but to the complementary operation. As a result, the merits of each operation are hotly debated.

Because the symptoms tend to improve, conservative treatment usually suffices. The patient is advised to take small dry meals, with fluids taken between meals. Foods which are found to produce symptoms are temporarily discontinued.

Metoclopramide and more recently bethanecol 5 mg q.i.d. before meals (Catchpole) have been used with success.

Diarrhœa may be controlled by codeine phosphate or similar drug. A course of phthalylsulphathiazole or neomycin is often beneficial and the improvement may continue after these drugs are stopped.

Recurrent ulcer after vagotomy.—The insulin test of gastric secretion confirms that vagotomy is incomplete. If the symptoms do not respond to medical treatment, re-operation is performed. If a vagal trunk is found and divided, provided that the existing drainage procedure is adequate, nothing more should be done. If no residual intact vagal fibres are found, gastric resection is necessary. If a gastric ulcer is found, gastrectomy should be performed. The possibility that the Zollinger-Ellison syndrome may be present should be borne in mind.

Intestinal Obstruction

(*a*) **Herniation Obstruction.**—In cases of a Polya gastrectomy with an antecolic anastomosis, small intestine herniates through a gap between the anastomosis and the transverse mesocolon, either from right to left or from left to right. It is easily prevented by a few stitches between the anastomosis and the transverse mesocolon at the time of gastrectomy (Stammers).

Clinical Features.—Usually symptoms of high intestinal obstruction commence between the third and eighteenth post-operative days, but often these symptoms soon become atypical because gastric aspiration is instituted after the first or second vomit. For the same reason colicky pains, so typical of intestinal obstruction, are often lacking; in most cases the pain is continuous, and increases in severity.

Treatment.—Even if the condition is only suspected, the abdomen should be re-opened. The hernia is usually reducible, but gangrene has occurred from long delay. The gap should be closed as detailed above and gangrenous bowel resected if necessary.

(*b*) **Bolus Obstruction.**—The commonest cause of this is a piece of unmasticated orange pith which, in the absence of the pylorus, is able to pass into the small intestine and lodges about two feet from the ileo-cæcal valve. Clinically, it is not unlike gall-stone obturation (Chap. 50) but occurs in a younger age group. *Every patient who has had the pylorus excised or by-passed must be told not to swallow orange pith or dried fruits.*

Pulmonary Tuberculosis

Gastrectomised persons show a slight but definite increased susceptibility to pulmonary tuberculosis, probably due to reactivation of a latent focus owing to diminished nutrition.

Carcinoma

Following a gastrectomy, especially for a gastric ulcer, the gastric remnant is slightly more prone to develop a carcinoma than is a normal stomach. This risk appears after a latent period of fifteen to twenty years or more. It is because of such a complication following gastrectomy that the recent results of vagotomy plus a drainage procedure must be accepted with some reserve. Carcinoma may follow this operation also.

Gallstones

There is evidence that the incidence increases after all types of gastric surgery.

GASTRIC NEOPLASMS

Benign Tumours.—Although they constitute 3 per cent. of all gastric neoplasms benign tumours of the stomach are so over-shadowed by the frequency and gravity of gastric carcinoma that they tend to be forgotten.

Leiomyoma is the most common benign neoplasm of the stomach. Sometimes it grows mainly towards the serosal coat, in which case it is symptomless, and attracts notice only when it is large enough to constitute a painless, smooth lump in the epigastrium. It may cause a low-grade fever. More usually a leiomyoma protrudes towards the lumen of the stomach, and gives rise to melæna, mild indigestion, and epigastric pain, in that order. Hæmatemesis is, however, the most common symptom, and results from deep central ulceration of the tumour. Radiography after

Bernard Newman Catchpole, Contemporary. Professor of Surgery, Perth, Western Australia.
Francis Alan Roland Stammers, Contemporary. Emeritus Professor of Surgery, Birmingham, England.

a barium meal reveals a space-filling lesion (fig. 895). Necrobiosis of the tumour has led to perforation of the serosal surface with intraperitoneal bleeding, and even to perforation of the stomach. Local excision is curative.

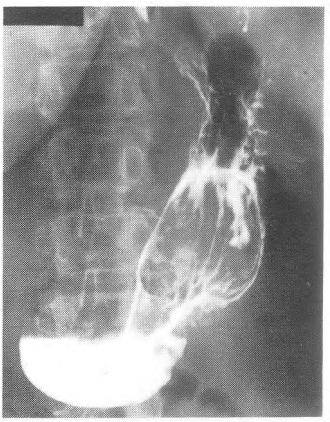


FIG. 895. — Leiomyoma of stomach. A round space filling defect in the mid-stomach with small ulcer cavity at 3 o'clock.

Neurofibroma and Neurilemmoma. — A tumour arising from a nerve sheath gives rise to exactly the same symptoms as a leiomyoma.

Adenomatous polypus, sometimes single, but more often multiple, is, as a rule, situated in the distal half of the stomach. Usually the symptoms are bleeding and abdominal pain. Achlorhydria is present in nearly all cases. Rarely a pedunculated adenomatous polypus of the pyloric antrum is carried into the pyloric canal, there to cause pyloric obstruction. The diagnosis rests on gastroscopy and/or X-ray. It is wise to regard multiple polypi as pre-malignant, and accordingly perform partial gastrectomy so as to include all the polypi; a single polypus can be excised locally.

Adenomatous Polypi and Pernicious Anæmia.—In pernicious anæmia there is severe atrophy of that portion of the gastric mucosa containing the fundal glands, and this provides a fertile bed for precarcinomatous adenomatous polypi to flourish. In about one in five cases of pernicious anæmia gastroscopy reveals adenomatous polypi. It is for this reason that patients suffering from pernicious anæmia are recommended to undergo annual gastroscopy and a barium meal.

An aberrant pancreas has been found arising in the wall of the stomach from time to time. Usually partial gastrectomy has been performed by a surgeon mistaking it for a carcinoma.

Menetrier's Disease.—A condition in which there is giant hypertrophy of the mucosa of the gastric fundus with cystic changes in the crypts, the antrum being spared. There is inconclusive evidence that the condition may predispose to carcinoma. It is associated with protein loss and may require surgical treatment, partial gastrectomy being the usual operation used. It should be distinguished from the hyperrugosity of the gastric mucosa, which may be associated with the Zollinger-Ellison syndrome (p. 953), but here there is no evidence of protein loss and there is acid hypersecretion.

CARCINOMA OF THE STOMACH

Carcinoma of the stomach is one of the 'captains of the men of death'. In 1959, 14,076 persons died from this cause in England and Wales but, fortunately, it now appears to be decreasing. While from early adult life to senility no age is exempt, the highest incidence is between forty and sixty years of age. Three times as many males as females fall victims to this disease.

Ætiology.—There are a few definite pre-malignant conditions. These are a gastric polypus, pernicious anæmia, and possibly, but only rarely, a gastric ulcer (p. 823). Ingested carcinogens seem to be important:—the hydrocarbons of cooking fat and tobacco juice. Furthermore, substances that cause an irritative gastritis, such as neat spirits as drunk in Scandinavia, Holland, and Czechoslovakia, may increase the incidence of carcinoma. The geographical distribution of this disease is important and may prove a most profitable avenue in the study of ætiology. An interesting example of this is the differing incidence in the North and South of Iceland. Crude smoked salmon was the staple diet of the Northerners who have a much higher incidence than those in the South. Aird found a higher incidence of gastric carcinoma in people with blood group A.

Pierre Menetrier, 1859–1935. French Physician.
Ian Aird, 1905–1962. Professor of Surgery, Royal Postgraduate Medical School, London.

Pathology.—*Macroscopically* five types can be recognised :

Type 1.—A cauliflower-like growth with sharply defined edges. Its surface is indurated. Later it ulcerates.

Type 2.—An ulcer with an irregular indurated edge. Sometimes small superficial ulcers are present in the immediate vicinity.

Type 3.—Colloid carcinoma.

Type 4.—Scirrhous localised or diffuse (leather-bottle, fig. 900).

Type 5.—Carcinoma secondary to a chronic gastric ulcer (p. 823).

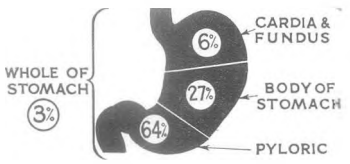


FIG. 896.—Incidence of carcinoma in various portions of the stomach.

Microscopically.—The growth is usually columnar-celled, but cubical, and even squamous-celled neoplasms arise near the œsophageal orifice. The last type probably arises within the œsophagus.

Site:—The most common site for the neoplasm is in the prepyloric region (fig. 896); when carcinoma follows pernicious anæmia however, it is more likely to be fundal and polypoid.

The Spread of Carcinoma of the Stomach.—No better example of the various modes by which carcinoma spreads can be taken than the case of the stomach.

1. *Direct Spread.*—As the growth enlarges, it tends to invade neighbouring structures. The pancreas, transverse colon, mesocolon, œsophagus, or liver may be involved.

2. *Lymphatic Spread* (fig. 902): both by emboli and permeation.

3. *Spread by the Blood-stream.*

4. *Transperitoneal Implantation.*—Carcinoma cells sometimes pass from the stomach into the peritoneal cavity. They gravitate to the pelvis, where secondary tumours palpable on rectal examination may develop. On occasions, in the female, they alight upon the ovaries, giving rise to **Krukenberg's tumours** (fig. 897), which are liable to cause diagnostic confusion. These tumours are premenopausal, because after the climacteric the ovaries atrophy and thus are unlikely to give sustenance to tumour cells. In some cases Krukenberg's tumours are not the result of transcœlomic implantation, but are due to retrograde lymphatic permeation, especially from a carcinoma of the colon.

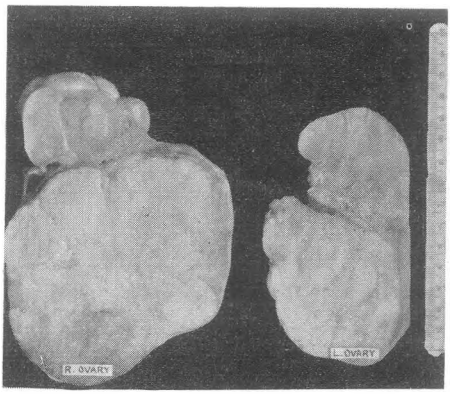


FIG. 897.—Krukenberg's tumours from a woman of thirty-seven. Scale in inches.

Clinical Features.—This is a very difficult disease to diagnose early, not only because of the diversity of its presentation but also because of the time-lag between the commencement of the growth and the appearance of symptoms:—

1. Gastric distension: inability to take a normal meal, vomiting.
2. Anorexia leading to loss of weight.
3. Anæmia, tiredness, weakness, pallor.
4. Persistent pain: no response to treatment—no periodicity.

Clinical groups.—i. The '*new dyspepsia*' *after forty*—vague but

Friedrich Ernst Krukenberg, 1870–1946. Ophthalmologist, Halle, Germany, wrote a classic paper on malignant tumours of the ovary in 1896.

persistent indigestion occurring in a patient who has never previously had ' stomach trouble '.

ii. *Insidious Onset.*—Especially in a man: he feels tired and weak, and the three A's may be in evidence—Anæmia, Anorexia, and Asthenia. Although the majority have an iron-deficiency anæmia, a few may present with a macrocytic anæmia.

iii. The *obstructive* types—carcinoma of the cardia presents with dysphagia; carcinoma of the pylorus with fullness, belching, and then vomiting.

iv. *Lump.*—The incidental discovery of a lump in the epigastrium, no other symptoms being present, is sometimes the cause of the patient seeking advice. In about 30 per cent. of cases a lump can be palpated.

v. *Silent.*—Carcinoma of the body of the stomach may be silent but give rise to features in other organs, such as obstructive jaundice due to secondary deposits in the liver, ascites from carcinomatosis of the peritoneum (Chap. 48), Krukenberg's tumours (see above), Trousseau's sign (phlebo-thrombosis of superficial veins of leg), or Troisier's sign (fig. 898).

INVESTIGATIONS IN A SUSPECTED CASE

If the diagnosis of this dread condition is to be made earlier, it is essential that attention is paid to the following investigations in *all* cases of indigestion.

Hæmoglobin—anæmia is present in 45 per cent. of cases.

Sedimentation rate—this is raised in 70 per cent. of cases.

Occult blood is present in the stools in 80 per cent. of cases.

Where necessary, repeated tests should be done. It is important to remember that a negative test does *not* exclude a neoplasm.

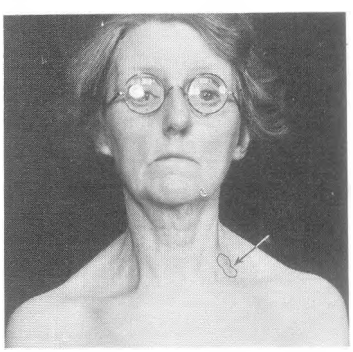
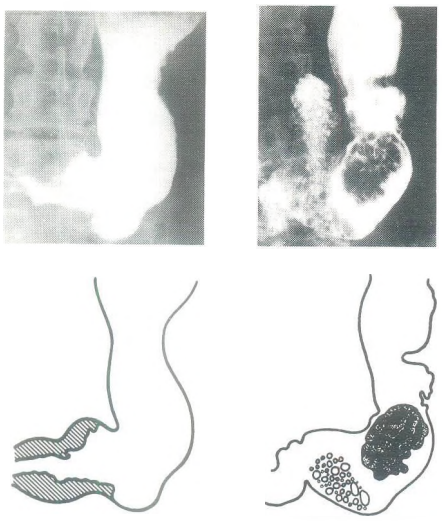


FIG. 898.—Troisier's sign found in the course of a routine examination for vague dyspepsia of recent origin. In this case there was a visible as well as a palpable mass of lymph nodes in the left supraclavicular fossa.

Radiology.—While a skilled radiologist attains a high degree of accuracy in the interpretation of a barium meal, small lesions are liable to escape notice, particularly if too much barium is given initially. Irregularities of the cardia or fundus are inclined to be overlooked unless the technique includes an examination in the inverted position. Flat growths may remain undetected in the early stages. Radio-diagnosis is accurate in 90 per cent. for pyloric growths, 75 per cent. for the cardia, and 60 per cent. for neoplasm of the body, but in only 47 per cent. of patients with conclusive radiological findings (filling defects) (fig. 899) is the neoplasm found to be resectable.

Gastric Secretory Studies.—The majority have either achlorhydria (p. 816) or hypochlorhydria, but occasionally some increase of acid is found. If the M.A.O. (p. 816) is more than 20 mEq/l (20 mmol/l) HCl a carcinoma is unlikely.

Armand Trousseau, 1801–1867. Physician, Hôtel-Dieu, Paris. It was this sign which first caused him to suspect his own gastric carcinoma.
Charles Émile Troisier, 1844–1919. Professor of Pathology, Paris.



(*a*) (*b*)

FIG. 899.—Barium-meal X-ray in carcinoma of (*a*) the antrum and (*b*) the body of the stomach.

Gastroscopy.—If the tumour can be visualised, its characteristics are obvious. Its nodularity, the presence of several irregular superficial ulcers, the multicoloured base, the immotility of the adjacent mucosa, all point to the nature of the lesion. Multiple biopsies are essential.

Exfoliative Cytology.—Examination of cells found in the washings after gastric lavage has proved sufficiently reliable to confirm a diagnosis of carcinoma of the stomach in doubtful cases in about 70 per cent. of cases. Negative results are of no moment. Chymotrypsin lavage to soften the mucous lining has aided this method of investigation.

Laparotomy.—If there is still reasonable doubt after all these tests, it is wise to explore the abdomen.

COLLOID CARCINOMA

The stomach appears infiltrated in all its layers by a kind of areolar tissue, the interspaces of which contain a transparent gelatinous substance. Groups of cancer cells often line the accumulations of colloid. The tubular glands are extremely distended with this substance. Colloid carcinoma represents about 6 per cent. of the total cases of gastric carcinoma. It is this type of carcinoma which classically gives rise to the Krukenberg phenomenon (p. 850).

The treatment is the same as that for leather-bottle stomach and the prognosis is almost equally gloomy.

LEATHER-BOTTLE STOMACH (*syn.* LINITIS PLASTICA)

There is a generalised and a localised form of leather-bottle stomach.

When localised, it is the pyloric antrum that is mainly involved. The stomach wall is enormously thickened (fig. 900), and feels, as its name implies, like leather.

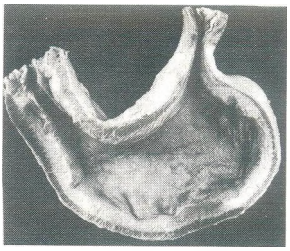


FIG. 900.—Leather-bottle stomach, showing the enormous thickening of the stomach wall.

The enormous proliferation of fibrous tissue involves especially the submucosa, which often appears as a dense layer, mother-of-pearl in appearance. Astonishingly, the whole of the mucous membrane looks and feels quite normal. It is often difficult or impossible to find any evidence of carcinoma, even in serial sections, but metastases are usually found in the regional lymph nodes. When distant metastases occur they are usually found in the liver or the ovary; on rare occasions bones are involved.

FIG. 901.—X-ray of a case of leather-bottle stomach.

The symptoms are those of pyloric obstruction, but the small capacity of the stomach as revealed radiologically by a barium meal (fig. 901) makes the diagnosis tolerably certain.

Treatment.—In the localised variety, partial gastrectomy, and in the diffuse, total gastrectomy, offer hope of prolonging life, but the results, especially in the more common generalised variety, are among the worst, if not *the* worst, of all varieties of carcinoma of the stomach.

Boeck's sarcoid of the stomach occurs occasionally (p. 196), and gives symptoms and X-ray findings identical with those of leather-bottle stomach.

Crohn's Disease (Chap. 49) may affect the stomach.

Carcinoma of the pancreas invading the posterior wall may be misleading.

TREATMENT OF CARCINOMA OF THE STOMACH

No description of the treatment of carcinoma of the stomach is comprehensible without a knowledge of its lymphatic drainage (fig. 902).

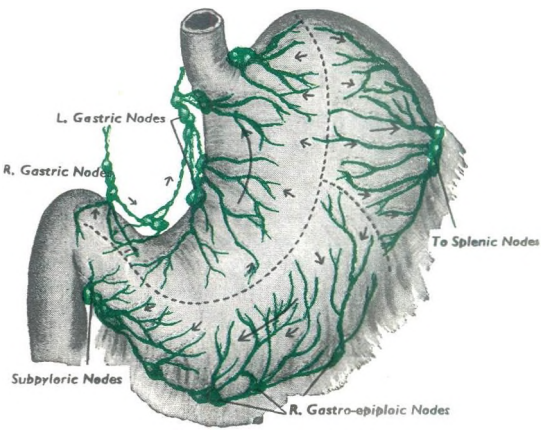


FIG. 902.—The lymphatic drainage of the stomach. (*After Jamieson and Dobson.*)

The lymphatic vessels of the stomach arise in its submucous and subperitoneal layers, and divide into four main sets that accompany corresponding blood-vessels. Each set drains both the anterior and the posterior surfaces of the stomach, and although they intercommunicate, their valves direct the lymph flow as follows :

The lymphatics of the proximal half of the stomach drain primarily into the left gastric and the splenic lymph nodes, and thence into the left, middle, and right superior pancreatic lymph nodes.

The lymphatics of the antrum drain into the right gastric lymph nodes superiorly and the right gastroepiploic and subpyloric lymph nodes inferiorly.

The lymphatics of the pylorus drain into the right gastric (suprapyloric) superiorly and the subpyloric lymph nodes, situated around the gastro-duodenal artery, inferiorly. The efferent lymphatics from the suprapyloric lymph nodes converge on the para-aortic lymph nodes around the cœliac axis, while the efferent lymphatics from the sub-pyloric lymph nodes pass to the main superior mesenteric lymph nodes situated around the origin of the superior mesenteric artery. The lymphatic vessels related to the cardiac orifice of the stomach communicate freely with those of the œsophagus.

Whether or not there is histological evidence of regional lymph node involvement makes a tremendous difference in the prognosis of *completely operable* cases of carcinoma of the stomach. It must be remembered that retrograde (or downward) spread may occur if the upper lymphatics are blocked.

Forty per cent. of patients without regional lymph node involvement survive five years or more, whereas less than 10 per cent. with regional lymph node involvement survive this length of time.

Prognosis.—Of recent years the extent of stomach resected has been on the increase without any coincident benefit. The important fact that has emerged is that, providing a reasonable amount of stomach is resected, it is the histology of the growth which determines the prognosis. The clinical counterpart of this is that the longer the history the better the outlook. Fifty per cent. of cases are inoperable when first seen; in the remainder exploration

is worthwhile, resection being possible in half of them. Five per cent. of the initial cases, and ten per cent. of those having resection, survive for more than five years.

Operative Treatment :

Nomenclature.—A ' curative ' resection consists of removing a block of tissue, including the growth, a margin of at least 4 cm beyond its palpable limits with the stomach unstretched, and the related lymph nodes. The term radical gastrectomy should be reserved for a monoblock dissection fulfilling these requirements, thus bringing the term ' radical ' into line with operations for carcinoma in other situations, e.g. the breast. Radical operations upon the stomach are of three varieties —(1) radical total gastrectomy, (2) radical upper gastrectomy, and (3) radical lower gastrectomy.

Incision.—Where possible an abdominal approach is to be preferred to an abdomino-thoracic (fig. 903). The latter carries an increased risk. The deciding factor for the use of an abdomino-thoracic incision is involvement of the gastro-œsophageal junction.

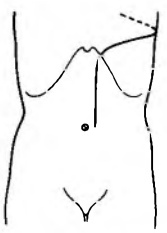


FIG. 903.—Abdomino-thoracic incision started as a left paramedian.

Operability.—As soon as the abdomen is opened, a firm plan, based on the extent of the growth, must be made. This decision is of profound importance and calls for considerable clinical judgement. There are three possibilities—radical surgery, palliative surgery, or the lesion may be inoperable.

Signs of Inoperability.—In general, these depend on the extent of the growth which include (1) fixation to the pancreas or posterior abdominal wall or involvement of the mesentery especially the origin of the superior mesenteric vessels, (2) gross local involvements of lymph nodes leading to fixity and evidence of retrograde spread downwards in the pre-aortic lymph nodes, (3) the presence of secondaries in the liver—an exception may be made in the case of a solitary resectable nodule, (4) peritoneal seedlings either locally or in the pelvis. A Krukenberg tumour does not of itself make the case inoperable.

Radical Operations

1. **Total gastrectomy.**—Indicated for growth involving the upper or middle two-thirds of the stomach. An upper left paramedian incision is made (fig. 903). The spleen with its hilar lymph nodes, the stomach, the splenic vessels, the tail and body of the pancreas are mobilised from left to right *en bloc*, the operation being essentially retroperitoneal. The left gastric artery and the splenic vessels are ligated retroperitoneally. The lymph nodes around the cœliac axis and cardiac orifice are freed, and the lesser omentum is detached as far from the stomach as possible. Similarly, the subpyloric lymph nodes are mobilised and the greater omentum is detached from the transverse colon. The cut edge of the pancreas is closed with sutures and a soft rubber drain is passed down to this site, to be brought out through a separate stab incision. The stomach having been excised, the continuity of the alimentary canal can be restored in the manner shown in fig. 904; this procedure is valuable because it prevents regurgitation of bile and pancreatic juice into the œsophagus, which is so vulnerable to these digestive ferments. An œsophagoduodenostomy should not be done as an *alkaline* œsophagitis so frequently follows. If a thoraco-abdominal incision has been necessary, the diaphragm must be carefully repaired with non-absorbable sutures. Waterseal drainage of the pleural cavity is instituted.

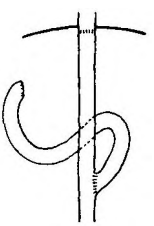


FIG. 904.—Total gastrectomy with œsophago-jejunostomy-en-Y (Roux's method).

Post-operative Care of Total Gastrectomy.—Here nutritional disorders, e.g. weight loss, steatorrhœa, and macrocytic anæmia are the rule. The first two appear early but the anæmia takes five or six years to develop. These patients need frequent small meals of high protein, high calorie content, and regular vitamin B_{12} supplements (100 to 200 μg. monthly).

César Roux, 1857–1934. Professor of Surgery and Gynæcology, Lausanne, Switzerland. Described this method of forming a jejunal conduit, which has so many uses in modern gastro-intestinal surgery, in 1908.

2. **Upper Radical Partial Gastrectomy.**—Indicated for growth involving the cardia and upper third of the stomach. The procedure here is as for total gastrectomy, but the pyloric end of the stomach is preserved and anastomosed to the œsophagus. A pyloroplasty is usually done to prevent post-vagotomy retention (p. 847).

3. **Lower Radical Partial Gastrectomy.**—This operation for carcinoma of the pyloric end of the stomach involves separation of the greater omentum from the colon in its entirety, separation of the lesser omentum from the liver, freeing the subpyloric lymph nodes, and removal of the spleen and body and tail of the pancreas. The left gastric vessels and the right gastro-epiploic vessels are divided at their origin, stripping all fatty and lymphatic tissue around them towards the stomach. The operation is then completed by a Billroth I anastomosis.

Palliative Operations.

(1) *Palliative Gastro-jejunostomy.*—For irremovable pyloric carcinoma gastro-jejunostomy should be performed some distance from the tumour, so that the advancing growth will not invade the stoma, at any rate for some time.

(2) *Exclusion Operation.*—Excluding the growth from the pathway of the food is often beneficial, especially where an antral growth is fixed and inoperable and the upper part of the stomach is free. The risk of bleeding is reduced, appetite is increased, and vomiting ceases. The stomach is transected 2·5 cm proximal to the edge of the growth, the distal end is closed with non-absorbable sutures, and a gastro-jejunostomy is done. If the pylorus is completely occluded, this operation is contraindicated.

(3) For *obstruction of the cardia* considerable relief can be obtained by the introduction of a plastic tube (p. 809). This is a most valuable manœuvre in those patients whose days are numbered. It is preferable to a palliative œsophago-jejunostomy which carries a definite operative mortality in these debilitated patients.

SARCOMA OF THE STOMACH

Sarcoma of the stomach accounts for about 1 per cent. of gastric neoplasms.

Lymphosarcoma is the most common variety. In contradistinction to lymphosarcoma of the small bowel which is multiple, this tumour is usually solitary. In all inoperable cases a piece of the growth should be removed for histological examination. Although radio-sensitive, the best treatment, if circumscribed, is gastrectomy. Chemotherapy may be useful.

Leiomyosarcoma.—The first symptom may be a massive hæmatemesis and/or melæna. The presence of a barium-filled sinus extending into the tumour visualised by X-rays is extremely characteristic of leiomyosarcoma. Unlike a carcinoma of similar dimensions, the gastric acidity is unchanged. Usually these tumours metastasise late, and, when they do, secondary growths are often found in the liver. The treatment is partial gastrectomy. The tumour is more vascular than a carcinoma. It is stressed particularly that leiomyosarcomata should be attacked with vigour, regardless of the magnitude of the procedure, as the results are so much more gratifying than those of carcinoma of the stomach. At least 50 per cent. of the patients are alive five years after the operation.

Neurofibrosarcoma behaves precisely as a leiomyosarcoma, from which it is distinguished only by histological examination.

Fibrosarcoma, derived from the subserosa, is the rarest variety and the least malignant; it sometimes gives rise to a tumour of immense size, and in the female such a tumour is liable to be mistaken for an ovarian cyst.

DUODENAL NEOPLASMS

Neoplasms of the duodenum are very rare. Adenomas have been reported as causes of severe melæna. Carcinoma of the first part of the duodenum is so rare that many of great experience have never seen a case. There is an apparent bar to spread of carcinoma of the pyloric end of the stomach into the duodenal mucosa, but there is no such obstacle to the spread in the subserous layer which, indeed, occurs with some frequency—hence the necessity for excising 2·5 cm of the duodenum

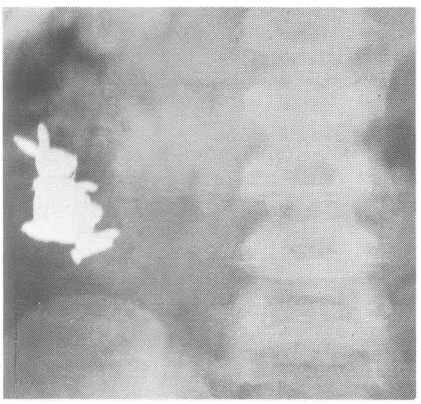


FIG. 905.—Embedded in Normacol, this foreign body was passed naturally in three days.

when performing radical lower partial gastrectomy. Carcinoma of the second and third parts occurs. It may occlude the ampulla of Vater.

MISCELLANEOUS CONDITIONS OF THE STOMACH AND DUODENUM

Foreign Bodies in the Stomach

A variety of ingested foreign bodies reach the stomach. Fortunately, for the most part they are opaque to X-rays (fig. 905). Sharply pointed or long objects are best removed promptly by gastrotomy because they have difficulty in negotiating the curves of the duodenum. Rounded, smaller foreign bodies may be left to pass along the natural passages. Suitable doses of Normacol form a gelatinous pabulum, in which the article becomes embedded during its transit along the alimentary tract. X-ray examinations should be undertaken sparingly.

Hair-ball of the Stomach (Trichobezoar).—An example of this rare condition is usually to be found on the shelves of pathological museums. Trichobezoars occur almost exclusively in females, and in 80 per cent. of cases the patient is a psychiatric case. Trichobezoars can give rise to gastro-duodenal ulceration leading to hæmatemesis, perforation, peritonitis, or obstruction. A trichobezoar shows well on radiography. The treatment is removal by gastrotomy.

Acute Dilatation of the Stomach

This dangerous complication can occur after any operation or even after such trauma as a fractured femur or the application of a plaster-of-Paris jacket.

Clinical features. Muffled hiccoughs, rising pulse rate, and shock, may occur up to three days after operation or accident. Vomiting starts, and unless prompt treatment is given, characteristic effortless vomits of dark, watery fluid occur. Inhalation of vomitus may cause cardiac arrest.

Treatment.—1. The stomach is emptied and kept empty by aspiration through a nasogastric tube.

2. Fluid balance is restored by intravenous infusion. After replacing lost chloride with normal saline, dextrose-saline is given. Hypo-potassæmia must be avoided (p. 92).

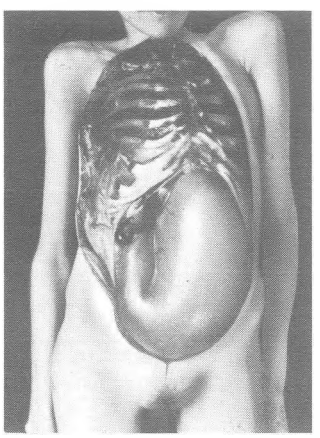


FIG. 906.—Acute dilatation of the stomach seen at necropsy.

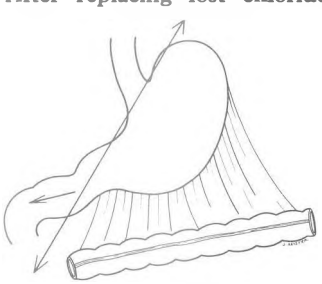


FIG. 907.—Volvulus of stomach —common axis of rotation.

Volvulus of the Stomach

Rotation of the stomach usually occurs around the axis made by its two fixed points, i.e. the cardia and the pylorus (fig. 907). Although the rotation can occur in a horizontal or vertical direction, in its common form the greater curve with the colon moves upwards to lie under the cupola of the left diaphragm (fig. 908). An important predisposing

cause of this event is eventration of the diaphragm from whatever cause. It is probable that the colon moves up first and takes the stomach with it. The condition is usually intermittent, but it may present in an acute form. The patient takes a small amount of food and he feels full; there is slight epigastric pain and retching. He

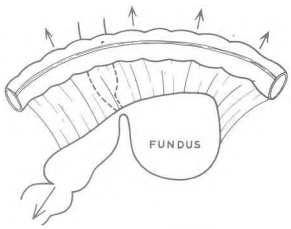


FIG. 908.—Volvulus of stomach—colon passes upwards and greater curve of stomach is inverted.

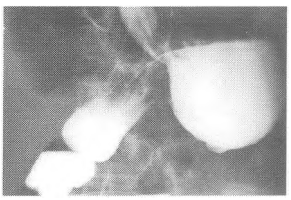


FIG. 909.—Volvulus of stomach—barium meal findings.

has to wait awhile, and he may find that if he lies flat he can then eat some more. The diagnosis is established by a barium meal (fig. 909). It is often impossible to pass a stomach tube.

Treatment.—Operation is the only satisfactory form of treatment. The greater curvature of the stomach must be completely freed from the colon by division of the gastro-colic omentum. It is probable that this of itself is adequate. A further safeguard, however, consists of fixing the greater curve to the duodeno-jejunal flexure (or better, the fourth part of the duodenum), as in a posterior gastro-enterostomy but without a stoma.

Where the rotation occurs as the result of and into a large hiatal defect, the essential treatment consists of closure of the hiatus around the œsophagus.

Gastric Tetany

Tetany due to alkalosis may occur where there is long-continued vomiting, gastric aspiration, or excessive ingestion of alkali. The extracellular alkalosis is always accompanied by cellular acidosis, potassium depletion, and a fall in the blood calcium (parathyroid tetany, p. 646). Clinically there are muscle spasms in the extremities, shallow respiration, and cyanosis. The stomach must be kept empty by a tube and the blood electrolytes estimated so that the specific deficiencies may be rapidly corrected. Whilst these are being done, continuous intravenous dextrose saline with calcium gluconate should be given. Even when the chloride deficit is corrected, the alkalosis may persist and this is indicative of the need for potassium (p. 92).

Prolapsing Gastric Mucosa.—This is an X-ray finding. Hypertrophied gastric mucosa prolapses through the pylorus (fig. 910(inset)). Pyloric obstruction has been attributed to this phenomenon.

Giant rugal hypertrophy of the stomach is a descriptive name given to a condition which may manifest itself clinically as a protein-losing gastro-enteropathy. Recognition of this feature may necessitate gastrectomy. The thick folds can be demonstrated radiologically, seen at gastroscopy, and palpated at laparotomy. There is not the slightest evidence that this condition is premalignant, nor is it inflammatory. The ætiology is unknown (Johnson).

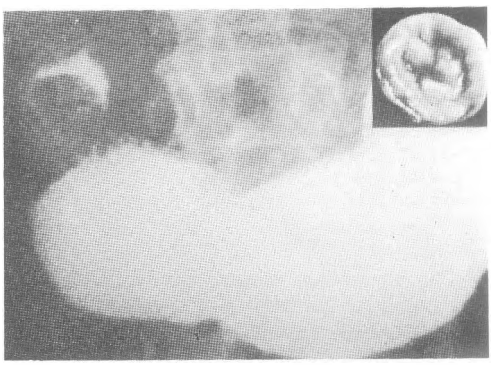


FIG. 910.—Prolapsing gastric mucosa. Inset.—The prolapse viewed from the duodenal aspect of the pylorus. (*L. H. Appleby, F.R.C.S., Vancouver*).

H. Daintree Johnson, Contemporary. Surgeon, Royal Free Hospital, London.

Duodenal Diverticulum

There are two varieties:

1. **Primary diverticulum** of the second and third parts of the duodenum. They occur on the concave border, are usually single but may be multiple, and commonly arise at the portal of entry of the blood-vessels into the duodenal wall. In 90 per cent. of cases they are an incidental finding during the course of a barium meal. It is wise not to incriminate them as a cause of symptoms (fig. 911) but to make a thorough search for pathology elsewhere. Rarely, a large diverticulum near the ampulla of Vater may cause jaundice. In addition, if a diverticulum is persistently tender when visualised during screening or if it retains barium for days, then it may be justifiable to operate. The duodenum is mobilised, turned medially, and the diverticulum dissected free. The duodenum is then opened opposite the diverticulum which is seized, inverted into the lumen, and is ligated. The duodenum is then closed and the fascia over the site of the diverticulum oversewn. The duodenum is deficient of peritoneum in this area and the operation may be complicated by a fistula.

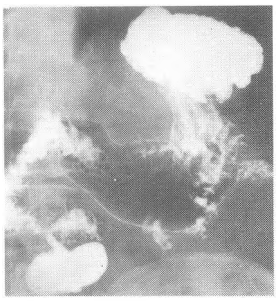


FIG. 911. — Duodenal diverticulum arising from the third part of the duodenum.

2. **Secondary Diverticula.**—These occur in the first part of the duodenum and are the result of scarring following duodenal ulceration. They are responsible for the trifoliate duodenum seen on X-ray (fig. 912).

Chronic duodenal ileus is a rare condition which may mimic pyloric stenosis. The duodenum is dilated up to the superior mesenteric vessels. The dilated segment has a characteristically smooth outline (fig. 913). It is a radiological diagnosis rather than a clinical one. Some advocate duodeno-jejunostomy for this condition, but the results are disappointing.

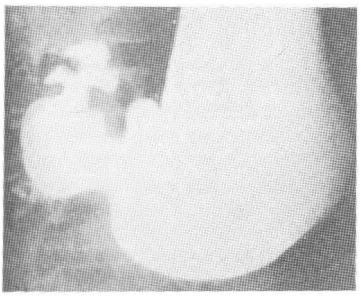


FIG. 912.—Duodenal ulcer showing typical trefoil or shamrock deformity. *(Dr. G. Stachurko, London.)*

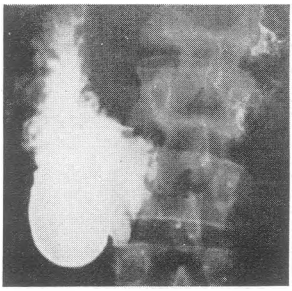


FIG. 913.—Duodenal ileus. Note the abrupt obstruction to the third part of the duodenum.

Duodenal Fistula

The most usual causes are as follows:

1. As a complication of gastrectomy (p. 843).
2. An abscess connected with a perforated duodenal ulcer.
3. Traumatic rupture of the duodenum (see below).
4. As a complication of transduodenal sphincterotomy, right nephrectomy, or right colectomy.

The fistula discharges bile and pancreatic juice which cause excoriation of the skin. Excessive discharge may quickly lead to dehydration, electrolyte imbalance, and hypoproteinæmia. The management consists essentially of three procedures:

1. Protection of the skin by 1 per cent. HCl in zinc oxide cream.

2. Continuous and efficient suction drainage of the fistula (fig. 1027) which may be facilitated by gravity—the patient lying prone.

3. Replacement therapy—when the fistula drainage is small, closure may be confidently expected within a few days using simple intravenous therapy. If the discharge is profuse, a severe illness can be prevented by a simple feeding jejunostomy on the left side of the abdomen. The valuable pancreatic and biliary juices may be returned to the jejunum by this route.

Traumatic Rupture of the Duodenum.—This is an increasingly common problem in hospitals near a fast main road (see seat belt syndrome, Chap. 48). If the rupture is extraperitoneal the initial symptoms are often slight and the condition is overlooked until an abscess forms. When the abscess is opened a fistula develops. With intraperitoneal rupture the clinical features resemble a perforated duodenal ulcer. Laparotomy is performed and the tear can usually be sutured.

Traumatic fistulæ of the first part usually close spontaneously—those of the second and third part are formidable indeed. They present such a grave problem that it is worth considering a duodeno-jejunal anastomosis to overlie the defect if this cannot be closed easily at the primary operation.

CHAPTER 44

THE SPLEEN

Surgical Physiology.—The functions of the spleen are not wholly understood.[1] In general they may be considered as:

1. **Destruction of effete and misshapen erythrocytes** and possibly of platelets. It is believed that during their sojourn in the spleen the lipoid coverings of ageing erythrocytes are rendered less strong. If red blood cells from a patient with hereditary spherocytosis are transfused into a normal person, they are destroyed in fifteen days. When transfused into a splenectomised person, they have a normal life span. After splenectomy the liver, bone marrow, and lymph nodes deal with effete red cells.
2. **Formation of erythrocytes** during fœtal life. In case of need, this function may be resumed during adult life.
3. **Maintenance of a reserve of erythrocytes.**—Anoxia, caused by high altitudes, hæmorrhage, carbon monoxide poisoning, or prolonged exercise, stimulates the spleen to contract, thereby causing reserve red cells idling in its mesh-work to be conscripted into circulation as oxygen-carriers. This reaction may be useful in some animals like dogs, but the spleen is relatively too small to be of much use in man (containing about 1 per cent. of the blood volume).
4. **Iron storage.**
5. **Phagocytosis of foreign substances** by the reticulo-endothelial cells of the spleen is well illustrated by the high concentration of opaque substance in the spleen in patients who have received an injection of thorium. Particulate matter and organisms, bacterial, fungal, protozoal are removed from the circulation. This function is also evident in the lipoid dystrophies, when phagocytosis of abnormal lipoids is the main cause of splenic enlargement.
6. **Antibody Production.**—It would appear that the spleen plays a part in the formation of antibodies. There is increasing evidence that splenectomised children are less able to combat severe infection than those with a normal spleen, and they require close observation so that immediate and energetic steps can be taken in cases of pyogenic infection.

PHYSIOLOGICAL EFFECTS OF SPLENECTOMY

1. **Splenunculi** (accessory spleens), if present, **hypertrophy.**
2. **Changes in the blood.**—Initially:

(*a*) Morphological changes in the erythrocytes, viz. target cells present and some containing nuclear remnants (Howell-Jolly bodies).

(*b*) Leucocytosis.

(*c*) Increased platelet with a tendency to:

(*d*) Increased coagulability.

After two months lymphocytosis is present invariably. *After many months* there is moderate eosinophillia, and the basophils increase in number.

Clinical Effects of Splenectomy.—The spleen is not essential for life. However, after its removal there is evidence of increased vulnerability to infection in infants and young children under the age of 3 years. The pneumococcus is the organism most commonly isolated. Adults do not appear to be similarly affected.

SPLENUNCULI (*syn.* ACCESSORY SPLEENS)

Instead of a spleen, some fishes are provided with islets of splenic tissue scattered through the cœlom. In man, accessory spleens, in common with the main organ, are

[1] Organum plenum mysterium (*Latin*) = Organ full of mystery (*Galen*).

William Henry Howell, 1860–1945. Professor of Physiology, Johns Hopkins University, Baltimore, U.S.A.
Justin Marie Jules Jolly, 1870–1953. Professor of Histophysiology, College de France, Paris.
Galen, 130–200. First experimental physiologist and personal physician to the Roman emperors Marcus Aurelius Commodus and Septimus Severus.

derived from the dorsal mesogastrium. One or more are present in 25 per cent. of children (Jolly), but they are to be found in only 11 per cent. of adults (Adami). Obviously then, as maturity is reached accessory spleens tend to disappear. Their usual locations are as follows:

1. In contact with the spleen (fig. 914), usually near the hilum; more than 50 per cent. are found in this situation.

2. Related to the splenic vessels and behind the tail and body of the pancreas—about 30 per cent.

3. The remainder are found in the splenic ligaments, the greater omentum, mesentery, mesocolon, and abutting on the left ovary or testis (carried thither during descent of the gonad).

When splenectomy has been performed for conditions that are known to be cured by removal of the spleen, return of symptoms after an interval of freedom is the result of hypertrophy of (usually) one accessory spleen. Occasionally a morsel of the spleen left attached to the pedicle during splenectomy continues to grow, and may be said to form a new spleen. Therefore not only must the whole of the enlarged spleen be removed, but, except in cases of rupture, the sites of an accessory spleen should be examined meticulously and any splenunculi present excised.

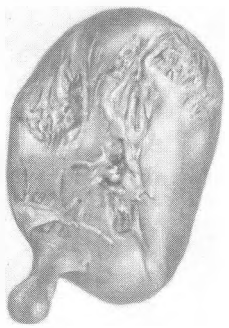


FIG. 914.—Spleen with a splenunculus.

Splenosis is a rare condition, and nothing to do with splenunculi. Fragments of splenic tissue, spilled at the time of bygone rupture of the spleen, are found disseminated widely over the peritoneum, having thrived in situations other than those to be expected on developmental grounds. Sometimes there are scores of them. Splenosis does not give rise to symptoms, and is found only at laparotomy or necropsy. It is liable to be confused with endometriosis of the peritoneum, carcinomatosis or multiple angiomata.

SPECIAL METHODS OF INVESTIGATING THE SPLEEN

1. **Radiography.**—In plain films of the upper abdomen the spleen is visible in whole or part, especially when the stomach and the splenic flexure contain gas, and the patient is not obese. These findings, repeated at intervals, are good evidence that the spleen is unruptured (p. 862).

2. **Splenic Puncture.**—Provided the following stipulations are adhered to rigorously, splenic puncture can prove a diagnostic aid.

(*a*) Only definitely palpable spleens should be punctured.

(*b*) No patient with a hæmorrhagic tendency should undergo splenic puncture.

(*c*) The puncture should not be performed in the presence of infective splenomegaly, or of a tender spleen.

(*d*) Unconscious patients and children must never undergo splenic puncture.

Technique.—An ordinary lumbar puncture needle equipped with a guard to regulate the depth of the thrust should be employed. With the patient lying on his back, the site puncture (fig. 915) is infiltrated with local anæsthetic. The puncture must be performed during full inspiration, with the guard set to the required depth. Strong aspiration with the syringe is performed for several seconds. Before the needle is withdrawn the negative pressure must be released. After splenic puncture the patient is kept in bed for six hours, as a precaution against hæmorrhage.

3. **Splenoportography** can be carried out under general anæsthesia prior to laparotomy, or under local anæsthesia. The needle is inserted as above. After withdrawal of the stilette of the lumbar puncture needle, blood drips from the lumen; only after this has been demonstrated is the dye-filled syringe connected, and 30–50 ml of Hypaque injected *rapidly*. Apnœa, voluntary or controlled, must be maintained during insertion of the needle and throughout the injection of the dye. If this stipulation is not adhered to rigorously, a laceration of the spleen as the spleen moves with the diaphragm, and possibly serious hæmorrhage, may result. Splenoportography permits visualisation of the spleen and the portal venous system (p. 900). The first exposure is made towards the end of the injection, and three or four exposures follow subsequently.

Hugh Reginald Jolly, Contemporary. Pædiatrician, Charing Cross Hospital, London.
John George Adami, 1862–1926. Strathcona Professor of Pathology, McGill University, Montreal, Canada.

CONGENITAL ABNORMALITIES OF THE SPLEEN

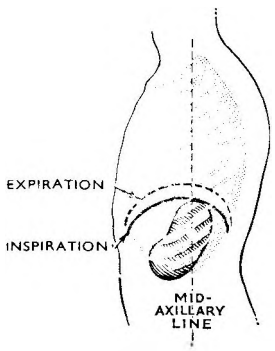


FIG. 915.—The puncture is made in the 9th intercostal space in the mid-axillary line during full inspiration.

Congenital absence of the spleen is exceedingly rare, and when it occurs it is nearly always associated with a gross congenital cardiac abnormality (usually a septal defect) and also the presence of accessory lobes to the lungs, and sometimes lobulation of the liver.

Movable Spleen (*syn.* Wandering Spleen).—The spleen, well supported by its two ligaments (the gastrosplenic and lieno-renal), and resting on a third (the costocolic), which acts as a shelf, is the one abdominal organ that maintains its correct anatomical position in general visceroptosis. Movable spleen is a congenital abnormality that occurs more often in women. The condition is of great rarity, and the diagnosis is seldom made correctly.

Torsion of the splenic pedicle sometimes complicates the wandering organ. The torsion may be acute or chronic, acute cases presenting the symptoms of an intra-abdominal catastrophe. Chronic torsion may result in atrophy of the spleen, and after a period of indefinite abdominal discomfort all symptoms abate.

RUPTURE OF THE SPLEEN

Usually a ruptured spleen occurs as the result of an industrial or traffic accident of the crushing or run-over type. Blows on the abdomen or the left lower thorax and falls on to a projecting object are other causes of rupture. When rupture of the spleen occurs as a result of a traffic accident, associated injuries are commonly present. A number of cases of spontaneous rupture of a normal spleen have been reported, but in all probability in some of these there has been an accident that has been forgotten or suppressed by the patient. The spleen in infectious mononucleosis ruptures easily.

Cases of ruptured spleen may be divided into three groups:

1. **The Patient Succumbs Rapidly, Never Rallying from the Initial Shock.**—This type is comparatively rare in temperate climates; tearing of the splenic vessels and complete avulsion of the spleen from its pedicle[1] give rise to the symptoms which characterise this group. In countries where malaria is rife, splenic rupture is often rapidly fatal, and advantage was once taken of this knowledge by murderers in the Orient, who achieved their end by digging the victim beneath the left ribs with an implement known as a larang (fig. 916).

FIG. 916. — A larang, which, being interpreted, means 'forbidden'. (*After J. Johnston Abraham.*)

2. **Initial shock; recovery from shock; signs of a ruptured spleen** is the usual type seen in surgical practice. After the initial shock has passed off, there are signs which point to an intra-abdominal disaster, and by correlating these signs it is often possible to arrive at a correct preoperative diagnosis.

General signs of internal hæmorrhage are inconstant. Perhaps the most helpful are increasing pallor, a rising pulse-rate, sighing respiration, and restlessness.

Local Signs.—(*a*) Abdominal rigidity is present in more than 50% of cases; it is most pronounced in the left upper quadrant.

[1] Favoured by adhesion of the spleen to the diaphragm.

(*b*) Local tenderness is found constantly.

(*c*) Shifting dullness in the flanks is often present.

Ballance's sign is positive in about 25% of cases. There is a dull note in both flanks, but on the right side it can be made to shift, whereas on the left it is constant. The interpretation is that there is blood in the peritoneal cavity, but the blood in the neighbourhood of the lacerated spleen has coagulated.

(*d*) Abdominal distension commences about three hours after the accident, and is due to intestinal paresis, an early stage of paralytic ileus.

(*e*) Kehr's sign is pain referred to the left shoulder. There may be hyperæsthesia in this area. This sign is present very often, especially if sought a quarter of an hour after elevation of the foot of the bed, and is due to blood in contact with the under-surface of the diaphragm, the pain being mediated through afferent fibres in the phrenic nerve.

(*f*) Rectal examination frequently reveals tenderness and sometimes a soft swelling, due to blood or clot in the rectovesical pouch.

3. **The Delayed Type of Case.**—After the initial shock has passed off, the symptoms of a *serious* intra-abdominal catastrophe are postponed for a variable period up to fifteen days, or even more. As a rule it is only a matter of minutes to an hour or so, during which time the patient often appears to have recovered from the blow. Thus a Rugby footballer may continue to play after a short rest, only to collapse later from internal hæmorrhage.

Considerable delay of serious intraperitoneal bleeding is explained in one of the following ways:

(*a*) The greater omentum, performing its well-known constabulary duties, shuts off that portion of the general peritoneal cavity in the immediate vicinity of the bleeding (fig. 1064 (*c*), p. 982).

(*b*) A subcapsular hæmatoma forms and later bursts.

(*c*) Blood-clot temporarily sealing the rent becomes digested by escaping ferments from the lacerated tail of the pancreas. What may happen is that a patient with a suspected rupture of the spleen is taken into hospital; the symptoms abate, and in due course he is allowed up. Suddenly he collapses, perhaps in the lavatory while straining at stool. At other times fresh hæmorrhage is heralded by a rising pulse-rate, increasing pallor, advancing to air-hunger and collapse. Such disasters can, and should, be prevented by careful re-examination and radiography.

Radiography.—A normal, well-outlined spleen is a reliable negative sign, but radiographs should be repeated if the decision not to operate *pro tem.* is taken. Indeed, every patient with a left-sided thoracic injury, especially where a lower rib is fractured, should be kept under radiological observation. When there is delayed rupture, or when the symptoms and signs are atypical, radiography often can help considerably in diagnosis. In decreasing frequency, the radiological signs of ruptured spleen are: (1) Obliteration of the splenic outline; (2) Obliteration of the psoas shadow; (3) Indentation of the left side of the stomach air-bubble; (4) Fracture of one or more lower ribs on the left side (present in 27% of cases); (5) Elevation of the left side of the diaphragm; (6) The presence of free fluid between gas-filled intestinal coils.

Treatment of Rupture of the Spleen.—Immediate laparotomy and splenectomy is the only reliable course. Blood is evacuated and, after injury to other viscera has been excluded, the abdomen closed completely.

Sir Charles Alfred Ballance, 1856–1936. Surgeon, St. Thomas's Hospital, London.
Hans Kehr, 1862–1916. Surgeon, Halberstädt, later Berlin. He died of septicæmia contracted during his work.

When the organ is damaged by a stab wound or missile penetrating the left pleural cavity, access to the spleen is best obtained by excising the thoracic and diaphragmatic wounds, and enlarging the opening in the diaphragm. Blood transfusions should be given as required.

Recently in children splenorrhaphy is advised and several techniques of repairing the torn spleen are described, but this should *not* be attempted unless the surgeon has experience with the technique.

Post-operative Complications

Acute Dilatation of the Stomach.—This preventable complication should not be allowed to occur. Routine gastric aspiration for at least 24 hours post-operatively has banished it.

Hæmatemesis occasionally occurs, possibly due to transient congestion of the gastric mucosa following ligation of the vasa brevia veins.

Left basal atelectasis, sometimes accompanied by left pleural effusion, is particularly common, and is due to bruising of the diaphragm at the time of the accident, or to trauma during splenectomy. Breathing exercises and forced coughing go a long way in preventing atelectasis.

Paralytic ileus (p. 1052) is rather common.

Peritoneal effusion or wound disruption may be secondary to concomitant injury of the tail of the pancreas. If there is any question of damage to the tail of the pancreas at operation, the area must be drained.

'*Rheumatism*' due to bone marrow activity (p. 860).

Persistent hiccough is the result of irritation of the under-surface of the left side of the diaphragm. It may indicate a collection of blood there.

Post-splenectomy Sepsis.—Septicæmia, pneumonia and meningitis are reported weeks or years after splenectomy, particularly in children. Such patients have an increased fatality rate over the normal. Pneumococcal sepsis is very deadly.

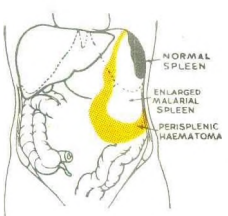


FIG. 917.—Normal spleen, malarial spleen, perisplenic hæmatoma. Note that the splenic contour is lost when the capsule has ruptured.

RUPTURE OF A MALARIAL SPLEEN

As has been mentioned, in tropical countries this is a frequent catastrophe. The delayed type of rupture (following a trivial injury) is also very common, and the patient is admitted with a perisplenic hæmatoma (fig. 917). If splenectomy can be performed before the hæmatoma bursts into the general peritoneal cavity, the prognosis is less grave.

The operation is considerably more difficult than in the case of a ruptured normal spleen. Surgeons with tropical experience have surmounted these difficulties by ligating the splenic vessels as they run along the superior border of the body of the pancreas (fig. 918), before disturbing the hæmatoma (Andreasen).

ANEURYSM OF THE SPLENIC ARTERY

Perhaps due to its tortuosity or to its liability to become arteriosclerotic (sometimes arteriosclerosis affects the splenic artery and no other), the splenic artery is more frequently the site of an aneurysm (fig. 918) than any other artery in the abdomen, the abdominal aorta excepted.

As a rule the aneurysm is symptomless unless it ruptures. It is discovered accidentally, on X-raying the upper abdomen, as a ring of calcification in the line of the artery. Exceptionally it gives rise to upper abdominal pain, in which event the condition has been diagnosed pre-operatively: (*a*) By a bruit over the left hypochondrium; (*b*) By radiography. An unruptured splenic aneurysm may be discovered at operation.

Rupture of the aneurysm occurs somewhat more often in women than in men, and it

Anthony Turner Andreasen, Contemporary. Surgeon, Kampala, Uganda.

is noteworthy that approximately 25 per cent. of the female patients are pregnant, and usually the pregnancy is advanced (six to eight months). In most instances rupture occurs into the peritoneal cavity, and the symptoms are precisely those of delayed rupture of the spleen (p. 863). During the stage of perisplenic hæmatoma (as in spontaneous rupture of the spleen), bursting often occurs in two stages and either or both the diagnostic signs set out above are frequently present. Hæmorrhage into the stomach or the retroperitoneum has been recorded.

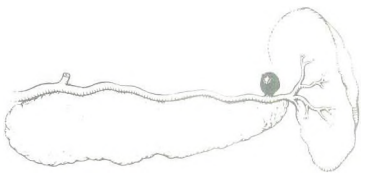


FIG. 918.—Usual position of a splenic aneurysm. (*After O. E. Owen, Caernarvon, Wales.*)

Treatment.—So dangerous is an aneurysm of the splenic artery that unless the patient is very old or infirm, excision of the sac should be advised in all cases. Rupture of the aneurysm demands urgent laparotomy and blood transfusion. The lesser sac must be opened and both the proximal and distal portions of the splenic artery are ligated. Proximal ligation alone is useless, for the distal end bleeds furiously. Splenectomy, although often performed, is not obligatory unless it is obvious that the arterial ligation has deprived the spleen of all its blood supply. Rupture of an aneurysm of the splenic artery carries a high mortality.

FIG. 919.—Infarct of the spleen.

INFARCTION OF THE SPLEEN (Fig. 919)

The patient, who is often suffering from subacute infective endocarditis or auricular fibrillation, is seized with agonising pain in the left hypochondrium. Symptoms usually mimic those of a perforated gastric ulcer, but the heart lesion helps the clinician to elucidate the diagnosis. When the embolus is aseptic the symptoms pass off in a few days. Infarct of the spleen has become a problem of air travel. At a height of over 10,000 feet patients with sickle-cell anæmia (a disease confined to Negroes) frequently sustain a splenic infarct. Patients with other hæmatological conditions often associated with splenomegaly such as chronic leukæmia or myelofibrosis may experience painful episodes of splenic infarction.

In bacterial endocarditis, the infection may be maintained by an infected infarct of the spleen. In a few instances splenectomy has proved to be the deciding factor in curing the patient of this serious condition.

ENLARGEMENT OF THE SPLEEN

The spleen is a meeting-place of medicine and surgery. The following is a useful table of the many causes of enlargement of the organ:

1. Infective	*Bacterial*—	Typhoid and Paratyphoid.
		Typhus.
		Anthrax.
		Tuberculosis.†
		Septicæmia.
		Abscess of the spleen.‡
	Spirochætal—	Weil's disease.
		Syphilis.
	Viral—	Infectious mononucleosis
		Psittacosis.
	Protozoal—	Malaria.
		Schistosominsis (Egyptian splenomegaly)†
		Trypanosomiasis
	Parasitic—	Kala-azar.
		Hydatid cyst.*

Adolph Weil, 1848–1916. Physician, Dorpat (Tartu), Estonia.

2. Blood diseases	Myelofibrosis.‡ Acute leukæmia. Chronic leukæmia (lymphocytic or granulocytic).‡ Pernicious anæmia. Polycythæmia vera. Erythroblastosis fœtalis. Hereditary spherocytosis.* Acquired hæmolytic anæmia.† Thrombocytopenic purpura.* Mediterranean anæmia (thalassæmia).‡ Hypersplenism and Tropical splenomegaly.†
3. Metabolic	Rickets. Amyloid. Porphyria. Gaucher's disease‡ (p. 875).
4. Circulatory	Infarct— Portal hypertension (p. 897). Infective endocarditis.‡ Mitral stenosis. Occlusion of the portal vein— Thrombophlebitis.† Neoplastic, e.g. carcinoma of tail of pancreas.‡
5. Collagen Diseases	Still's disease (p. 237). Felty's syndrome.† (p. 875).
6. Non-parasitic cysts	Congenital.* Acquired.*
7. Neoplastic	Angioma.* Primary fibrosarcoma.* Hodgkin's disease.‡ (p. 191). Lymphosarçoma and other malignant lymphomas.‡

* Benefited by splenectomy. † Often benefited by splenectomy.
‡ Splenectomy sometimes indicated.

In connection with the above Table, the following points should be noted. In thrombocytopenic purpura the spleen, although somewhat enlarged, is seldom palpable. In psittacosis the enlarged spleen can be palpated regularly after the first few days of the illness; this enlargement is helpful in differentiating the condition from other varieties of pneumonia. In portal hypertension the spleen is enlarged secondary to hepatic cirrhosis or portal vein thrombosis (p. 897).

PERISPLENITIS

Thickening of the splenic capsule has been noted at necropsy, usually in subjects with advanced hepatic cirrhosis. The best example of perisplenitis occurs in association with multiple serositis (Pick's disease). In this obscure malady the spleen appears as though covered with sugar icing. The covering is hard, structureless, and gives staining reactions similar to those of hyaline cartilage.

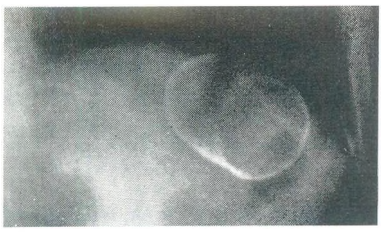


FIG. 920.—Splenic cyst, about the size of a grapefruit. (*Professor Carl Krebs, Aarhus, Denmark.*)

CYSTS OF THE SPLEEN

Non-parasitic cysts of the spleen are of two varieties: (*a*) *True cysts*, comprise 10 per cent. of the total, and are dermoids. It is difficult to understand how the spleen, which is mesochymal in origin, should contain epithelium. (*b*) *False cysts*, which often reach a large size and are more common than true cysts (fig. 920). They have no lining membrane, and usually are due to past

Friedel Pick, 1876–1926. Professor of Laryngology, Prague, Czechoslovakia. (*See footnote p. 769.*)

hæmorrhage, and contain blood-stained fluid and cholesterol crystals. Their origin is likely to be an ancient subcapsular hæmorrhage or degeneration of an infarcted area. In a number of cases there is a history of malaria. On very rare occasions cysts may be multiple.

Hydatid Cyst of the Spleen.—Cases of hydatid cyst have been reported.

A cyst of the spleen should be treated by splenectomy.

HÆMOLYTIC ANÆMIA

Two forms of this disorder may need to be considered surgically: (1) congenital (hereditary spherocytosis) and (2) acquired.

1. **Hereditary Spherocytosis.**—The essential lesion is a congenital increased permeability of the red cell membrane to sodium. As the sodium leaks into the cell its osmotic pressure rises, it swells and becomes more spherical and in consequence more fragile. The mechanism by which the cell rids itself of sodium—the sodium pump—has to work harder. This has two consequences (*a*) there is greater loss of membrane phospholipid and so a weakening of the membrane which thus becomes more fragile and (*b*) the energy and the oxygen requirements increase. These requirements are particularly difficult to satisfy in the spleen where there is a deficiency of both glucose and oxygen in the pulp. Thus it is here that a large number of red cells are destroyed and therefore splenectomy helps the patient by reducing red cell destruction.[1] Splenectomy does not, of course, cure the congenital red cell membrane defect but it lessens the anæmia and makes the red cell survival time normal. The defect is transmitted by either parent as a Mendelian autosomal dominant.[2] Males and females are equally affected.

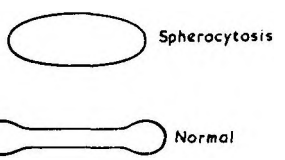


FIG. 921. — Diagrammatic representation of normal (biconcave) and biconvex red cells of spherocytosis.

The circulating bilirubin is unconjugated with glycuronic acid, is attached to albumin and is not excreted in the urine (acholuric jaundice).[3]

The spleen enlarges in the disease because of its increased work.

Clinical Features.—Once the disease manifests itself, spontaneous remissions are almost unknown. As a rule the patient is pale and has jaundice, which varies in intensity and at its height is of a pale daffodil hue. Careful enquiry must be made into similar disease in other members of the family. In established cases lassitude and undue fatigue are seldom absent, but they vary with the amount of hæmolysis that is proceeding.

Sometimes the patient is born jaundiced, or becomes so early in life. In certain families the disease is characterised by severe crises of red-blood-cell destruction; thus, with the onset of a crisis, an erythrocyte count may fall from $4\frac{1}{2}$ millions to $1\frac{1}{2}$ millions in less than a week. Such crises are characterised by the sudden onset of pyrexia, abdominal pain, nausea, vomiting,

[1] 'The spleen dislikes, distrusts, and devours spheroidal red cells' (A. Rendle Short).

[2] In 1865 Mendel described 'dominant' and 'recessive' traits in hybrids. His work passed unnoticed for thirty-five years.

[3] Although there is excessive breakdown of red cells with transformation of liberated hæmoglobin to bilirubin, the bilirubin compound so produced is excreted by the liver and not by the kidneys, thus favouring the formation of pigment gall-stones.

Arthur Rendle Short, 1880–1953. Professor of Surgery, Bristol, England.
Gregor Johann Mendel, 1822–1884. Augustinian Monk and Naturalist, who later became Abbot of Brunn, Austria, (now Brno, Czechoslovakia). His work on inheritance was published in 1866 but passed almost unnoticed during his lifetime.

and extreme pallor, followed by increased jaundice. These crises may be so severe as to cause death in infancy or childhood. They may be precipitated by acute infections. More usually the jaundice, although variable, is very mild, and may not appear until adolescent or even adult life. In adult cases there is often a history of attacks of gall-stone colic; indeed, 68% of untreated patients over the age of ten years have pigment stones in the gall-bladder. Every child with gall-stones should be investigated for evidence of hereditary spherocytosis.

On examination the spleen is large, and in thin subjects it can be palpated easily. Sometimes the liver is also palpable. Chronic ulcers of the legs are a common occurrence in adult sufferers.

Hæmatological Investigations

The Fragility Test.—Increased fragility of erythrocytes characterises this disease. Normally, erythrocytes begin to hæmolyse in 0·47%. saline solution. In this condition hæmolysis occurs in 0·6%., or even in stronger solutions.

The Reticulocyte Count.—To compensate for the loss of erythrocytes by hæmolysis, the bone marrow discharges into the circulation immature red cells, which differ from the adult cells by possessing a reticulum. This cannot be seen in the usual blood films, but can be demonstrated readily by vital stains. Crises are associated with reticulocytopenia, and hypoplasia of the erythroid element of the bone marrow. After a crisis the reticulocyte count is greatly increased.

Fæcal urobilinogen is increased, as most of the urobilinogen is excreted by this route.

Use of Radioactive Chromium.[1]—Labelling of the patient's own red cells with ^{51}Cr will demonstrate the severity of red cell destruction and if this is accompanied by daily scanning over the spleen it will show the degree of red cell sequestration by the spleen. If splenic radioactivity is high, splenectomy will be of value.

Treatment.—All patients who have hereditary spherocytosis should be treated by splenectomy. Not, of course, that the spleen is at fault. The trouble lies with the red cells, but it is the spleen which will not let them survive sufficiently long to be of much service. After splenectomy, the spheroidal cells have a normal life span.

In the great majority of cases splenectomy can be undertaken as an elective operation during a remission. In juvenile cases the age at which operation is recommended has been decreasing. If it is not imperative before, the optimum time seems to be about 7 years, i.e. before gall-stones have had time to form, but subsequent vulnerability to infection is reduced.

At operation (splenectomy) the gall-bladder should be palpated for gall-stones, and if they are present cholecystectomy can be undertaken then or at a later date, depending on the patient's fitness.

Following splenectomy, the patient soon recovers from the anæmia, which does not return. The jaundice disappears, but the tendency to hæmolysis persists; it has been demonstrated twenty-five years after removal of the spleen. Ulcers of the leg due to this disease heal rapidly. In short (ruptured spleen excepted), in no other condition is splenectomy more triumphant.

[1] A solution of radioactive chromium salt is mixed with the patient's own blood and becomes attached to the red cells. When these are re-injected into the circulation the concentration of red cells in such organs as the spleen and liver can be counted by an external counter.

Owing to the absence of adhesions, the operation can be undertaken easily and expeditiously, which in part accounts for the very low operative mortality.

When a patient is so anæmic as to render blood transfusion advisable, more than ordinary precautions are required. Coombs' test must always be performed —in *hereditary* spherocytosis it is negative (cf. *acquired* hæmolytic anæmia). Correctly matched blood is usually well tolerated. In such circumstances whole blood should be avoided and a cautious transfusion of packed red cells substituted. As a rule packed red cells are accepted.

Acquired Hæmolytic Anæmia

Whereas hereditary spherocytosis is characterised by an abnormality inherent in the erythrocytes, the acquired form is held to be due to abnormal immunological responses in the plasma. Erythrocytes of the correct ABO group from normal donors are hæmolysed quickly by patients suffering from this type of hæmolytic anæmia. Thus the disease is acquired by the production of auto-antibodies that attach themselves to normal circulating erythrocytes and bring about their early dissolution. This phenomenon is detected by the Coombs' test which is usually, but not uniformly, positive.

Clinical Features.—An onset commencing in middle or late life is suggestive that the disease is acquired; the absence of a family history makes it almost certain. The spleen is palpable in 50 per cent. of cases, and in many of the patients with an enlarged spleen, the liver is palpable also. Sometimes there is a generalised enlargement of lymph nodes.

Laboratory Tests.—Anæmia is present. Spherocytosis may or may not be present. A positive Coombs' test helps to establish the diagnosis.

Treatment.—As in this form of the disease spontaneous remissions are not uncommon, a trial of corticosteroid therapy is warranted. If a sufficiently good response is obtained, splenectomy can be postponed in the hope that the remission will be sustained. An additional advantage of taking this course is that there is some evidence that severe transfusion reactions—always a concern in this disorder—are reduced in number and severity in those who receive steroid therapy (Coller).

Splenectomy may be of value in those cases where there is (1) a failure to respond to safe doses of steroids and (2) the ^{51}Cr studies show (*a*) an abnormally great sequestration of red cells in the spleen and (*b*) a high proportional count over the spleen as compared with the liver (high spleen/liver ratio).

PURPURA

Purpura is a condition of great importance to the surgeon. It forcibly intrudes into his diagnostic arena; on rare occasions it is the cause of an intussusception, but more frequently subserosal hæmorrhages produce signs similar to those of acute intestinal obstruction; it may give rise to profound hæmaturia or alarming hæmorrhage from a mucous lining of the body, e.g. epistaxis, hæmatemesis, melæna or, in the case of young females (in whom the condition is particularly common), menorrhagia so severe as to render the patient in danger of exsanguination. The condition frequently calls for blood transfusion, and certain cases are benefited by splenectomy.

FIG. 922.—Purpura. The patient presented with symptoms of acute intestinal obstruction due to submucosal hæmorrhage of the small intestine.

Robert Royston Amos Coombs, Contemporary. Quick Professor of Biology, Cambridge, England.
Frederick A. Coller, 1887–1964. Professor of Surgery, University of Michigan, Ann Arbor, Michigan, U.S.A.

The hæmorrhages themselves vary from tiny spots easily mistaken for a flea bite to the large bruised areas seen in very old, thin people with senile purpura, in whom there may be dramatically severe bruising after minor injuries. In some forms of purpura, like the allergic anomaly of Henoch-Schönlein purpura, the distribution may be prominently on the extensor surfaces (fig. 922).

Purpura is of two varieties :

1. **Primary**.—Idiopathic Thrombocytopenic purpura—acute or chronic.

2. **Secondary Purpura.**—(*a*) Thrombocytopenic—secondary to certain infections such as acute septicæmias, tuberculosis and brucellosis; to Boeck's sarcoid (p. 196); as an idiosyncrasy to certain drugs, particularly arsenicals, quinine, alkylating agents and gold salts; to X-ray therapy; to extensive burns; to aplastic anæmia, leukæmia or carcinomatosis of bone; and possibly to food allergy.

(*b*) Symptomatic—as in Senile purpura, Henoch-Schönlein purpura and Ehlers-Danlos disease of the skin.

IDIOPATHIC THROMBOCYTOPENIC[1] PURPURA

This is a relatively frequent hæmatological disorder that manifests itself by hæmorrhages of all grades of severity. At one end of the scale there is merely a tendency to bruise easily; at the other the disease is fulminating in its onset, and disastrous in its consequences. It occurs in an acute and a chronic form. The former is a disease chiefly of children; it is self-limiting and affects both sexes equally. The latter chiefly affects females.

Ætio-pathology.—The number of platelets falls to fewer than 60,000 per mm^3 (60×10^9/l) of blood.[2] There is increased capillary fragility, increased bleeding time, normal clotting and prothrombin time, and a non-retractile clot—a most significant finding.

Clinical Features.—Its various manifestations are as follows:

1. Ecchymoses (fig. 922) or purpuric patches in the skin and mucous membrane. There is a tendency for these lesions to appear in the more dependent areas, because of the higher intravascular pressure.

2. Sustained bleeding from wounds, which in themselves are often trivial.

3. Bleeding, frequently serious, from mucous membranes, epistaxis, bleeding from the gums and, in women during the period of reproductive life, menorrhagia, are of frequent occurrence. Urinary and gastro-intestinal hæmorrhages are less common.

4. Intracranial hæmorrhage occurs rarely, and not until other sites of hæmorrhage have given ample warning of the existence of thrombocytopenic purpura. Nevertheless, intracranial hæmorrhage is the most frequent cause of death in idiopathic thrombocytopenic purpura occurring in adults. This tragic complication can be averted by timely splenectomy.

5. Hæmarthrosis is rare (cf. Hæmophilia, p. 80).

[1] Thrombocytopenia = poverty of thrombocytes (blood-platelets).

[2] The normal blood-platelet count is 250,000 to 400,000 per mm^3 (250 to 400 $\times 10^9$/l).

Eduard Heinrich Henoch, 1820–1910. Physician, Berlin.
Johannes Lucas Schönlein, 1793–1864. Physician, Berlin.
Eduard Lauritz Ehlers, 1863–1937. German Dermatologist.
Henri Alexandre Danlos, 1844–1912. Dermatologist, Paris.

Cerebral Purpura.—Usually the onset is sudden, often with pyrexia. The leading features are thrombocytopenia with cutaneous hæmorrhages and neurological lesions attributable to involvement of cortical cerebral vessels. As a rule the patient lingers for a few weeks; in a few instances partial or complete recovery has occurred.

On Examination.—Except for cutaneous ecchymoses or a positive tourniquet test, physical examination is usually unrevealing. In only a quarter of the cases is the spleen palpable, and in these it extends but little below the costal margin. Pronounced splenic enlargement makes the diagnosis of thrombocytopenic purpura highly improbable.

Sternal Puncture.—The bone marrow shows an increase in the number of megakaryocytes[1] with absent platelet budding.

The behaviour of the disease in adults is dissimilar from that in children.

In Children.—After one acute episode consisting of cutaneous purpura, often accompanied by epistaxis and bleeding from the oral mucous membrane, the disease undergoes a spontaneous cure in 75% of cases.

In Adults.—Although the initial attack is seldom as severe as in children, the disease tends to become cyclic, and often the relapses are of increasing severity, uterine or gastro-intestinal hæmorrhages being much in evidence. This difference has an important bearing on treatment.

Treatment:

In Children.—As intracranial hæmorrhage is exceptional, and for reasons given above, a conservative policy can be followed and, if necessary, continued for three months. No active treatment is given if the symptoms are mild, but should the bleeding be severe or persistent, in addition to maintaining a safe hæmoglobin level, a course of corticosteroid therapy often effectively controls the hæmorrhagic manifestations. Such a course lasts two or three weeks. Splenectomy is advised in (1) severe cases; (2) when relapses occur; (3) especially in girls approaching the menarche. At least two days' steroid therapy should be given prior to operation. The results of splenectomy are usually satisfactory, in spite of the fact that after an initial rise, the low platelet count persists. If, after splenectomy, relapses occur, an overlooked accessory spleen may be present, but in some 25 per cent of cases no cause is found.

In Adults.—Steroid therapy, if necessary combined with blood transfusion, should be employed in preparation for splenectomy.

Blood transfusion is necessary when hæmorrhage causes severe anæmia. The volume of any one transfusion should not exceed 1 litre, because massive and rapid transfusions often aggravate the thrombocytopenia and the bleeding tendency. For the same reasons repeated transfusions are also contraindicated. Freshly taken blood is essential; any older than 6 hours is less useful for hæmostasis.

Steroid Therapy.—Unless the patient has already lost much blood, steroid therapy frequently controls the hæmorrhage and renders blood transfusion unnecessary. Its mode of action appears to be that of making the capillaries less permeable, it also re-

[1] Megakaryocytes, the giant cells of bone marrow, give origin to blood-platelets.

sults in a reduction of the time of clot retraction, but without a change in the platelet count.

Initially 1 to 2 mg kg of prednisolone is given by mouth in divided doses daily for two or three weeks then gradually reduced. In adults when bleeding is still troublesome after repeated courses for 2–3 months, splenectomy should be considered.

Splenectomy.—In slightly more than half the cases the bleeding tendency is controlled by the above measures, the operation can be converted from an emergency procedure to one of election which, being unhurried, permits a thorough search for accessory spleens.

In the remainder, operation must be undertaken with as little delay as possible. Following incision into and through the parieties there is profuse and sometimes frightening bleeding. This must be ignored until the splenic artery is reached and clamped. After waiting for twenty minutes or more, clotting will proceed more or less normally and the operation can be completed.

Splenectomy in primary thrombocytopenic purpura is followed by good results in over 60% of cases, in spite of little change in the platelet count after an initial rise lasting about a week.

Treatment of Secondary Purpura.—Obviously, if the cause is known and can be removed, considerable faith can be placed in conservative measures. Up to a few years ago it was thought that splenectomy was contraindicated in secondary purpura, but a number of cases of cure by splenectomy have been reported. Particularly encouraging have been cases where the purpura is due to splenic tuberculosis and in rare examples of Boeck's sarcoid. If the bleeding is so profuse or prolonged as to tax the resources of the blood bank, splenectomy should not be delayed (Lowdon). In most instances, however, it is safe to wait for a remission. Half of the cases are benefited by splenectomy.

MEDITERRANEAN ANÆMIA (*syn.* COOLEY'S ANÆMIA; THALASSÆMIA[1])

Cooley's anæmia is a hereditary disease due to faulty synthesis of hæmoglobin.

Originally thought to be confined to those who dwelt near the Mediterranean Sea, or who had a Mediterranean heritage, it is now known that this is only partially true, for scattered reports from various parts of the world show that the disease occurs from time to time in families who have no such heritage; a number of cases have been reported in natives of almost every country, including the U.K. The condition is inherited. In a severely affected infant both parents show characteristic blood changes. While the mild form is asymptomatic and can be detected only by an examination of blood, severe forms should be suspected by the following characteristics:

Clinical Features.—The patient is of short stature and the facies are reminiscent of a mongoloid; the skull is large and bossed, and the bridge of the nose is depressed (fig. 44, p. 48). In addition the maxillæ are prominent, also the upper front teeth. There is a mongoloid slant of the eyes. The complexion is muddy; the scleræ have an icteric tinge. Especially in a young child, the abdomen is prominent because of an enlarged spleen and liver. In patients with moderate anæmia these signs are correspondingly less, and in mild cases all are absent.

Blood Examination.—Increased resistance of red cells to hæmolysis in hypotonic saline solution is a characteristic finding. It is always present in Mediterranean anæmia, irrespective of the severity or mildness of the disease. In the more severe

[1] Thalassæmia = Greek, Thalassa — Sea. (Because it occurs in persons of Mediterranean stock.)

Andrew Gilchrist Ross Lowdon, 1911–1965. Professor of Surgery, Newcastle-upon-Tyne, England.
Thomas Benton Cooley, 1871–1945. Professor of Pædiatrics, Wayne University, Detroit, U.S.A.

forms, not only is there a diminished number of erythrocytes and a low hæmoglobin level, but nucleated red cells and other immature blood cells abound.

Radiography of the skeleton shows expansion of the marrow with typical ' hair-on-end ' appearance of the skull.

Treatment.—In selected cases splenectomy reduces the number of transfusions required.

PORPHYRIA, WITH SPECIAL REFERENCE TO THE ABDOMINAL CRISES OF THE DISEASE

Porphyria is an hereditary error of katabolism of hæmoglobin in which porphyrinuria occurs. The abdominal crises, which are characterised by violent intestinal colic with constipation, are liable to be precipitated by the administration of barbiturates, to which these patients have an idiosyncrasy. The patient is anæmic, frequently suffers from photosensitivity, and in advanced stages of the disease neurological or mental symptoms (from damage to the brain) are often present.[1] On examination the spleen will be found to be enlarged. On a number of occasions the splenic enlargement, which is usually well marked, has been overlooked and *the abdomen has been opened on the diagnosis of intestinal or appendicular colic*, with negative findings. Another manifestation of acute porphyria is spasmodic abdominal pain followed by jaundice. This may be due to spasm of Oddi's sphincter.

Two methods of establishing the diagnosis are available:

The urine is sometimes normal in colour. Usually it is orange (which is often dismissed as ' concentrated '). If the specimen is left exposed to daylight for a few hours it develops a port-wine colour, particularly near the surface, where it is exposed to the air. There are several conclusive laboratory tests for porphyrinuria.

Radiography of the abdomen. Serial X-ray films show areas of intestinal spasm causing short segments of gaseous dilatation of the small and large intestine, especially of the cæcum.

Treatment.—Often there is a striking decrease in the serum sodium level and the patient is improved considerably by infusion of normal saline solution with careful control of electrolytic balance. To relieve the abdominal pain methadone is the best drug. If a sedative is required, one of the phenothiazines (e.g. chlorpromazine) should be given. Splenectomy is not of value except in the uncommon erythropoietic type with splenomegaly.

SCHISTOSOMASIS

This condition, also known as endemic hepato-splenomegaly or Egyptian splenomegaly, is common among the fellaheen (peasants) who dwell in the delta of the Nile, but it is by no means confined to Egypt. It is prevalent in many parts of Africa, and is met with in Asia and South America, particularly Venezuela.

Ætiology.—In 75% of cases the disease is due to infestation by the *Schistosoma mansoni*, and the remainder by *Schistosoma hæmatobium*. In some parts of the world chronic malaria appears to be the cause of a similar condition, e.g. Bengal Splenomegaly (Basu), Tropical Splenomegaly.

Pathology.—A diffuse periportal fibrosis of the liver (fig. 923) is the first manifestation. Bilharzia ova in various stages of degeneration can be found in the portal vein and its tributaries. Next, enlargement of the spleen occurs, due, it is believed, (1) to bilharzial toxins and the products of disintegrated worms and ova, all of which are filtered by the spleen, causing active hyperæmia and hyperplasia of its reticulo-endothelial elements. (2) to the portal hypertension caused by the hepatic fibrosis. The splenic capsule becomes thickened, due to perisplenitis, and the splenic vessels are often tortuous and sclerotic. Diminished splenic contractility causes stagnation of blood in the spleen, and results in its further enlargement. About this time the

[1] King George III is believed to have suffered from this malady (a Royal malady).

Ruggery Oddi 1845–1906. Physiologist, Perugia, Italy.
Sir Patrick Manson, 1844–1922. Practised in Formosa and Hong Kong. Later Physician, Dreadnought Hospital, Greenwich, London. He is regarded as the ' Father of Tropical Medicine '.
Ajit Kumar Basu, Contemporary. Director-Professor, Department of Surgery, Institute of Post-Graduate Medical Education and Research, Calcutta, India.
Theodor Maximilian Bilharz, 1825–1862. Professor of Zoology, Cairo.

enlarged liver commences to contract, and the degree of enlargement of the spleen is roughly proportional to the degree of fibrosis of the liver (Halawani). Advanced cases are accompanied by ascites.

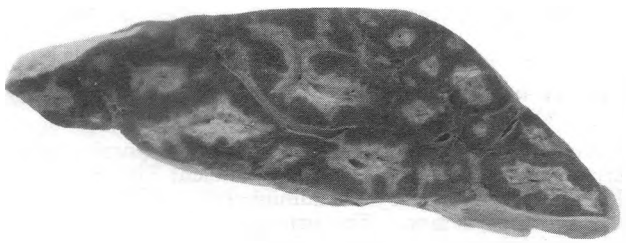


FIG. 923.—Periportal fibrosis of the liver in Egyptian splenomegaly. (*Dr. Halawani, Cairo.*)

Clinical Features.—Egyptian splenomegaly is encountered at all ages from ten to seventy years, frequently reaching its zenith in the fourth decade. Ninety per cent. of the patients are males.

1st Stage.—There is gradual enlargement of the spleen, hypochromic anæmia, eosinophilia, leucopenia and a relative lymphocytosis. The liver becomes moderately enlarged and is tender in 70% of cases. During this stage there is little or no disturbance of general health.

2nd Stage.—The spleen becomes further enlarged, extending to the pelvic brim (fig. 924). The liver gradually shrinks. The major symptoms at this time are:

Splenic pain or 'heaviness'	90%
Weakness	78%
Spasmodic abdominal pain	75%
Diarrhœa (often blood-stained)	60%
Hæmaturia	38%
Hæmatemesis	4%

The patient suffers from lassitude and may fall a victim to intercurrent infection.

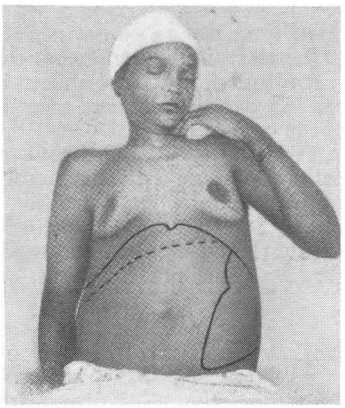


FIG. 924.—Egyptian splenomegaly. Splenectomy was successful. (*Owen Richards, F.R.C.S.*)

3rd Stage.—The liver atrophies more and more. Ascites supervenes. The superficial abdominal veins sometimes become dilated and tortuous. The general health deteriorates, and usually the patient dies in six to twelve months from the appearance of ascites. As a rule it takes eight to twelve years to pass from the first to the final stage.

Laboratory Findings

The urine and the fæces are examined for ova.

Liver function tests reveal a varying degree of hepatic impairment. A hypochromic anæmia is always present.

Treatment.—Many early cases respond to the general treatment of bilharzia mansoni detailed in p. 1103. In more established cases no substantial reduction in the size of the spleen results from a six-weeks' course of this treatment. It is in the latter class that, after careful pre-operative preparation, splenectomy with pre- and post-operative blood transfusions is under-

Ahmed Halawani, Contemporary. Director of the Institute of Tropical Medicine, Cairo.

taken under penicillin cover, and is often curative. The consensus of opinion in Egypt is that splenectomy should be performed in cases where there is no evidence of ascites, liver damage or severe renal insufficiency. Following splenectomy, over 70% of the patients are able to return to full work. When ascites has supervened, portacaval or splenorenal anastomosis (p. 903) is the only form of treatment that can slow the course of the disease.

FELTY'S SYNDROME

A moderate number of patients with chronic rheumatoid arthritis develop mild leucopenia; in a few of these, neutropenia becomes extreme and usually is associated with enlargement of the spleen. This combination is referred to as Felty's syndrome. A remarkable characteristic of this syndrome is that the leucopenia and splenic enlargement are apparently unrelated to the severity of the arthritic changes; indeed, in some instances the arthritis has commenced to improve or has become quiescent by the time the low white-cell count and the splenomegaly become unmistakable. In those cases in which the arthritis is slight but the splenic enlargement and the blood changes are much in evidence, a diagnosis of primary splenic neutropenia is sometimes made.

Clinical Features.—Sufferers from Felty's syndrome are very prone to pyogenic infections of all varieties, infected ulcers around the ankles being particularly frequent. They are fatigued easily, lean, and unable to gain weight. It is impossible to say whether these symptoms should be attributed to the arthritic process or to the leukopænia and splenomegaly. As a rule the degree of splenic enlargement is not great. Enlargement of lymph nodes and generalised brown pigmentation of the skin are sometimes present.

Blood Examination.—The white-cell count shows persistent leucopenia varying between 1,000 and 2,000 per mm^3 ($1{\cdot}0–2{\cdot}0 \times 10^9/l$) with less than 20% of neutrophils. The greatest reduction is in the granulocytes. Neutrophils range from slightly below normal to virtually absent. There is almost always an associated anæmia of moderate severity.

Sternal Puncture.—The bone marrow shows increased cellularity with, in some cases, a deficiency of mature granulocytes.

Treatment.—The results of splenectomy are variable. Usually there is an improvement in the blood picture with increased neutrophils, but this improvement is not maintained. However, the liability to infections seems to be decreased in many cases and rheumatoid arthritis that had become resistant to steroid therapy may react favourably to steroid therapy once more.

OCCLUSION OF THE SPLENIC VEIN

The splenic vein is most often found to be obliterated in young children, probably following umbilical sepsis in the neonatal period and here the portal vein may also be affected giving rise to portal hypertension. In such cases splenectomy with lieno-renal or lieno-caval anastomosis may be indicated. Where portal hypertension does not exist splenectomy will suffice. In adults the vein may become blocked by local infection or by neoplastic infiltration as in carcinoma of the body or tail of the pancreas. Here, again, splenectomy may be worth while if the neoplasm is deemed to be removable.

GAUCHER'S DISEASE

As mentioned on p. 860, the spleen may take an active part in the storage of abnormal lipoids, as does the remainder of the reticulo-endothelial system. In the case of Gaucher's disease the lipoid in question is glucocerebroside. Gaucher's disease, which is rare, is characterised by enormous enlargement of the spleen, which may weigh 8 or 9 lb (3·6 or 4·1 kg). In the majority of cases the splenic enlargement begins in early childhood, often before the age of twelve, although the patient rarely seeks advice before adult life. Until the splenic enlargement becomes massive the symptoms are few. There is anæmia, a yellowish-brown discoloration of the skin of the hands and face, and a curious conjunctival thickening (pinguecula) that helps to clinch

Augustus Roi Felty, 1895–1964. Physician, Hartford Hospital, Hartford, Connecticut, U.S.A.
Philippe Charles Ernest Gaucher, 1854–1918. Physician, Hospital St. Louis, Paris.

the clinical diagnosis. Slavonic and Jewish races appear to be more prone to this disease than the rest of humanity. The diagnosis is confirmed by finding Gaucher's cells in the bone marrow (fig. 925).

Treatment.—Splenectomy rids the patient of a large abdominal swelling, but the operation is difficult because of perisplenitis and friability of the splenic pulp. It does not greatly influence the course of the disease but because it reduces the hypersplenism (anæmia, leucopenia and thrombopenia) and makes the patient more comfortable it is worth doing.

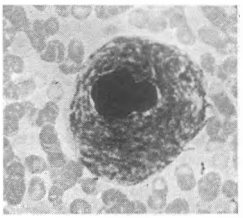


FIG. 925. — Typical Gaucher's cell from bone marrow obtained by sternal puncture. (*Dr. M. L. Thomson, Manchester.*)

LEUKÆMIA

This is of surgical importance in the differential diagnosis of enlarged lymph nodes and splenic enlargement. The diagnosis is made by an examination of the blood. The main treatment (and this is palliative) is deep X-ray therapy and chemotherapy. In chronic granulocytic leukæmia both the liver and the spleen are greatly enlarged. Splenectomy has been tried without proven success as yet. In chronic lymphocytic leukæmia splenectomy is occasionally indicated.

TUBERCULOSIS OF THE SPLEEN

Tuberculosis of the spleen is not so uncommon as is sometimes believed. It occurs chiefly in adults between twenty and forty years of age. When a patient has splenomegaly with asthenia, loss of weight and an evening temperature, it is well to bear in mind the possibility of the enlargement of the spleen being due to tuberculosis. Too often these signs lead to the erroneous diagnosis of leukæmia or some other disorder for which splenectomy is not indicated. Occasionally tuberculosis of the spleen produces portal hypertension. Another form is cold abscess, which is very rare. Splenic puncture, followed by culture, or guinea-pig inoculation, will yield positive results. A therapeutic test with P.A.S. and streptomycin usually brings about some improvement, and there is less danger of dissemination of tubercle bacilli if splenectomy is undertaken. The operation, which usually is rendered difficult because of adhesions, is contraindicated only if other active tuberculous lesions are present. Otherwise the results of splenectomy in tuberculosis of the spleen are excellent (Patel).

ABSCESS OF THE SPLEEN

If a splenic embolus is infected, and the primary condition does not prove fatal, a splenic abscess may be expected to follow. Other sources of metastatic abscesses of the spleen are typhoid and paratyphoid fever, osteomyelitis, otitis media and puerperal sepsis. An abscess in the upper pole of the spleen may rupture and form a left subdiaphragmatic abscess. If the abscess is in the lower pole, rupture results in diffuse peritonitis.

Treatment.—As a rule, owing to dense adhesions, drainage of the abscess is the only course. Very rarely, splenectomy may be possible with the abscess *in situ*.

NEOPLASMS

New-growths of the spleen are strikingly uncommon. Of the benign tumours, cavernous hæmangiomas and lymphangiomas have been encountered. Of the malignant, a few examples of primary fibrosarcoma have been described. Lymphosarcoma is somewhat more common. Metastases of carcinoma in the spleen are rare. The primary tumour is usually of lung or breast or melanoma. The commonest causes of neoplastic enlargement of the spleen are the malignant lymphomas of which about half the cases are Hodgkin's disease. Splenectomy often plays an important role in the clinical staging and management of these conditions (p. 194).

HYPERSPLENISM

Various conditions involving the spleen, e.g. portal hypertension, may on occasion cause anæmia, neutropenia, thrombocytopænia or any combination of these. This is probably due to destruction of the relevant cells in the spleen and, if symptoms warrant it, splenectomy is indicated.

Jean Patel, Contemporary. Professor of Clinical Surgery, Paris, France.

SPLENECTOMY

The common indications for splenectomy in this country are trauma, either following an accident or at operation (e.g. gastrectomy), thrombocytopenic purpura, hereditary spherocytosis, or preparatory to spleno-renal anastomosis.

Other indications for splenectomy are tabulated on pp. 865 and 866.

Technique of Splenectomy.—Most surgeons use a left paramedian or left subcostal incision. For large spleens adherent to the diaphragm a thoraco-abdominal incision may be necessary. Before operation, however, the passage of a naso-gastric tube enables the stomach to be emptied. This facilitates the operation and prevents the post-operative complications of dilatation of the stomach and paralytic ileus.

If the operation is for *traumatic rupture*, a quick mobilisation is necessary. The hand is passed round the outer surface of the spleen, the posterior layer of lieno-renal ligament (fig. 926) divided largely by blunt dissection, and the spleen rotated medially

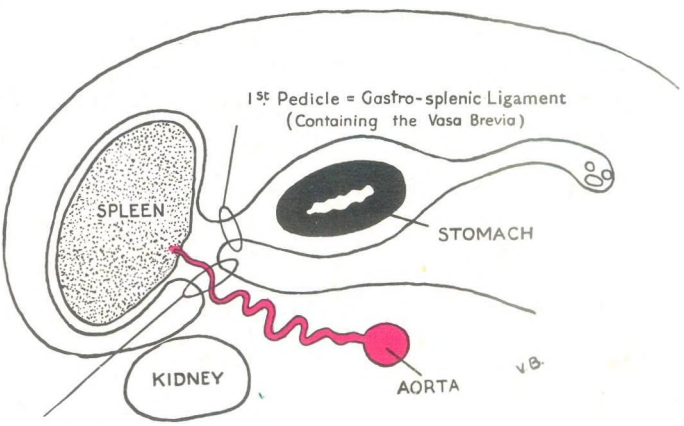


FIG. 926. — From the surgical standpoint the spleen may be said to have two pedicles—the gastro-splenic ligament and the lieno-renal ligament. The splenic artery and vein lie in the latter.

into the incision. A large pack is inserted and the short gastric vessels and those in the pedicle are ligated and divided. It is important to separate the tail of the pancreas from the vessels in the hilum before ligation.

For *hypersplenism and other conditions* requiring splenectomy.—The first step is to open the gastro-splenic ligament and divide the short gastric vessels (fig. 926). The splenic vessels at the superior border of the pancreas are under-run with silk and ligated. The posterior surface of the spleen is exposed, the posterior leaf of the lieno-renal ligament divided by long curved scissors, and the spleen rotated medially, together with the tail and body of the pancreas. The pancreas is separated from the hilum and the vessels dissected out, ligated, and divided. Careful search must be made for accessory spleens. It is wise to drain the wound in case damage to the pancreatic tail has occurred.

Unexplained abdominal pain and fever in the post-operative period may herald thrombosis of the portal vein, when anticoagulants and antibiotics must be given.

CHAPTER 45

THE LIVER

Surgical Anatomy and Physiology.—The dual afferent blood supply, consisting of the hepatic artery and the portal vein, singles out the liver from all other organs. That the portal vein has no valves is basic to the understanding of portal hypertension. Both the portal and the hepatic veins have well-defined coats of unstriated muscle which together with the hepatic artery control the blood flow through the liver.

The blood in the sinusoids is derived from two sources:

1. The portal system conveys between 60 and 80 per cent. of the afferent blood to the liver, together with the products of digestion from the alimentary canal and the internal secretions, from the pancreas and small intestine. No less than one-fifth of the portal blood comes from the spleen.

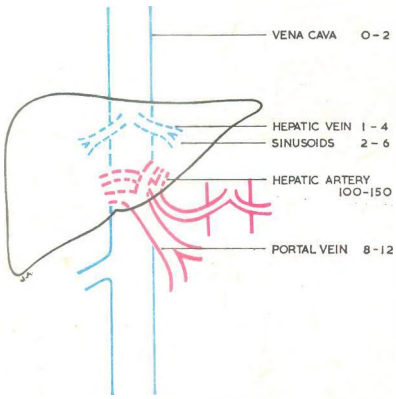


FIG. 927.—Pressure (in mmHg) in various components of the hepatic circulation.

2. The hepatic artery transmits oxygen to the liver and, by a delicate pressure arrangement with the portal venules determines the blood flow through the liver lobules. The pressure relationships of the hepatic artery, the portal vein and the hepatic vein testify further as to the unique nature of the circulation through the liver (fig. 927). During exercise the blood flow through the liver, normally about 40 per cent. of the cardiac output, falls sharply in favour of the heart, brain and muscles.

Surgical Lobes of the Liver:

The right and left branches of the hepatic artery do not supply the two main anatomical lobes as such. Their territory has a boundary running along the gall-bladder bed and thence upwards over the anterior surface to the inferior vena cava, down this vessel to return to the porta hepatis. The biliary ducts and branches of the portal vein follow the hepatic arterial branches. In this way the liver is divided into two surgical lobes, right and left, by a major avascular fissure. The blood from the liver is drained by three major hepatic veins, the middle one of which is located in the major fissure. Knowledge of the anatomy of the surgical lobes is particularly important in planning hepatic resections for injury or tumours.

Functions of the Liver:

1. The secretion of bile.
2. The formation of albumin, fibrinogen, and prothrombin.
3. Storage and metabolism of carbohydrates, including the conversion of monosaccharides (e.g. dextrose) into glycogen, and vice versa.
4. Formation of phospholipids and cholesterol, synthesis of fatty acids from carbohydrate, and other steps in fat metabolism.
5. Deamination of amino-acids with formation of urea. Removal of ammonia from portal blood.
6. Heat production.
7. Reticulo-endothelial activities.
8. Storage of vitamin B12 (cyanocobalamin) and vitamin A.
9. Iron and copper storage.
10. Destruction of bacteria. Bacteria, especially Gram-positive cocci, have been

Hans Christian Joachim Gram, 1853–1938. Professor of Medicine, Copenhagen.

found repeatedly in samples of portal blood taken at laparotomy for non-infected conditions. It is therefore believed that the liver destroys many bacteria that gain entrance to the body through the portal tributaries.

11. Detoxication of drugs and hormones, e.g. short-acting barbiturates; œstrogens.

Unlike other organs, the liver possesses a remarkable ability to replace lost tissue rapidly and completely by compensatory cellular hypertrophy and hyperplasia. This permits wide resection for a localised hepatic neoplasm, and removal of large portions of doubtfully viable liver tissue in cases of severe trauma.

HEPATOCELLULAR FAILURE

Hepatocellular failure can complicate almost all forms of liver disease and may be precipitated by intercurrent infection, a surgical operation, administration of a general anæsthetic, bleeding or even narcotics.

Clinical Features.—The most marked features are weakness and general failure in health. Jaundice is usually present. The hyperdynamic circulation is manifested by flushed extremities and bounding pulse. The patient may be cyanosed. Fever is common and septicæmia is found terminally. Fœtor hepaticus, a sweetish, musty odour, is frequently noticeable. Spider angiomas are found in the vascular territory of the superior vena cava. Appearance of fresh crops of spiders suggest progressive liver disease and their disappearance accompanies improving hepatic function. Palmar erythema (liver palms) affects mainly the thenar and hypothenar eminencies.

Other changes include loss of axillary and pubic hair, and white nails. In the male, gynæcomastia and testicular atrophy may be found in chronic hepatic failure of the cirrhotic.

In acute hepatocellular failure, personality changes occur early proceeding to delirium, nightmares and unco-operative behaviour.

Treatment.—Precipitating factors must be looked for and treated. Gastrointestinal hæmorrhage may require blood transfusion; an acute infection may be dealt with by antibiotics. Electrolyte abnormalities, especially hypokalæmia, should be corrected.

If the patient can eat, a high carbohydrate diet should be given. Nutrition and fluid intake have to be maintained in the unconscious patient by the use of a nasogastric tube or intravenous infusion. Diuretics, morphine and paraldehyde should be avoided because they may precipitate or potentiate coma.

In acute failure with coma, very skilled and intensive treatment is required. Hypoglycæmia and electrolyte abnormalities will require particular attention. For renal failure with hyperkalæmia, peritoneal- or hæmodialysis should be performed. Bleeding due to abnormalities in clotting mechanisms and infection will require skilled hæmatological and bacteriological treatment.

For patients whose condition is not responding, but who are thought to have potentially reversible failure, temporary hepatic support should be considered. These measures include exchange blood transfusion, cross circulation between donor and patient, extracorporeal perfusion of patient's blood through a pig's liver and, recently, perfusion of patient's blood through columns of activated charcoal. The results of these various treatments are uncontrolled and their long-term benefit remains doubtful.

PORTA-SYSTEMIC ENCEPHALOPATHY

This syndrome occurs when much blood is bypassing the liver (either because of cirrhosis or via a surgically-created porta-systemic shunt) and when there is a degree of hepatic insufficiency (p. 902). Toxic products of bacterial degradation of nitrogenous material within the bowel are normally absorbed and are removed by the liver. In chronic liver disease—and also because of direct access to the systemic circulation via collateral vessels, surgical shunts or intra-hepatic communications—these toxic products may reach the brain where they exert a deleterious effect. Ammonia is probably one of the toxic products responsible for hepatic encephalopathy but methionine and short-chain fatty acids have also been incriminated. These products are normally metabolised in the liver, and the syndrome rarely occurs unless the liver function is impaired. It thus tends to be precipitated by a large protein meal or an upper gastro-intestinal hæmorrhage. Other precipitating factors include potassium depletion, frequently accompanying drug-induced diuresis, uræmia and narcotics. In individual patients it is often difficult to determine to what extent the symptoms are due to this cause or to liver-cell damage. In patients who have had symptoms for some years, organic changes have been found in the cerebral cortex or in the spinal cord.

Clinical Features.—It is characterised by disorientation and a flapping tremor of the outstretched hands. Cogwheel rigidity of the limbs may be elicited, and the patient may pass rapidly into deep coma. Intellectual deterioration and changes in personality, slurring of speech, can be observed frequently. On the electroencephalogram there is marked slowing of the frequency down to the delta range. Sometimes he may recover equally rapidly when the source of the toxic products is removed from the alimentary canal.

Treatment.—Measures to reduce the amount of nitrogenous material in the bowel, its breakdown by bacteria and its absorbtion are effective, viz:

1. Restriction of dietary protein.
2. Removal of blood from bowel by purgatives and enemas.
3. Administration of antibiotics orally or rectally, especially non-absorbed antibiotics such as neomycin.
4. Oral lactulose (a disaccharide), whose mode of action is still unclear.

Other measures include the avoidance of sedatives and the correction of electrolyte deficiencies.

SPECIAL METHODS OF INVESTIGATING THE LIVER

A. Liver Function Tests.—Owing to its manifold functions, there is no single test by which the liver can be stated to be functioning normally. Therefore several tests are usually undertaken in each patient, and in some instances the individual test must be repeated. No less than 80 per cent. of the liver can be out of action without affecting individual tests, and in patients with cirrhosis they may all be within normal limits. Among a large number of tests available the following are the most useful:

1. **Serum Bilirubin Estimation.**—A value of conjugated bilirubin exceeding 7 μmol/l (0·4 mg/100 ml) is one of the most valuable indices of either stasis within the biliary tree or hepato-cellular damage, but it is of no value in distinguishing the one from the other.

2. **Serum alkaline phosphatase** (normal value 3 to 13 King–Armstrong (K–A) or 1·5–4 Bodansky units). Values below 30 K–A favour hepatogenous jaundice; those above 30, obstructive jaundice.

3. **Estimation of serum albumin** is a good general test of liver function, as it is the only site of production. A level below 20 g/l (2·0 g/100 ml) indicates that liver function is greatly impaired and, if an operation is essential, only the minimum should be done. Above 30 g/l (3·0 g/100 ml) is satisfactory.

4. **Serum Transaminases (aminotransferases).**—These are enzymes present in liver cells and heart muscle. Increased values (over 100 units) are found in hepatocellular disease but only in prolonged obstructive jaundice when liver damage occurs.

5. **Plasma prothrombin index,** if *low*, is an indication for pre-operative vitamin K therapy. Where there is little or no response to vitamin K, extensive hepatocellular damage is almost certain. Owing to large reserves a satisfactory response does not exclude considerable liver damage.

6. **Bromsulphalein Test.**—The dye bromsulphalein (B.S.P.) is rapidly removed by the liver and excreted in the bile. In the absence of jaundice, it is a sensitive test of derangement of hepatic function. After an intravenous injection of 5 mg/kg of the dye, not more than 5% of it should be present in the serum at the end of 45 minutes.

7. **5—nucleotidase.**—This enzyme originates in the same part of the hepatic parenchymal cells as alkaline phosphatase but its level in the blood is not affected by osteoblastic activity. It is therefore of value in determining the origin of a rise in alkaline phosphatase, which may be influenced by bony and intestinal, as well as hepatic, activity. The normal level is 8·5–9·0 international units per litre.

8. **Alphafetoprotein.**—Very high levels of this abnormal protein are almost diagnostic of hepatoma.

B. Liver biopsy is a common procedure of considerable diagnostic value. It should never be attempted until the presence or absence of a bleeding tendency on the part of the patient has been ascertained by all accepted tests, and if present, remedied. The puncture is carried out on the right side or in the epigastrium. The Menghini needle (fig. 928) is the best. Sections are cut from the cylindrical specimen obtained. Hæmorrhage or biliary peritonitis are complications. Liver biopsy is of particular value in generalised liver disease, e.g. cirrhosis, reticulosis and hepatitis. Local disease such as tumours, focal granulomas, etc., may be missed. A combination of peritoneoscopy with needle liver biopsy enables a tissue sample to be taken directly from a focal abnormality.

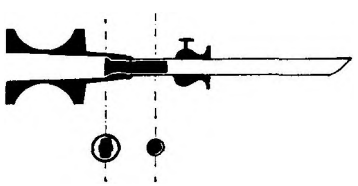


FIG. 928.—Menghini 'one-second' liver biopsy needle. The nail in the cannula allows fluid to pass but prevents the biopsy specimen from being fragmented by violent aspiration into the syringe. (*From* Gastroenterology.)

C. Imaging techniques.

1. **Non-invasive methods.**—(*a*) *A straight X-ray* of the abdomen is only of limited diagnostic value.

(*b*) *Ultrasound.*—This is a safe, simple and cheap method which is of particular value in demonstrating dilatation of bile ducts and stones in the gall-bladder (p. 911).

(*c*) *Computerised axial tomography.*—Although a promising technique, its primary disadvantage is its expense and limited availability.

(*d*) *Radio-active scanning.*—A gamma emitting isotope such as ^{198}colloidal gold and ^{69}Technetium^{m} is taken up by the reticulo-endothelial system and can be useful in localising lesions in the liver. Rose-bengal labelled with ^{131}I is taken up by the liver parenchyma but unfortunately its concentration in the gallbladder interferes with delineation of the liver edge.

The normal liver presents an even distribution of activity and lesions which do not take up isotopes, e.g. abscesses, cysts and tumours, appear as defects in the scan. Also

Earl Judson King, 1901–1962. Professor of Chemical Pathology, University of London (Royal Post-Graduate Medical School).
Arthur Riley Armstrong, Contemporary. Director of Department of Laboratories, Hamilton Health Association, Hamilton, Ontario, Canada.
Aaron Bodansky, 1887–1961. Biochemist, Hospital for Joint Diseases, New York City, U.S.A.
Georgio Menghini, Contemporary. Physician, Perugia, Italy.

extrinsic lesions such as subphrenic abscesses can also be detected. Lesions greater than 2 cm can be detected readily, but false positive scans are a problem.

Generalised decrease in uptake suggests a diffuse hepatocellular disorder such as cirrhosis.

2. **Invasive methods.**—(*a*) Percutaneous transhepatic cholangiography (p. 933).

(*b*) **Angiography** by means of cœliac axis or superior mesenteric arteriograms is occasionally valuable in detecting changes in the vascularity of the liver, either due to vascular obstruction or to an avascular lesion such as a cyst or abscess, or to a vascular tumour. In cases of portal hypertension films in the venous phase of superior mesenteric angiography are useful if the spleen has been removed and splenic injection is thus impossible.

(*c*) Endoscopic retrograde cholangiopancreatography (ERCP), (see p. 933).

INJURIES

Rupture of the liver, is an extremely grave accident, and in a considerable proportion of cases it is associated with other serious injuries. Consequently the mortality is high. The violence that produces this injury is usually of a crushing type or from the steering wheel of a car in collision. Rupture of the right lobe is much more common than of the left, because it is larger and less mobile; usually the tear is on the anterior or the superior surface of the organ. Often a rib, or ribs, on the right side are fractured.

Most of the problems of injury of the liver are caused by hæmorrhage, leakage of bile and devitalisation of liver tissue. Hæmorrhage may be free into the peritoneal cavity and the patient presents with the clinical picture of massive intra-abdominal hæmorrhage, perhaps indistinguishable from rupture of the spleen but suggested by localising signs of pain, tenderness and rigidity in the right upper quadrant of the abdomen. Bleeding within the liver may form a large hæmatoma, which may later become infected to form an abscess, or it can rupture into the bile duct giving rise to hæmobilia. Biliary leakage can lead to biliary peritonitis with subsequent abscess or cyst formation. Infarction of liver tissue due to occlusion of blood supply can be complicated by an abscess within the liver or immediately above or below that organ.

Treatment.—In the first three types a blood transfusion is of cardinal importance, and in all cases laparotomy must be undertaken.

Operation.—The abdomen is opened either through a right paramedian incision, with extension into the right thorax if required for major hepatic lobectomy, or through a midline incision which can be extended into the chest as a median sternotomy. Bleeding vessels in the wall and depths of fissures and tears should be ligated, but it is important that all pulped liver and portions of doubtful viability should be removed. Failure to debride non-viable liver tissue favours infection and secondary hæmorrhage.

Simple tears may be opposed by inserting sutures parallel to the wound, and then coapting stitches are placed so that they under-run the mattress sutures (fig. 929).

Packing should be avoided except as a temporary measure to allow the patient to be transported to a centre where resection can be performed. With extensive hæmatoma formation and rupture of liver tissue, a major hepatic resection may be required, frequently a right hepatic lobectomy, i.e. removal of the right anatomical lobe and medial segment of left lobe, along with quadrate and caudate lobes. Following such major operations the abdominal cavity must be drained because leakage of bile occurs from torn ducts. It is still a matter of discussion whether the bile ducts should be drained after resection. Following operations such as hepatic resections, complications such as abscess formation, hæmobilia, hypoalbuminæmia and coagulation defects may occur. The mortality for hepatic injury is about 10%.

Penetrating Wounds of the liver occur as a result of gunshot injuries (p. 13) and stab wounds. When, as is commonly the case, the wound of entrance is in the right lower thorax, the thoraco-abdominal approach, excising the wound of entrance, is advisable. In some cases the wound of the liver is comparatively small, and no serious bleeding occurs; but in all cases, laparotomy must be performed to exclude damage to other organs. If the wound is a large one, the principles of arresting hæmorrhage given above are employed.

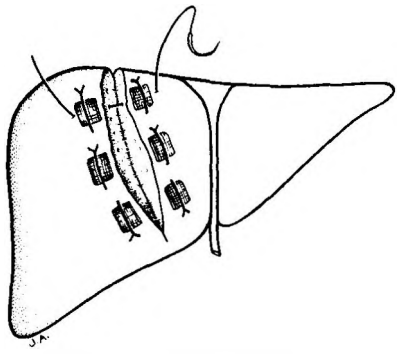


FIG. 929. — Repair of liver tear. Mattress sutures parallel to the tear are then under-run with coapting stitches.

ENLARGEMENTS OF THE LIVER

- *General enlargement*
  - With jaundice
    - Regular
      - Virus hepatitis.
      - Cholangitis.[1]
      - Pylephlebitis.[1]
      - Carcinoma of head of pancreas.
      - Carcinoma of the common bile duct.
    - Irregular
      - Hepatic cirrhosis.
      - Late secondary tumours.
  - Without jaundice
    - Regular
      - Hepatic cirrhosis.
      - Budd–Chiari's syndrome (p. 893).
      - Failing heart.
      - Leukæmia.
      - Rickets.
      - Amyloid.
    - Irregular
      - Secondary tumours.
      - Gummas.
- *Localised swelling*
  - Riedel's lobe.
  - Hydatid cyst.
  - Amœbic abscess.[1]
  - Hepatoma.
- *Massive irregular enlargement*
  - Polycystic disease.
  - Cholangio-hepato-adenoma.
  - Secondary tumours.

VIRUS HEPATITIS

Two types of disease are described. ' Virus A ' hepatitis is used for short-incubation disease, formerly called ' Infective Hepatitis '; ' Virus B ' hepatitis is used for long-incubation disease, hitherto called ' Serum Hepatitis '. Our knowledge of the latter type of disease has been advanced by the discovery of hepatitis B (Australia) antigen.[2]

Virus A hepatitis occurs in sporadic and epidemic forms. The virus (type A) is excreted in the fæces and this is probably the method of spread. It is the commonest form of jaundice in children and young adults, and there is no evidence that one attack confers immunity. The incubation period is from three to five weeks.

The condition commences with anorexia, nausea and perhaps vomiting, with malaise and fever, and jaundice appears after about three days. The liver may be palpable and tender, and occasionally there is transient ascites. The fever subsides shortly after

[1] Intermittent pyrexia, sometimes rigors, occur.

[2] So-called, because it was discovered in an Australian Aboriginal.

Bernhard Riedel, 1846–1916. Professor of Surgery, Jeno, Germany.

the jaundice appears. Usually the jaundice disappears after two or three weeks, but it may persist very much longer. In such cases, the diagnosis from extrahepatic biliary obstruction may be difficult, and liver function tests may not give a clear distinction.

Sometimes infective hepatitis is followed months or years later by portal cirrhosis, but the factors which cause this are unknown, as most patients recover completely. However, a few develop acute or subacute hepatic necrosis which accounts for the mortality of 3%.

Immune serum globulin is effective in preventing or modifying Type A virus hepatitis and is usually given to household contacts who have not already had the disease. Antibiotics are of no value. Steroids may be given to those with prolonged cholestasis to lower the serum bilirubin. It is regarded as important that the patient should avoid alcohol or strenuous exercise for some months after all symptoms and signs have subsided. A small proportion of patients have recurrent attacks of mild symptoms persisting for a year or more.

Virus B hepatitis.—This may follow blood transfusion, plasma infusion, and, rarely, the administration of sera, and resembles infective hepatitis except that the incubation period is about twelve weeks. Transmission by plasma has been reduced by avoiding the pooling of plasma from a large number of donors. Transmission by syringes is prevented if all syringes are disposable. It occurs amongst those who are drug addicts and possibly after tattooing, ear piearcing, or sexual activities.

Hepatitis B antigen (HBAg) is found in 80% of sera of those who develop Virus B hepatitis. Under electron microscopy, particles can be seen in the sera of these patients and are associated with the virus. These particles are antigenic. Such patients usually suffer the severest form of hepatitis. The antigen is the scourge of renal dialysis and transplantation units, and hospital staff must avoid contact with blood from such patients.

CHOLANGITIS (*syn.* INTERMITTENT HEPATIC FEVER OF CHARCOT)

By cholangitis is meant a state of inflammation of the bile ducts, but it is the radicles of the biliary tree within the liver that are the main site of the infection. Cholangitis can occur as the result of obstruction of the common bile duct by a gall-stone (p. 928) or a stricture, or in mucoviscidosis (p. 939). Suppurative cholangitis (p. 929), like pylephlebitis, may be followed by the formation of multiple liver abscesses. There is often septicæmia with a positive blood culture for *E. coli* and other intestinal organisms. Gram-negative shock may develop.

PYLEPHLEBITIS (*syn.* PORTAL PYÆMIA)

Pathology.—Pylephlebitis can follow a suppurative disease in any part drained by the portal system, but usually arises as a complication of appendicitis or diverticulitis. The process commences as a thrombophlebitis of a small vein draining the infected lesion. The thrombus spreads to a larger collecting vessel, and pieces of infected thrombus break off and are swept into the liver, where they lodge and form abscesses. Abscesses so formed are usually small and multiple, in which case they are connected together, forming a 'canal and cavity' system (Turnbull). Cultures show a mixed infection in which *Esch. coli* is prominently represented. In rare instances there is localisation of the infection.

Clinical Features.—Pylephlebitis is characterised by a hectic temperature and the occurrence of repeated rigors. In relevant cases the rigors serve to differentiate the condition from a sub-diaphragmatic abscess, but do not entirely exclude it. If tested for, urobilinogen will be found in the urine in every case. Diarrhœa is a frequent accompaniment. Moderate

Jean-Martin Charcot, 1825–1893. Physician La Salpêtrière, Paris.
Hubert Maitland Turnbull, 1875–1955. Professor of Pathology, The London Hospital.

ascites is sometimes present. The patient soon becomes slightly jaundiced, the liver is somewhat enlarged and tender and the serum bilirubin is raised.

Blood culture is positive in 50% of cases if blood is withdrawn during or immediately after a rigor. Sensitivity tests must be done on the organism so that the correct antibiotic can be given.

Prophylaxis.—The most important means of preventing pylephlebitis is to administer a broad spectrum antibiotic in cases of fulminating appendicitis or diverticulitis.

Treatment.—As the organisms responsible are mixed, initial chemotherapy should include broad-spectrum antibiotics such as amoxycillin and gentamicin administered parenterally. If bacteroides are identified, metronidazole should be given. Cloxacillin should be administered if staphylococci are identified. Blood transfusion is often required. If, in a protracted case, it is considered that there is a sizeable abscess within the liver, laparotomy should be performed with a view to draining the abscess.

Prognosis.—When pylephlebitis is fully established the outlook was formerly almost hopeless, but with the use of antibiotics many cases recover. Extrahepatic portal venous obstruction is likely to develop later.

IDIOPATHIC PYOGENIC LIVER ABSCESS

Acute abscesses are usually multiple and may be associated with biliary or abdominal disease, but often, especially in elderly patients suffering from malnutrition, no cause can be found. The clinical features and treatment are similar to those described for pylephlebitis (see above).

Chronic abscesses usually are single and occur mainly in elderly females. The onset is often insidious, or even silent (cryptogenic abscess). A cause for the infection may be found in the biliary passage or intestinal tract, but in half the cases no source can be discovered. The patient presents with pyrexia, abdominal discomfort, an enlarged liver, and, in advanced cases, hepatic coma. Leucocytosis is absent, but the serum albumin level rapidly falls, while the level of Vitamin B12 rises (Butler). Hepatic scintiscanning and ultrasonic scanning have proved most helpful not only in permitting earlier diagnosis of fluid-filled hepatic masses but also in aiding the localisation of the abscess before exploration and drainage.

The condition is fatal unless, after excluding an amœbic abscess, the abscess is drained, when the mortality is reduced to 30%. Laparotomy is performed, and the abscess is discovered by aspiration. Dependent drainage is effected, after loculi have been broken down by digital manipulation. Examination of the pus will indicate a suitable antibiotic.

In most instances the cause of a liver abscess is apparent, or can be ascertained, infection reaching the liver in one of several ways, viz:

CAUSES OF ABSCESSES OF THE LIVER

Via the Portal Vein

- Suppurative appendicitis.
- Diverticulitis.
- Amœbiasis.
- Infected carcinoma of the colon (especially after resection).
- Ulcerative colitis (rare).
- Actinomycosis of the right iliac fossa.
- Inflamed hæmorrhoids (rare).
- Typhoid and paratyphoid (rare).

Thomas Joseph Butler, 1916–1972. Surgeon, Frenchay Hospital, Bristol, England.

Along the Bile Ducts	Stone impacted in common bile duct. Stricture of common bile duct.
Via the Hepatic Artery	Septicæmia and pyæmia. Facio-cervical actinomycosis. Infection of a hydatid cyst.
From the Umbilicus	Along the umbilical vein of the new-born. Along the para-umbilical veins.
By Direct Extension	From a subdiaphragmatic abscess. From an empyema thoracis (rare). From a penetrating wound.

AMOEBIC LIVER ABSCESS (*syn.* TROPICAL ABSCESS, DYSENTERIC ABSCESS)

Amœbic abscess of the liver is one of the terminations of amœbic hepatitis, which in turn is a complication of amœbic dysentery—not so very uncommon.

Pathology.—*Entamœbæ histolyticæ* (fig. 930) pass from a focal lesion in the colonic wall into a radicle of the inferior mesenteric vein and via the portal vein they enter the liver to take up residence there, usually in the upper and posterior portion of the right lobe. In the liver the entamœbæ colonise and live at the expense of the liver cells, causing localised liquefaction necrosis. The amount of liver destruction is proportional to the size of the colony, the resistance of the host and the extent of secondary infection. In 70% of cases, the abscess is solitary; in 30% more than one abscess is present. Characteristic pus from an amœbic liver abscess is chocolate-coloured, or like anchovy sauce, and consists of broken-down liver cells, leucocytes, and red blood cells; nevertheless, in an appreciable number of instances the pus is green, from being admixed with bile. In about half the cases the pus contains staphylococci, streptococci, and *Esch. coli*, as well as *E. histolytica*. In the remainder the pus is sterile, but except in long-standing cases, motile entamœbæ can be demonstrated in the last few drops of pus to be withdrawn, or from a scraping of the abscess wall at operation. Commonly perihepatitis causes the liver to become fixed to the diaphragm or the abdominal wall.

FIG. 930.—Entamœba histolytica.

Course.—An amœbic abscess of the liver runs a variable course:

1. In early stages of amœbic hepatitis with abscess threatening, resolution may occur under emetine or metronidazole treatment, but some authorities doubt whether there is really a stage of hepatitis without abscess formation; it is more probable that treatment results in cure of a small abscess or abscesses (Milroy Paul).

2. When an abscess forms the liver enlarges, most often in an upward direction. It is at this stage that surgical intervention is called for.

3. It may become encapsulated and remain dormant for long periods.

4. Unrecognised and untreated, it may burst into (*a*) the right lung, (*b*) the peritoneal cavity, (*c*) the right pleural cavity, in that order, or, more

Milroy Paul, Contemporary. Formerly Professor of Surgery, University of Ceylon.

rarely, into a hollow viscus (fig. 931). Exceptionally the abscess points subcutaneously. Erosion into the lung and the expectoration of a quantity of chocolate-coloured sputum sometimes results in a natural cure.

Bacterial infection is a rather frequent and serious complication of amœbic abscess. While a solitary amœbic abscess is usually amenable to combined specific drug and surgical treatment, the prognosis in cases of multiple amœbic liver abscesses is extremely poor.

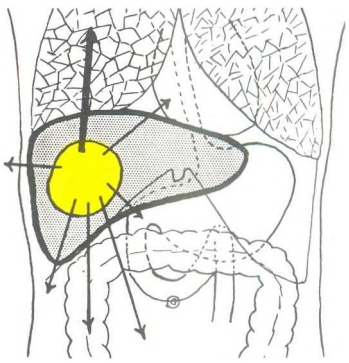


FIG. 931.—Directions in which a tropical liver abscess may burst. (*After Sir Zachary Cope, F.R.C.S.*)

Clinical Features.—As a rule, the condition develops soon after an attack of amœbic dysentery while the patient is resident in a tropical or sub-tropical country. Less frequently its appearance is delayed, sometimes for many months; exceptionally it has occurred more than thirty years after returning home from the tropics. Occasionally an amœbic abscess develops in a carrier who has not had overt dysentery; indeed, it sometimes appears in persons who have had mild diarrhœa not diagnosed as dysentery, and consequently have not had treatment for that condition. There is a striking predominance in young males.

Early Symptoms.—Anæmia, loss of weight, and an earthy complexion are often the first symptoms.

Pyrexia rising to 38°C. or more, at night, with profuse sweating, is usually present. Rigors occur, especially in the early stages.

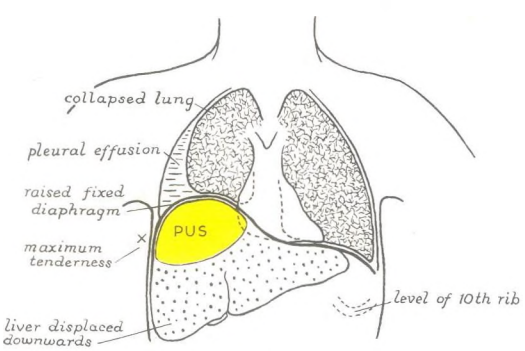


FIG. 932.—The physical signs of a tropical liver abscess (commonest site). (*After A. T. Andreasen.*)

Pain is constantly present in the liver area, and is occasionally referred to the right shoulder. Jarring increases the pain so that the patient learns to try to support his enlarged liver with his hands when he walks.

Tenderness and *rigidity* in acute cases are comparable to that of acute cholecystitis. In chronic cases tenderness may be absent.

Enlargement of the liver may be demonstrated (fig. 932), but it is not unusual for an abscess to be present in a liver which is fixed by perihepatitis, and therefore the liver cannot enlarge in a downward direction.

Scanning of the liver, using Rose Bengal, Indium, Technetium, radioactive colloid gold, or by employing *ultra-sound* techniques, may help to localise the abscess.

Basal lung signs on the corresponding side.

Leucocytosis is present in nearly all cases. Polymorphonuclear cells constitute, at the most, only 75% of the total count. Anæmia, if present at all, is not marked.

Examination of several stools for amœbæ and cysts should be undertaken, but their absence does not exclude the diagnosis.

Sigmoidoscopy sometimes reveals the characteristic ulcers (p. 1001).

Radiography (antero-posterior and lateral positions) often reveals an elevation and fixation of the right cupola of the diaphragm.

Often an absolute diagnosis is possible only on finding typical pus by exploratory aspiration.

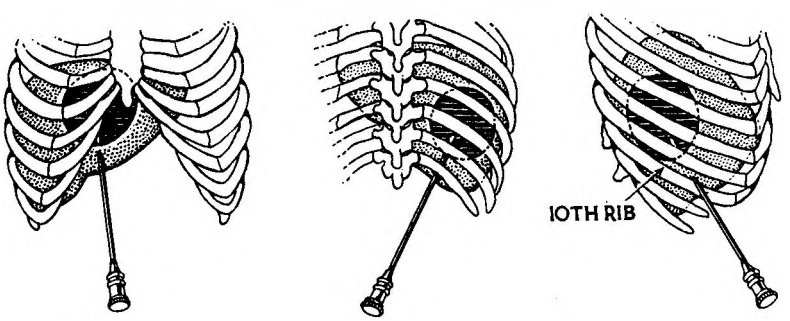
Occasionally an amœbic liver abscess does not give rise to symptoms; healed lesions have been found at necropsy and calcified lesions (that may have been abscesses) have been revealed radiologically.

Liver function tests are often unhelpful and liver biopsy is not recommended because of the risk of contamination of the pleural cavity. Aspiration of the abscess will not necessarily confirm the diagnosis, because often amœbæ are not seen.

Serological Tests.—The indirect hæmagglutination test, which is sensitive and reliable, is positive in most affected patients. The cellulose acetate precipitant test is a good indicator of active disease.

Treatment.—The treatment of choice is metronidazole (Flagyl) 800 mg three times daily for 7–10 days. Failures are few and side effects much less than with the more toxic emetine and chloroquine.

Aspiration.—Small abscesses respond without aspiration. Needle aspiration should be used if there is a lack of response to five days of metronidazole treatment. A secondarily infected abscess should be treated similarly in the first instance; indeed, such infection can be discovered only by bacteriological examination of the pus.



(*a*) (*b*) (*c*)

FIG. 933.—Aspiration of an amœbic hepatic abscess: (*a*) in the anterior part of the liver; (*b*) in the posterior part of the liver; (*c*) located near the dome of the diaphragm.

Technique.—Aspiration must be conducted in the operating theatre. A long needle is necessary, and its bore should be wide (1–2 mm), as the pus is usually thick in consistency. The important technique of percutaneous introduction of the hollow needle (under local anæsthesia) into the abscess cavity in various locations is shown in fig. 933. In some cases the abscess eludes the aspirating needle, in which event laparotomy must be performed, thus allowing the liver to be explored by the aspirating needle more thoroughly. At the same time laparotomy permits the exclusion of a primary carcinoma of the liver which, in coloured races, sometimes closely

resembles an amœbic liver abscess in its onset and physical signs, and at times is even accompanied by a low pyrexia. When the abdomen is open, packs, impregnated with a solution of erythromycin 1:1000, are so arranged as to isolate the liver. On aspiration the abscess is thoroughly emptied by syringe suction. The incision is closed completely and on no account should a drainage tube be employed. Its use leads to invasion of its track by amœbæ and in some cases a fatal spreading infection of the abdominal wall Amœbiasis cutis (fig. 934).

After Treatment.—A full course of specific drug therapy should be given after aspiration.

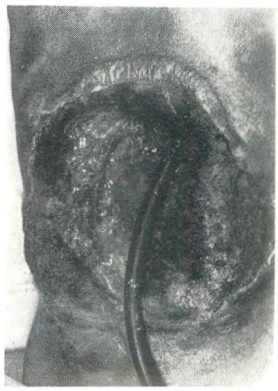


FIG. 934. — Amœbiasis cutis following drainage of a ruptured amœbic abscess. (*Prof. R. Milnes Walker, Bristol.*)

ACTINOMYCOSIS OF THE LIVER

Actinomycosis (p. 38) is due to the fungus *Actinomyces israeli*, and produces the well-known 'honeycomb' liver. Actinomyces reach the organ in one of the following ways:

1. Via the portal vein from actinomycosis of the right iliac fossa (60 per cent).
2. Via the hepatic artery from a more distant primary focus, e.g. facio-cervical actinomycosis—about 30 per cent.
3. From contiguous viscera, e.g. a penetrating peptic ulcer invading the liver—rare.

The disease is slow to develop, and causes swelling of one or other lobe. Gradually the liver tissue is destroyed and replaced by multiple abscesses.

Treatment.—Exploration is essential, and in every case of liver abscess of doubtful origin the pus should be examined for actinomyces. Long-acting penicillin in large doses, or other more suitable antibiotic therapy (p. 17) must be continued for some months. Usually by the time the liver is involved the prognosis is grave, but not hopeless.

TUBERCULOSIS OF THE LIVER

In 50 per cent. of necropsies upon patients who have died from tuberculosis, miliary tubercles are present in the liver. Local tuberculosis of the liver is usually diagnosed at necropsy, but occasionally at laparotomy. There is a large mass containing necrotic material. This lesion may be seen in children, and in negroes with little immunity. Less uncommon is a tuberculous abscess. The symptoms are identical with those of an amœbic abscess.

HEPATIC SYPHILIS

Syphilis, always an accomplished actor, can, and does, deceive the clinician. Gummas give rise to rounded masses in the liver. These swellings may simulate a liver abscess. Syphilis of the liver is now a rarity.

HYDATID DISEASE OF THE LIVER

Although the larval stage of the parasite *Echinococcus Granulosus* can thrive in many parts of the body, in 80 per cent of cases it does so in the liver; after ingestion it enters this organ through a radicle of the portal vein, and usually the right lobe is affected.

Source of Infection.—Dogs are the chief mediators of hydatid disease to human beings by direct contact. Humans may also eat salads on which the ova have been deposited. Animals, particularly sheep, are infected by eating contaminated grass (fig. 935). Once in the stomach, the ova burrow through the stomach wall to enter the portal system and the liver. Dogs become infected by feeding on offal of infested sheep and, to a lesser extent, cattle. As would be expected, the disease is relatively common in the sheep-rearing districts of Australasia and South America, Greece, Turkey, Iran

and Iraq, while, for the same reason, in the British Isles, Wales shows the highest incidence.

In other parts of the world where the disease is common the life-cycle can be completed in other animals; thus pigs (not kept in styes) and occasionally horses can take

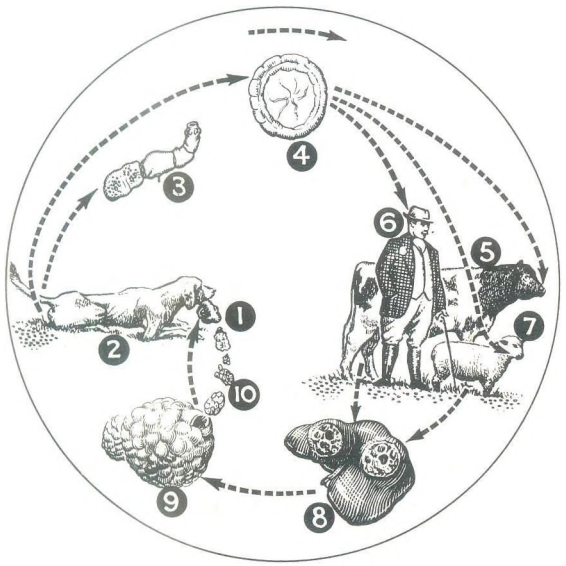


FIG. 935.—The life-cycle of the *Echinococcus Granulosus*. (1) Offal infected with hydatid cysts is eaten by a dog (2). The *Echinococcus Granulosus* (3) develops in the dog's intestine. This parasite is about 1 cm long and is made up of a head and three segments, the last of which contains about 500 ova (4). The ova are expelled from the dog's intestine on to grass, vegetables, etc. Cattle (5), human beings (6), or sheep (7) ingest the eggs. The liver (8) is the organ most frequently infected with hydatid cysts, a larval form of *Echinococcus Granulosus*. Such cysts (9) harbour thousands of heads of the parasite (scolices) (10).

(*After V. P. Fontana, Montevideo.*)

the place of sheep and cattle, while in the frozen North (the disease is common among Red Indians and Eskimo) the wolf → moose → wolf often maintain the cycle.

Pathology.—A hydatid cyst consists of three layers.

1. *The adventitia* (*pseudocyst*), consisting of fibrous tissue, the result of reaction of the liver to the parasite, is grey in colour and blended intimately with the liver, from which it is inseparable.

2. *The laminated membrane* (*ectocyst*), formed of the parasite itself is whitish and elastic, and contains the hydatid fluid. Indeed, this membrane closely resembles a child's uncoloured balloon filled with water, and unless bacterial infection has occurred it peels readily from the adventitia.

Hydatid fluid is crystal-clear; it registers a specific gravity of 1·005–1·009,

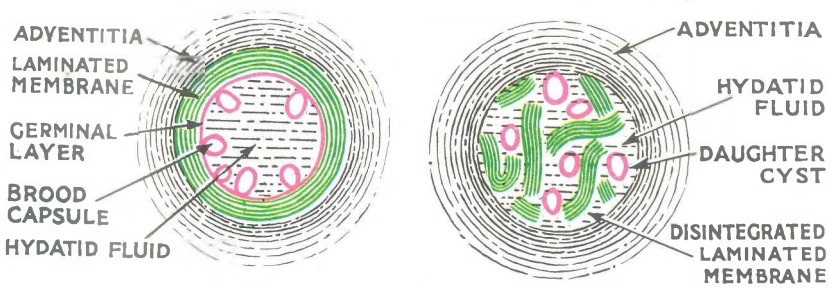


FIG. 936.—(*a*) Typical hydatid cyst.
(*b*) Development of daughter cysts (not common).

contains no albumin, and when not too old, hooklets and scolices. The cyst grows very slowly.

3. The only living part of a hydatid cyst is a single layer of cells (germinal epithelium) lining the cyst (*endocyst*). This secretes: (*a*) internally: the hydatid fluid; (*b*) externally: the laminated membrane (fig. 936 (*a*)). Brood capsules within the cyst develop from the germinal epithelium and are attached by pedicles to its innermost wall. Within the brood capsules, scolices (heads of future worms) develop. Should the laminated membrane become damaged, it disintegrates (Dew), and the brood capsules, becoming free, grow into daughter cysts (fig. 936 (*b*)). In this event the mother cyst ceases to exist as such, the hydatid fluid and its content being confined by the adventitia only.

Clinical Features. — For a long time, perhaps for some years after the original infestation (which often occurs in childhood), a hydatid cyst remains symptomless (fig. 937). In the course of time a visible and palpable swelling in the upper abdomen is discovered. The size which a hydatid may attain without causing serious disturbance to health, would seem to be limited only by the capacity of the peritoneal cavity.

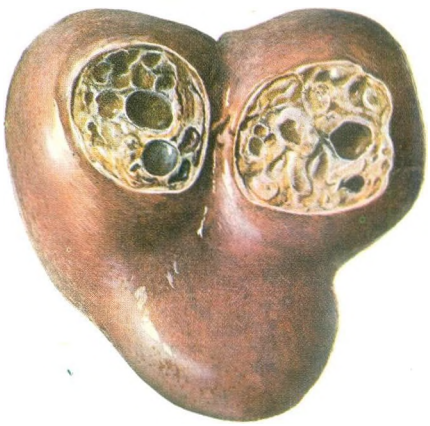


FIG. 937.—Multiple hydatid cysts in the liver. The patient, who had never left England, died after a street accident.

Naturally, when a patient hails from a locality where hydatid disease is rife, the diagnosis is simplified. In obscure cases radiography (figs. 938 and 940), scanning (fig. 939) and immunological tests are of great diagnostic assistance.

The intradermal test (Casoni's test) is positive (fig. 941) in 75 per cent of cases.
Recent serological tests, although more complicated, are of greater accuracy.
A blood-count often, but not invariably, shows an eosinophilia (6 per cent or more).
Hepatic scintiscanning and ultrasonic scanning are invaluable in the diagnosis and localisation of the cyst and in following the course and effects of the surgical treatment.

Course of the Disease.—1. Occasionally the parasite dies. The fluid is absorbed, and all that remains is an encapsuled, laminated, bile-stained membrane. In cases of long standing the walls of the cyst may calcify (fig. 938).

2. Usually the cyst enlarges gradually, and becomes manifest by its size. Sometimes sessile cysts float in the peritoneal cavity, or they may be confused with mesenteric, pancreatic or renal cysts (Talib).

3. Complications arise. ' It is the onset of complications that makes the morbidity not much inferior to that of malignant disease ' (Fortacin).

Sir Harold Dew, 1891–1962. Professor of Surgery, Sydney, Australia.
Tommaso Casoni, 1880–1933. Physician, Ospedale Coloniale, Vittorio Emanuele III, Tripoli.
H. Talib, Contemporary. Surgeon, Baghdad University Medical School, Iraq.
José Blanco y Fortacin, Contemporary. Formerly Professor of Surgery, Madrid, Spain.

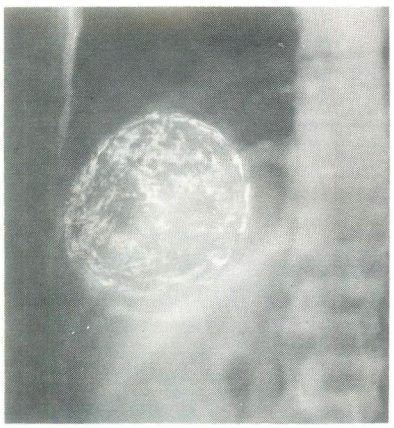


FIG. 938.—Calcification of hydatid cyst of the liver. *(H. Talib, F.R.C.S., Baghdad.)*

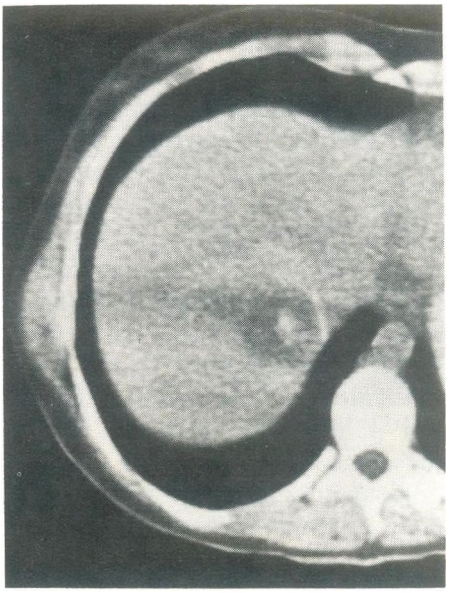


FIG. 939.—Liver CT scan (viewed from the feet of the patient). Calcifying hydatid cyst in posterior (paravertebral) part of right lobe.

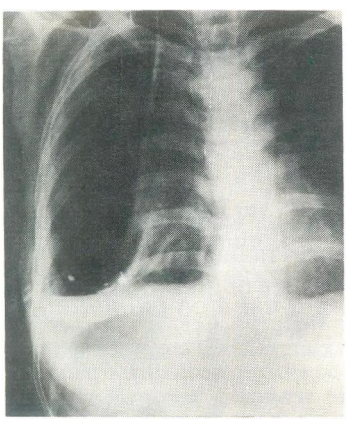


FIG. 940.—Biliary-pleural-bronchial fistula from hydatid cyst of the liver. See fluid levels in cyst and pleural cavity. *(H. Nassir Pur, Teheran.)*

FIG. 941.—Casoni's test. Positive reaction.

Complications

- Jaundice
  - due to pressure of cysts on the biliary ducts
  - due to cysts within the bile ducts
- Rupture
  - Into the peritoneal cavity
  - Into the alimentary canal
  - Into the biliary channels
  - Into a pleural cavity (fig. 940)
- Suppuration

Rupture into the peritoneum is accompanied by profound shock, and all the signs of diffuse peritonitis. As with any case of rupture of a hydatid cyst, anaphylactic phenomena, notably urticaria, are prone to occur. It is wise to give hydrocortisone by injection for a day before, during and for a day after, any surgical procedure. The treatment of intraperitoneal rupture must be immediate, and directed to combating shock and cleansing the peritoneal cavity. Even in those who survive, the ultimate prognosis is poor, for if, as is usual, the cyst contains brood capsules, however meticulous the cleansing, the disease tends to become disseminated within the peritoneum.

Treatment of Hydatid Cyst of the Liver

Hitherto, the only treatment has been surgical but recently the drug, mebendazole, in doses of 400 to 600 mg three times a day for 21 to 30 days, has been found to be effective in some patients. The drug is still under trial and not all reports so far have been encouraging.

Operation.—The cyst is exposed by an incision that gives the best access. Abdominal packs are arranged meticulously so as to isolate that portion of the liver containing the cyst from the peritoneal cavity. A large black pack, wrung out in 2 per cent. formalin, is tucked around the exposed liver—*black* so that daughter cysts and scolices will show up against the background—*formalin* because this antiseptic kills the parasites. The cyst is aspirated, and a suitable quantity of 10 per cent. formalin solution is injected so as to render the cyst about three-quarters full. An incision is made through the liver overlying the cyst, and the adventitia is opened. This brings the rubbery laminated membrane into view. Very gently the laminated membrane is grasped with sponge forceps and separated from the adventitia with a finger. The aim should be to separate the laminated membrane and deliver the cyst intact; usually this can be accomplished in cysts uncomplicated by the effects of past infection. In complicated cases it is sometimes necessary to remove the laminated membrane piecemeal. When the intact cyst can be enucleated, the resulting cavity in the liver can be closed completely. In less favourable circumstances it is advisable to drain the cavity, particularly when infection is present, when the contents of the cyst are bile stained, or when there is uncertainty of its complete removal. An attempt should be made to remove the adventitia in order to allow the cavity to collapse. Every precaution must be taken to prevent spilling the contents of the cyst into the peritoneal cavity or the layers of the abdominal wall, otherwise dissemination is likely.

NON-PARASITIC CYSTS OF THE LIVER

A solitary traumatic cyst usually follows abdominal trauma and the symptoms are delayed weeks or months after the injury. The clinical manifestations include abdominal pain, frequently referred to the shoulder, a lump that is usually palpable, and less frequently jaundice. In most instances the cyst is located in the right lobe and contains blood and bile. Drainage cures the condition.

A localised collection of cysts is probably the result of sequestration of bile ducts during fœtal life. If large, the mass may require excision.

Congenital polycystic disease of the liver is rare, and a third of the cases are associated with congenital polycystic kidneys. Slow, irregular enlargement of the whole liver in adult life is the only clinical feature. The patient may eventually succumb from renal or hepatic failure, or the effects of portal hypertension.

BUDD-CHIARI'S SYNDROME

This syndrome results from obstruction to the hepatic veins. Neoplastic encroachment from other organs accounts for the majority of cases. The syndrome is also associated with clotting diseases, especially polycythæmia and the use of hormones for contraception and infertility. Some cases, presumably congenital, are caused by an obstruction of the suprahepatic portion of the inferior vena cava with a membraneous web. The importance lies in its potential curability by operation.

An acute form of the disease is seen in Jamaica and may be caused by plant extracts such as Senecio and Crotalaria used in herbal tea.

Clinical Features.—*In acute cases*, nausea, vomiting and severe pain, due to rapid

George Budd, 1808–1882. Professor of Medicine, King's College Hospital, London. He was probably the first to describe the Budd-Chiari syndrome.
Hans Chiari, 1851–1916. Professor of Pathological Anatomy, Prague.

enlargement of the liver as a result of congestion, are often followed by death from hypotensive shock.

A less sudden onset is characterised by rapidly accumulating ascites and signs of portal hypertension with a developing collateral circulation. Signs of hepatic insufficiency are usually pronounced, and early death and hepatic coma are the rule. The simultaneous development of œdema of the legs testifies to involvement of the inferior vena cava in the occlusive process.

Chronic cases closely resemble hepatic cirrhosis which, indeed, may develop. Liver function tests indicate severe hepatic parenchymal damage, and rarely does the patient survive more than a few months. A few patients who do not die from hepatic coma succumb to hæmorrhage from œsophageal varices, mesenteric infarction, or intercurrent infection. In short, the Budd-Chiari syndrome, when the occlusion is, or becomes, complete, must be looked upon as a harbinger of death.

Treatment.—In patients with membranous obstruction of the inferior vena cava, a transatrial meatotomy may be considered. In those in whom the portal vein and inferior vena cava are patent, a portacaval shunt should be tried for the relief of portal hypertension (but *not* an end-to-side shunt, fig. 955).

CIRRHOSIS OF THE LIVER

Hepatic cirrhosis is essentially a necrosis of the liver followed by fibrosis and regeneration of liver cells, and involving the whole organ.

Ætiology.—It is convenient to divide the ætiological factors into those occurring in infancy and childhood and those occurring in adult life. It will be understood that some of the factors appertaining in early life take years to manifest themselves as hepatic cirrhosis. Obstructive jaundice is a cause common to all ages.

Causes in Infants and Children:

1. *Atresia of the bile ducts.*—The commonest cause in infants is congenital atresia of the bile ducts (p. 914), irremediable by operative means. The jaundiced child soon shows signs of hepatomegaly, splenomegaly, ascites, and œsophageal varices. Most survive only a few months; a few up to three or more years.

2. *Nutritional.*—(*a*) Infantile cirrhosis in India occurs mainly in high-caste Hindu vegetarians. It often progresses rapidly to an early fatal termination. It is suggested that the condition is due to a susceptibility of protein-deficient children to massive necrosis of the liver during viral hepatitis. (*b*) Kwashiorkor in Africa is characterised by an enlarged liver and peripheral nutritional œdema. This disease is caused by protein malnutrition. Children are most commonly affected 6–18 months after weaning. Generalised œdema and skin disorders develop along with apathy and arrested growth. There is no great evidence that this disease proceeds to cirrhosis. (*c*) Galactosæmia, patients with this inborn error of metabolism who continue to take lactose invariably develop cirrhosis.

3. *Infective.*—A history of severe jaundice in early infancy is obtainable in a number of instances. Virus hepatitis is believed to play a part in the production of cirrhosis.

4. *Congenital Hepatic Fibrosis.*—An abnormality often associated with medullary sponge kidney. The liver shows a dense, fibrous band surrounding normal hepatic lobules but function usually remains good. Portal hypertension is common. If hæmorrhage occurs, these patients are excellent candidates for shunt operations.

5. *Erythroblastosis Fœtalis.*—Isolated instances have occurred.

Causes in the Adult:

1. *Alcohol.*—Over-indulgence in alcoholic beverages is a leading cause. Nutritional deficiency is probably an accessory factor.

2. *Virus Hepatitis.*—The exact percentage of cases in which cirrhosis is an aftermath of virus hepatitis is debatable, and it varies in different localities.

3. *Chronic (Surgical) Biliary Obstruction,* leading to Secondary Biliary Cirrhosis.—This is to be distinguished from Primary Biliary Cirrhosis (Hanôt) an autoimmune disease of middle-aged women. In the latter the mitochondrial immunofluorescence test is positive (in 98%, Sherlock).

4. *Infestation of the liver by schistosomiasis* undoubtedly is a principal cause of the condition in districts where schistosomiasis is rife (p. 1103).

5. *Nutritional Deficiency.*—In the tropics this appears to be a major factor. Its mode of action is as follows: some toxic agents and mild infections, harmless to the well-nourished, are liable to have a deleterious effect on the liver of protein-deficient individuals. Therefore protein deficiency is indirectly responsible for the cirrhosis.

6. *Hæmochromatosis* (Bronzed Diabetes).

7. *Hepato-lenticular degeneration* (p. 897).

8. *Chemical poisons:* carbon tetrachloride, arsenic, chloroform.

9. *Unknown.*—In many instances the cause is obscure. These account for half the cases seen in Great Britain (Sherlock).

Morphological Types

Three anatomical types are recognised:

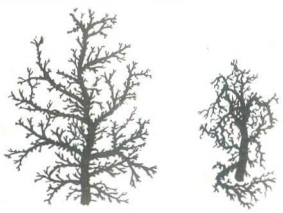
(*a*) *micronodular* is characterised by irregular thick bands of fibrous tissue and by small regenerating nodules. It is often associated with alcoholism.

(*b*) *macronodular* is characterised by bands of fibrous tissue of varying thickness and nodules of variable size. This type is also called postnecrotic.

(*c*) *mixed*—liver shows both micro- and macronodular features.

Pathology.—As a result of widespread parenchymal destruction and overgrowth of fibrous tissue, the tiny radicles of the portal and hepatic veins become compressed and distorted (fig. 942). In some cases there may be spasm of the portal veins. Arterioles resist this compression for a longer time than veins. As a consequence the cirrhotic liver becomes dependent upon the hepatic artery for the major portion of its blood supply, and in the desperate effort to maintain the circulation through the liver in these adverse circumstances, extensive intrahepatic communications develop between branches of the hepatic artery and the portal vein, and between the tributaries of the portal and hepatic veins.

Nature's intrahepatic shunts, combined with the opening up of the better-known extrahepatic porta-systemic communications, divert a large portion of the portal blood past the hepatic parenchyma into the systemic venous system. Thus the remaining hepatic cells are deprived of their fair share of portal blood.



A B

FIG. 942.—A, Cast of branches of a normal portal venule; B, Those of the branches of a portal venule of a cirrhotic liver, showing their attenuation and distortion. (*After I. S. Ravdin.*)

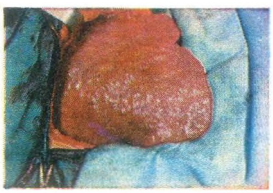


FIG. 943.—Portal cirrhosis.

Usually there is a fine fibrosis producing slight nodularity (Laënnec's cirrhosis[1] or hob-nail liver) (fig. 943), but coarse lobulation may result from larger regeneration nodules (fig. 944).

Clinical Features:

The early stages of cirrhosis may be quiet and long. The liver hypertrophies. In some cases there are repeated attacks of slight jaundice with epigastric pain and vomiting. At each attack areas of the liver are destroyed and replaced by fibrous tissue, while the remainder hypertrophies.

[1] Laënnec introduced the term from the Greek *κιρρός* = tawny, as the nodules are orange-yellow in colour.

Dame Sheila Patricia Violet Sherlock, Contemporary. Professor of Medicine, Royal Free Hospital, London.
René Théophile Hyacinthe Laënnec, 1781–1826. Professor of Medicine, College de France, invented the stethoscope in 1819.

Cirrhosis results in two major events—hepatocellular failure and portal hypertension. Later the liver commences to shrink. The spleen becomes enlarged, and usually increases in size with the raised portal blood pressure of which hepatic cirrhosis is the principal cause in adults. Functioning liver tissue is gradually destroyed and hepatocellular failure develops. About this time, especially in alcoholic subjects, two classical signs may appear: *Spider nævi*[1], which are usually located around the face, neck, shoulders and the upper arms. Histologically they prove to be an overgrown end-artery with branching arterioles (fig. 945). *Palmar erythema*—the hands are warm, and the palms are bright red.

FIG. 944.—Portal cirrhosis with large regeneration nodules. (*Prof. R. Milnes Walker, F.R.C.S., Bristol.*)

FIG. 945.—Spider nævus.

Bleeding from œsophageal varices (p. 898) occurs in a high proportion of cases, and may be the first evidence of the disease. Not infrequently the superficial veins radiating from the umbilicus enlarge, forming a Caput Medusæ. Progressive ascites occurs (fig. 946). Testicular atrophy and gynæcomasia are common in cirrhosis, no doubt due to increase of œstrogens in the blood. The failing liver is unable to neutralise these substances. In women there is a tendency to masculinisation. Dupuytren's contractures may develop.

Once hepatic decompensation has occurred (if the patient escapes death

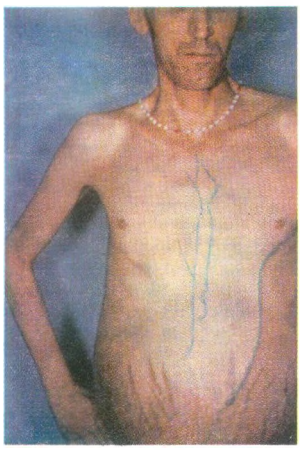


FIG. 946.—Caput Medusæ, and striæ in cirrhosis with ascites.

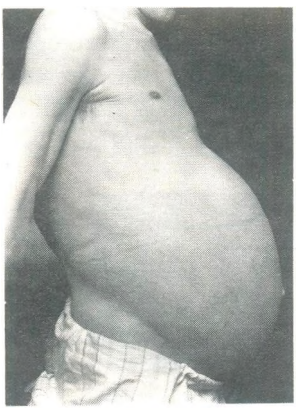


FIG. 947.—Hepatic cirrhosis with ascites.

[1] An older Miss Muffet
Decided to rough it
And lived upon whisky and gin.
Red hands and a spider
Developed outside her—
Such are the wages of sin.
(*W. B. Bean.*)

William Bennett Bean, Contemporary. Professor of Internal Medicine, University Hospitals, Iowa City, U.S.A. Medusa, one of three Gorgons whose fine hair was turned into snakes (Greek mythology).

from torrential hæmorrhage from œsophageal varices), liver failure (p. 879), like a sword of Damocles, sooner or later falls upon the sufferer from this disease.

Clinical Pathological Associations.—Cirrhosis, whatever its type, has certain clinical and pathological associations including splenomegaly, pancreatitis (especially in the alcoholic), gastrointestinal bleeding from varices, peptic ulcer or gastric erosions, primary liver cancer and liability to infections.

Liver Function Tests (p. 880).—In compensated cirrhosis, tests may be normal, the most frequent abnormality being impairment of B.S.P. clearance. In decompensated disease, urobilinogen is usually present in the urine in excess and the plasma albumin is depressed.

Liver biopsy (p. 881) is usually necessary only for the diagnosis of a well-compensated case in order to confirm the diagnosis and assess prognosis.

Treatment.—There is no specific remedy for established cirrhosis, but the following measures may be helpful: (*a*) increased glucose intake; (*b*) alcohol forbidden; (*c*) high protein diet if there is no evidence of liver failure; (*d*) Steroids should only be given in active chronic hepatitis.

Hepato-lenticular degeneration (*syn.* Wilson's disease) is an uncommon condition that is confined to children and young adults due to an hereditary error in copper metabolism. In infants the symptoms are those of cirrhosis. In adolescents a coarse tremor is commonly an initial symptom. As the disease progresses, muscular rigidity dominates the picture. Kayser-Fleischer rings in the eyes due to peripheral corneal opacity are present. Liver function tests give varying results. Liver biopsy shows cirrhosis. Treatment is by penicillamine, which may have to be given for several years.

PORTAL HYPERTENSION

Ætiology.—Increase of the portal venous blood pressure is due to an obstruction which can be (1) Pre-hepatic; (2) Intra-hepatic; (3) Post-hepatic.

1. **Pre-hepatic.**—About 20 per cent. of patients belong to this group, most of whom are children. The obstruction arises in one of two ways:

(*a*) *There is congenital absence or abnormality of the portal vein.*

(*b*) *Thrombosis of the portal vein* due to extension of the normal obliterative process of the umbilical vein and ductus venosus (fig. 948) sometimes associated with omphalitis of the new-born (p. 1126).

The vein becomes replaced by a mass of collateral channels which have been described as a 'cavernoma'.

2. **Intra-hepatic** accounts for nearly 80 per cent of all cases. The causes are cirrhosis (frequently alcoholic in origin) and schistosomiasis in which the blood-flow through the liver is obstructed, but hepatic function is remarkably preserved.

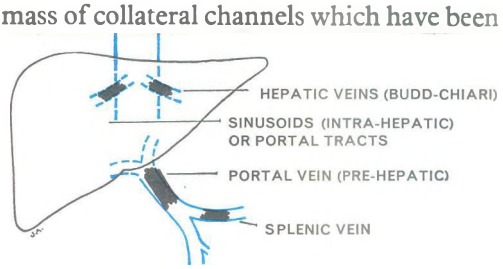


FIG. 948.—Sites of obstruction. The blocks in the veins represent possible sites of thrombus formation. (*After Sir James Learmonth, Edinburgh.*)

3. **Post-hepatic** is rare. It may be caused by constrictive pericarditis and tricuspid valvular incompetence, and it is also a component of the Budd-Chiari syndrome (p. 893).

Damocles, a Greek who was feasted with a sword suspended above his head by a single hair (Greek mythology).
Samuel Alexander Kinnier Wilson, 1878–1937. Neurologist, King's College Hospital, London.
Bernhard Kayser, 1869–1937. German Ophthalmologist.
Richard Fleischer, 1848–1909. German Physician.

Collateral Circulation.—When there is obstruction of blood flow to or from the liver, Nature endeavours to relieve the obstruction by opening up normally insignificant anastomotic channels.

When the obstruction is pre-hepatic, collaterals between the portal vein distal to the obstruction and the portal vein proximal to the obstruction enlarge. Thus almost unheard-of venæ comitantes of the hepatic artery and of the portal vein wax proud. Depending upon the site of the obstruction, some of the collaterals between the portal and systemic venous systems, notably the œsophageal plexus, also become dilated.

When the obstruction is intra-hepatic, anastomotic channels outside the liver between the portal and systemic systems become engorged, dilate, and so

ANASTOMOSES BETWEEN THE PORTAL AND SYSTEMIC VENOUS SYSTEMS

	Site of Anastomosis	*Portal Vessels*	*Systemic Vessels*	*Signs and Symptoms*
1	Plexus around lower end of œsophagus.	Œsophageal branches of left gastric vein.	Lower systemic œsophageal veins.	Hæmatemesis or melæna.
2	Around umbilicus.	Para-umbilical veins (accompany the round ligament of the liver).	Superficial veins of the anterior abdominal wall.	Caput Medusæ.
3	Plexuses around lower third of rectum and anal canal.	Superior hæmorrhoidal vein.	Middle and inferior hæmorrhoidal veins.	Hæmorrhoids (rare).
4	Extraperitoneal surfaces of abdominal organs.	Tributaries of superior and inferior mesenteric veins.	Subdiaphragmatic and retroperitoneal veins.	Silent.

an increasing proportion of the obstructed portal venous blood by-passes the liver. The only ones which are dangerous to life are those in the submucosa of the œsophagus and upper end of the stomach. In patients with portal hypertension, hæmorrhoids, if present, are usually of the idiopathic type (p. 1089).

FIG. 949.—Barium swallow showing filling defects due to œsophageal varices.

Œsophageal Varices.—*Although called 'œsophageal', it is important to realise that these varices extend well into the stomach.* Œsophageal varices (fig. 949) are the most important collaterals of the portal circulation. Sometimes, as intra-abdominal pressure rises, e.g. when straining at stool or during heavy lifting, they rupture; in other cases the overlying mucosa becomes abraded by a rough bolus or excoriated by regurgitated gastric juice. Bleeding can occur slowly or catastrophically. The presence of œsophageal varices can be demonstrated by:

(*a*) *Radiology* after a barium swallow (fig. 949). In the great majority of cases œsophageal collaterals can be revealed by this method as filling defects which may extend even as far up as the pharynx. Varices can also be demonstrated by the injection of a radio-opaque dye percutaneously into the splenic pulp (splenic portography,

p. 861). This investigation is valuable because not only can the cause of portal hypertension be demonstrated, e.g. portal thrombosis or cirrhosis, but the splenic pulp pressure may be determined at the same time as an estimate of portal vein pressure. Occasionally superior mesenteric angiography and umbilical vein catheterisation may have to be performed.

(*b*) *Œsophagoscopy* will demonstrate them in all cases, and should be carried out along with gastroscopy to confirm the presence of varices and determine if they are the source of bleeding. The risks are not great in skilled hands.

Clinical Features.—If the patient is known to have hepatic cirrhosis, it should be assumed that the hæmorrhage is coming from œsophageal varicose veins, although patients with cirrhosis of the liver are particularly likely to suffer also from a gastric or duodenal ulcer. In nearly all cases the spleen is palpably enlarged. Liver function tests, if abnormal, greatly favour hepatic cirrhosis as the cause of the hæmorrhage. They are of value in deciding whether operation should be carried out because in those with a low serum albumin (below 25 g/l) and a high serum bilirubin the results of operation are poor.

The decision to operate in a patient with portal hypertension requires nice judgment and considerable experience. It has been proved beyond doubt that a *prophylactic* shunt operation, that is an operation to reduce portal pressure in a patient with portal hypertension but who has not yet had a hæmorrhage, is of no benefit and is probably deleterious. In addition, any operation in a patient, especially an elderly one, with severe encephalopathy and marked impairment of liver function is usually followed by high mortality. On the other hand, a patient who is otherwise fit and young, without evidence of a severe liver disease, will probably benefit from an early shunt after he has had a hæmorrhage, especially if there has been a short interval to restore him to hæmodynamic equilibrium by blood transfusion. In general, emergency operations carried out while the patient is still bleeding are followed by poor results.

Treatment of Massive Hæmorrhage from Œsophageal Varices:

1. **Conservative Treatment.**—Rest, sedation, and replacement of blood will carry a few of the patients who are actively bleeding past the emergency period. For transfusion, fresh blood is particularly desirable. Careful monitoring of the blood pressure and central venous pressure is necessary. Encephalopathy may be prevented or minimised by giving magnesium sulphate and neomycin orally or through the Sengstaken tube (p. 900). Occasionally ice water lavage of the stomach may be performed to reduce bleeding and remove clots. Pituitrin (vasopressin) which causes lowering of the portal blood pressure by constricting the splanchnic arterioles, is a valuable adjunct. Conventionally, 20 units diluted in 200 ml of isotonic saline solution is given intravenously over a period of 20 minutes. Side effects include abdominal griping and coronary artery vasoconstriction. A better method of administration is a *continuous* infusion of vasopressin at a

Robert William Sengstaken, Contemporary. Surgeon, Garden City, New York.

rate of 0·3 units per minute over a period of 24 hours. Occasionally vasopressin may be transfused directly into the splanchnic circulation via a catheter inserted into the superior mesenteric artery from the femoral artery, but it is doubtful if this route of administration carries greater benefit than the intravenous one. For the prothrombin deficiency consequent upon liver damage, the administration of vitamin K is necessary.

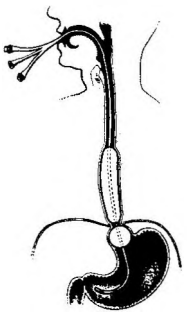


FIG. 950.—The Sengstaken trilumen tube with gastric and œsophageal balloons *in situ*.

2. **Tamponade** is required when the foregoing measures fail after a short trial. A small balloon tied firmly to the end of a gastric aspiration tube can be employed, but the Sengstaken trilumen tube (fig. 950) is better. The œsophageal and gastric balloons are inflated and moderate tension applied by strapping the tube to the forehead. While the Sengstaken tube controls œsophageal hæmorrhage in every case, and is therefore a diagnostic as well as a therapeutic measure, it has its drawbacks. Chief of these is the inspiration of saliva into the lungs, which must be anticipated. In addition to respiratory difficulty, ulceration and rupture of the œsophagus may occur if the balloon is retained for more than seventy-two hours. But the greatest danger is renewed hæmorrhage when the balloon is deflated and withdrawn. Therefore, after twenty-four hours the balloon should be deflated, but left in place for four hours. If bleeding recommences, immediate operation is necessary. Provided great care is taken, the Sengstaken tube represents a valuable means of stopping bleeding in the majority of patients.

3. **Urgent Operation for Bleeding Varices.**—If bleeding does not stop with vasopressin, or recurs after removal of a Sengstaken tube, operative intervention is required. Such operative intervention, no matter the procedure, carries a high mortality (40%) especially in those who have evidence of impairment of liver function. Either a shunt procedure (portacaval), or a mesenterico-caval graft can be performed, or the varices may be directly ligated or injected, although after such latter procedures bleeding may recur (see p. 904).

4. **Evacuation of Blood from the Alimentary Tract.**—As soon as possible the alimentary tract must be cleared of blood; otherwise encephalopathy, due to the absorption of protein metabolites, may follow. All unclotted blood must be aspirated from the stomach, and colonic wash-outs given until all blood has been evacuated.

Portal Hypertension *au Froid*

By this is meant that the patient has portal hypertension, but is not bleeding obviously or dangerously. First let us consider how pre-hepatic portal obstruction differs from intra-hepatic portal obstruction.

The patient with pre-hepatic portal obstruction is nearly always young, and often a child. The most usual cause for advice being sought is either a sudden hæmorrhage or listlessness due to anæmia. On examination the liver is impalpable, but the spleen is obviously enlarged (fig. 951). The anæmia is usually due to oozing from œsophageal varices, but sometimes hypersplenism is a factor (p. 876). As the liver is normal, or nearly so, liver function tests remain normal as the disease advances. Ascites does not occur unless there is active phlebitis in the portal system of veins.

Splenoportography (p. 861) will usually display the site of the block, but if the portal vein does not fill, the cause may lie in a reversed flow in the splenic vein. If, therefore, there is suspicion that the portal vein may be obstructed, a venogram may also be done by injection into a mesenteric vein at laparotomy.

The patient with intra-hepatic portal hypertension is usually an adult and suffers from hepatic cirrhosis. Enlarged porta-systemic venous communications are present long before serious œsophageal hæmorrhage occurs. Œsophageal varices can be demonstrated radiologically or seen through an œsophagoscope. Enlargement of veins of the abdominal wall radiating from the umbilicus may be present (fig. 952). As long as hypertrophy of liver tissue is sufficient to compensate for the cellular destruction by cirrhosis, the liver function tests remain within normal limits.

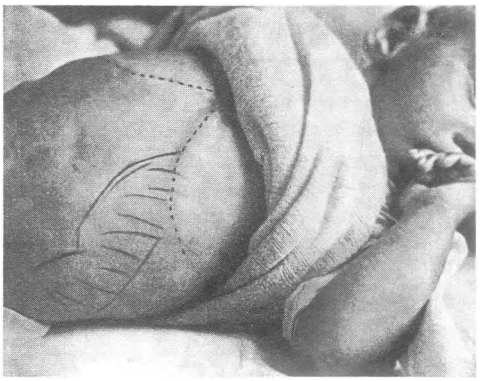


FIG. 951.—Enlargement of the spleen in pre-hepatic portal obstruction.

Measuring the Portal Blood Pressure.—One method is to record the intrasplenic pressure, and this can be done at the same time as transplenic portavenography. In cases of intra-hepatic obstruction an alternative method is to catheterise a hepatic vein. The technique is not especially difficult, and can be undertaken in any centre equipped

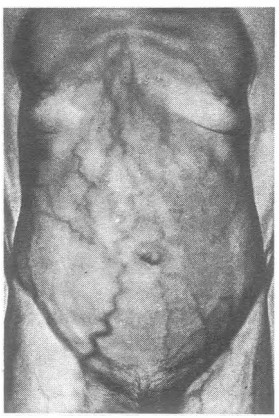


FIG. 952. — Enlargement of the veins of the abdominal wall in a case of portal hypertension. Infra-red photograph. (*The late A. H. Hunt F.R.C.S., London.*)

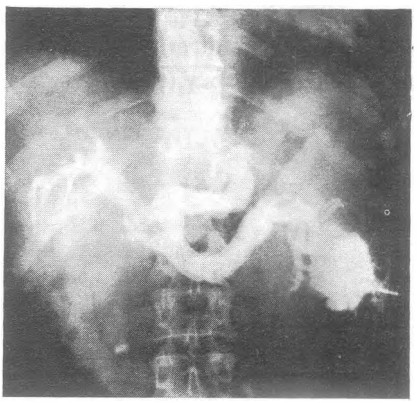


FIG. 953.—Spleno-portograph in a case of intra-hepatic portal hypertension showing dilated splenic and portal veins. (*Professor R. Milnes Walker, F.R.C.S., Bristol.*)

for cardiac catheterisation. Following local anæsthesia and opening an antecubital vein as for cannulation, a cardiac catheter with only a terminal opening is passed into the vein. The catheter is advanced through the right auricle and inferior vena cava under radiological control. The tip is then insinuated into a hepatic vein until a peripheral radicle is occluded (fig. 954). It is important that the tip of the catheter should be in a peripheral position, so as to obviate obstruction by a bifurcation of the vein, and thus falsify results. Pressure recordings are then taken. The normal portal pressure is 8 to 12 mmHg (1·06–1·6 kPa). In cases of established cirrhosis it may rise

to 30·0 mmHg (4 kPa) or more. The portal pressure is always high in cirrhotic patients who have œsophageal varices. The ascites of cirrhosis does not bear a direct relationship to elevated portal blood pressure, as was formerly believed.

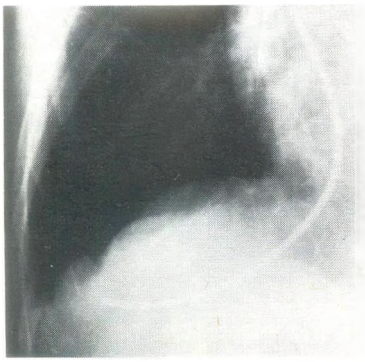


FIG. 954.—Catheter in hepatic vein.
(*Dr. Frank Ross, Bristol.*)

Indication for a Porta-systemic Shunting Operation:

If bleeding cannot be controlled, emergency operation is required but is followed by high mortality and morbidity rates. If the patient has had several episodes of bleeding in the past, the operation may be performed electively after the patient's condition has been stabilised and provided that hepatic function is not severely impaired.

Contraindications:

1. If the patient's serum albumin[1] is less than 30 g/l (3 g/100 ml).
2. If the patient is jaundiced significantly.
3. Massive ascites. Evacuation of this by suction at operation may lead to death on the table.
4. Operation should not be performed in a patient who has not bled from varices. Prophylactic operations do not prolong life and may reduce it.

Pre-operative treatment is directed mainly to correcting the accompanying hypochromic anæmia; blood transfusion is often required. Control of a bleeding tendency (secondary to impaired production of prothrombin) is

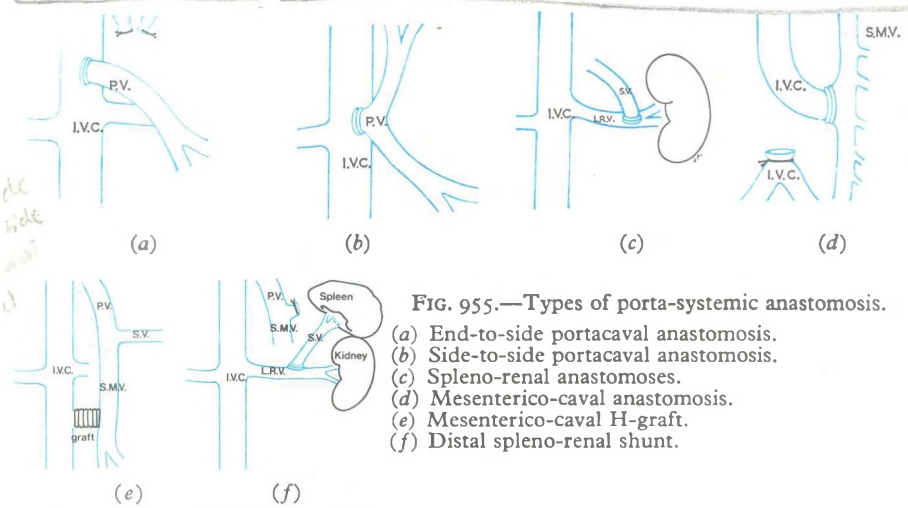


FIG. 955.—Types of porta-systemic anastomosis.
(*a*) End-to-side portacaval anastomosis.
(*b*) Side-to-side portacaval anastomosis.
(*c*) Spleno-renal anastomoses.
(*d*) Mesenterico-caval anastomosis.
(*e*) Mesenterico-caval H-graft.
(*f*) Distal spleno-renal shunt.

essential and for this purpose vitamin K is given parenterally. If there is no immediate danger of renewed hæmorrhage, those in whom a porta-systemic shunting operation is indicated are often benefited greatly by a course of pre-

[1] Normal level 3·6 to 4·5 g/100 ml (36–45 g/l).

operative medical treatment. If ascites is present, a low sodium diet, in conjunction with diuretics, often causes absorption of ascitic fluid. If possible, paracentesis should be avoided.

OPERATIONS FOR PORTAL HYPERTENSION

An anæsthetic that allows a high concentration of oxygen to be given is important. Throughout the operation the blood volume is maintained by transfusion.

Operative Portavenography.—When the transplenic portavenographic films are inconclusive, or have not been obtainable because the spleen has been previously removed, the site of the block can be demonstrated by portavenography after the abdomen has been opened. Thirty ml. of contrast material are injected into a suitable vein in the gastro-hepatic omentum, or into the superior mesenteric vein. A radiograph is then taken with a portable machine during the injection of the last few ml. Usually the site of the block can be determined accurately.

1. **Portacaval Anastomosis.**—The traditional operation to decompress the splanchnic circulation is end-to-side portacaval shunt, provided that liver function is adequate and the portal vein is patent. The serum albumin level should be at least 30 g/l, and the serum bilirubin not more than 17 mmol/l.

The best approach is a subcostal one. The peritoneum is divided along the lower border of the right lobe of the liver and the lateral border of the duodenum. The portal vein is exposed by an incision through the peritoneum forming the anterior wall of the epiploic foramen, and a length of about 3 cm freed. An incision in the peritoneum over the vena cava exposes its anterior surface. The portal vein, or its two main branches, is now ligated at the hilum of the liver, a Blalock clamp placed on its lower end, and the vein divided.

After placing a side clamp on the vena cava, an incision is made in its anterior wall, and an end-to-side anastomosis made using everting fine silk sutures, the wound is closed with drainage.

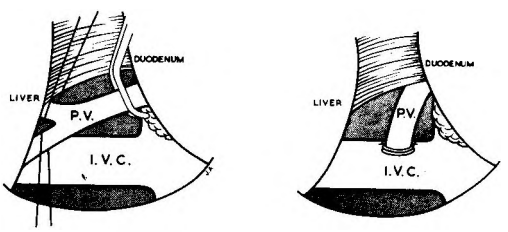


FIG. 956.—Portacaval anastomosis.

With careful selection of cases the mortality is about 6 per cent., and recurrent bleeding from varices after this operation is most unusual.

About 15–20% of patients may have post-shunt encephalopathy because of venous deprivation of the liver and slow deterioration of liver function.

2. **Spleno-renal Anastomosis.**—This may be performed if there is thrombosis of the portal vein, but it is contraindicated if the splenic vein is less than 1 cm in diameter as shown by venography. This operation is less effective than a portacaval anastomosis in preventing further hæmorrhage, but carries a lower risk of encephalopathy. Owing to the small size of the splenic vein, this operation is rarely of value in cases of extra-hepatic obstruction in children.

The operation is performed through a left thoraco-abdominal incision. The spleen is removed but during the dissection of the hilum as great a length as possible of the vein is carefully preserved. Enough vein is separated from the pancreas to allow its apposition to the renal vein, and it is temporarily occluded with a bull-dog clamp. The renal vein is dissected out and occluded by clamps or threads and the splenic vein anastomosed by everting sutures to an opening in its upper surface. To prevent engorgement of the kidney a temporary clamp may be put on the renal artery, but it should be released after fifteen minutes to allow a fresh flow of blood to the kidney.

3. **Superior Mesenterico-caval Anastomosis.**—When the portal vein is thrombosed this alternative may be possible. The inferior vena cava is divided just above its lower end, and ligated below. The proximal end is joined to the side of the superior mesenteric vein in the root of the mesentery. This operation is particularly indicated in children, who can tolerate complete division of the inferior vena cava without developing œdema of the lower limbs. Alternatively a shunt of synthetic material (Dacron) or of endogenous vein can be interposed between the superior mesenteric

Alfred Blalock, 1899–1964. Surgeon-in-Chief, Johns Hopkins Hospital, Baltimore, U.S.A.

vein and the inferior vena cava. This mesentericocaval shunt (H-graft, 'jump' graft) is generally simpler to perform than the other operations, an advantage in emergency circumstances. It also can be carried out when the portal vein is occluded by thrombus.

4. **Distal Splenorenal Shunt.**—Distal splenic vein is joined to the renal vein without splenectomy. The extrinsic gastric and lower œsophageal veins are ligated except the short gastric veins draining the stomach into the splenic bed. The objective is to decompress the critical area of the lower end of the œsophagus, yet preserving a venous blood supply to the liver. The incidence of encephalopathy is said to be low. The operation presents technical difficulties in the obese patient and those with splenomegaly.

Other shunt operations are being evaluated, including left gastric venocaval shunt, umbilicocaval shunt and arterialisation of the portal vein with portacaval shunt.

5. **Œsophageal Varices Interruption.**—In an emergency or when the patient's liver function is inadequate for a porta-systemic shunt or there is no suitable patent vein, it is desirable to make an effort to interrupt the communications between the left gastric (portal system) and œsophageal veins (azygos system)—porto-azygos disconnection—so cutting off the flow of portal blood to the varices.

(*a*) **Œsophageal Transection** (Milnes Walker).—The aim of this operation is to cut off the flow of portal blood to the varices. Through a left trans-thoracic approach the lower end of the œsophagus is mobilised and the vagus nerves separated from it. The œsophagus is transected at the level of the hiatus in the diaphragm, and resutured with continuous sutures in the mucosa and submucosa which obliterate the vessels.

(*b*) **Suture of the Varices** after opening the lower part of the œsophagus longitudinally (Crile), just above the diaphragm. The varices, extending through the cardia are underrun with continuous chromic catgut.

(*c*) **Gastric Transection** (Tanner).—The aim is similar to that of œsophageal transection. Through a left thoraco-abdominal approach, the upper part of the stomach is transected and reanastomosed with continuous sutures to obliterate the blood vessels.

(*d*) **Œsophageal Stapling** (Johnston).—A simple way of interrupting dilated varices within the œsophageal wall is to pass a stapling instrument into the lower œsophagus through a small hole in the anterior wall of the stomach. When operated, the instrument simultaneously divides the œsophagus and joins it together again with staples which occlude the vessels. The results of this operation have still to be assessed but it has the merit of simplicity.

All of these operations may be helpful in emergency circumstances or when the splanchnic vessels are thrombosed, thus averting a shunt operation, but rebleeding is common.

6. **Splenectomy.**—This has no place in the treatment of portal hypertension except in the very rare cases of thrombosis confined to the splenic vein, or in combination with some other operation.

7. **Injection of Varices.**—Because of the high mortality of open operation, injection sclerotherapy is being used increasingly (Rodgers, Johnston). Varices can be injected once the bleeding is under control and the site of bleeding confirmed. A good view is necessary, 5–6 ml of 5% ethanolamine oleate is injected into each varix down the rigid 50 cm œsophagoscope using a long Macbeth needle. The lip of the œsophagoscope is rotated to compress the varix that has just been injected.

Injection sclerotherapy can also be performed using a fibre-optic œsophagoscope through which a flexible needle is threaded. When the œsophagoscope is in position, a sheath is passed over the instrument down into the œsophagus. The end of the sheath in the œsophagus is slotted so that varices will protrude into the lumen and therefore be easily injected. The injected varices are then compressed by rotating the sheath. If bleeding occurs after injection, a Sengstaken tube can be inserted, but this is only rarely required. Injections should be repeated at regular intervals until all varices have been obliterated.

Careful anæsthetic technique includes intermittent Althesin (alphaxalone and alphadalone acetate) and suxamethonium, and artificial ventilation with nitrous oxide and oxygen (Ward).

Robert Milnes Walker, Contemporary. Professor Emeritus of Surgery, Bristol, England.
George Crile, Jnr., Contemporary. Head of the Department of General Surgery, The Cleveland Clinic, Cleveland, Ohio, U.S.A.
Norman Cecil Tanner, Contemporary. Consulting Surgeon, Charing Cross Hospital, London.
George Johnston, Contemporary. Surgeon, Royal Victoria Hospital, Belfast.
Harold Rodgers, Contemporary. Formerly Professor of Surgery, Royal Victoria Hospital, Belfast.
Ronald Graeme Macbeth, Contemporary. Formerly Lecturer in Otolaryngology, Oxford.
Michael Elliott Ward, Contemporary. Anæsthetist, Nuffield Department of Anæsthetics, Radcliffe Infirmary, Oxford.

NEOPLASMS OF THE LIVER

Benign:

Hæmangioma occurs more commonly in the liver than in any other internal organ, and usually the neoplasm is of the cavernous type. As a rule, a hæmangioma of the liver is solitary, and is found either at necropsy or incidentally at operation. Exceptionally, it becomes large enough to cause symptoms, in which event laparotomy is required to establish the diagnosis. The compressibility of the tumour makes the diagnosis unmistakable, and on no account should biopsy be performed, for the resulting hæmorrhage is terrific. Radiotherapy is often useful in reducing the size of the tumour. Excision of tumour confined to a lobe necessitates hemi-hepatectomy (p. 907).

Hepato-adenomas are composed of hepatic cells. Frequently multiple and small, they are distinguished with difficulty from the nodular hyperplasia of cirrhosis. There is no evidence that they become malignant. Their removal is unnecessary.

Cholangio-adenomas originate from the bile-ducts and form small subcapsular masses. They are found at operation or necropsy, and can simulate closely metastatic deposits, which should not be assumed without biopsy. They are harmless.

Malignant:

Primary carcinoma of the liver is uncommon in European races, but is by no means rare in African natives, Malayans, Chinese, and Japanese. The highest recorded incidence is in Mozambique where it is a thousand times as common as in most of Europe. In the majority of these patients it arises as a complication of cirrhosis. A similar ratio is not found among the coloured and white North American peoples. Two pathological varieties are described:

1. **Hepatocarcinoma** arises in the liver cells. There are great variations in the malignancy of this tumour, which may be multicentric in origin, particularly when it occurs in a cirrhotic liver. At one end of the scale is a relatively benign, firm, slowly growing tumour (fig. 957), and at the other a rapidly growing, soft neoplasm prone to undergo necrosis, which soon metastasises within the liver, or to the lymph nodes in the hilum of the liver, mediastinum or neck.

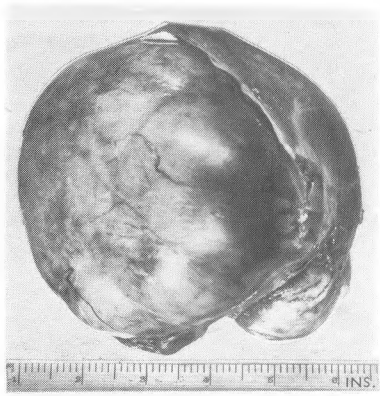


FIG. 957.—Hepatoma resected from the liver successfully.

2. **Cholangiocarcinoma.**—See p. 936.

Clinical Features of Primary Carcinoma of the Liver.—These tumours occur in children and in older people, but it is rare between the ages of 20 and 40 except in those countries where the incidence is high and then this is the age group most affected. The usual train of events is anorexia, rapid loss of weight, and asthenia. On examination an enlarged liver is found and often a localised mass. Less frequently the patient presents on account of a swelling he has discovered in the upper abdomen: the swelling may, or may not, be tender, depending on the rate of growth. Neoplastic proliferation is sometimes so rapid as to cause a low pyrexia, in which event, in tropical countries, an amœbic abscess will be considered

as a possible diagnosis. Every effort is made to seek a possible primary growth, bearing in mind that, at any rate in white races, secondary carcinoma of the liver is immeasurably more common than primary carcinoma. In patients with primary liver cancer, α-1-fetoprotein (AFP) may be present in the plasma long before clinical features appear. AFP is a normal component of the plasma protein of the human fœtus older than six weeks and disappears a few weeks after birth. It is a valuable screening method for patients with primary liver carcinoma with cirrhosis. Radio-scanning (p. 881) may be helpful and needle biopsy of the liver may establish the diagnosis. Additional diagnostic techniques include ultrasonic scanning and cœliac axis angiography.

Ascites is present in about 40 per cent. of patients at the time of the first examination; diagnostic aspiration often reveals blood-stained ascitic fluid. Such ascites resists therapy, and re-accumulates rapidly after paracentesis.

SECONDARY NEOPLASMS OF THE LIVER

Secondary Carcinoma.—As is well known, the liver is a favourite site for carcinomatous metastases. Characteristically, secondary growths in the liver, owing to degeneration of cells in the centre, are umbilicated. As a rule, carcinomatous deposits in the liver are multiple (figs. 958 and 959).

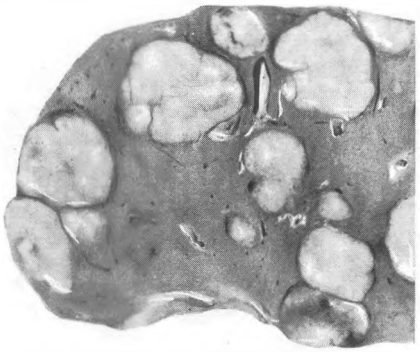


FIG. 958.—Secondary carcinoma of the liver.

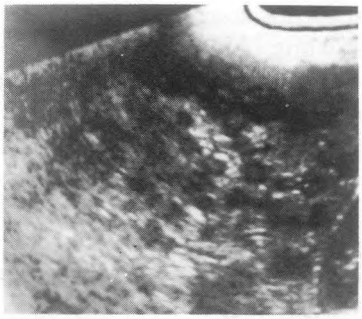


FIG. 959.—Multiple ' echo-free ' or ' transonic ' metastases in the liver from carcinoma of the breast. (*Dr. J. McIvor, London*).

Occasionally at laparotomy a secondary growth in the liver is found to be apparently solitary and accessible. In such circumstances, provided that the primary growth can be or has been resected, excision of that part of the liver containing the neoplasm sometimes results in a long-lasting survival.

Secondary melanoma occurs particularly when the primary growth is in the eye. Fifteen years or more have elapsed between the removal of the primary growth and the appearance of secondary deposits (p. 523).

Secondary carcinoid (argentaffin) tumours (p. 1016) are rare, where they occur they often grow to a large size. There are always many hepatic metastases when malignant carcinoid of the small intestine or bronchus is associated with vasomotor abnormalities and pulmonary stenosis. The increased urinary excretion of 5-hydroxy-indole-acetic acid is helpful in making a diagnosis.

Secondary Sarcoma.—The liver is not an uncommon site for secondary deposits in cases of sarcoma, but the lungs show a much higher incidence.

Treatment of Neoplasms of the Liver

Secondary tumours of the liver are most resistant to treatment. Ligation of the hepatic artery to deprive the tumours of blood-supply and infusion of chemotherapeutic agents are not very effective. Occasionally where a solitary secondary nodule is located in one lobe of the liver, hepatic lobectomy may be carried out. This procedure is not curative but may prolong life.

The management of *primary hepatic tumours* is depressing because untreated patients survive only 3–5 months after diagnosis. Radiotherapy and chemotherapy have been disappointing. Attempts have been made to improve results by ligating the hepatic artery, considered to be the major feeding vessel for such tumours, but there is no evidence that the results are improved. Hepatic transplantation has been carried out for liver tumour but the results in terms of long-term survival are disappointing. Where primary hepatoma is located to one or other lobe of the liver, and provided that the rest of the liver itself is normal and not cirrhotic, the affected lobe of the liver may be removed surgically.

Resection of a Benign Tumour of the Liver.—In order to conserve blood, the hepatic artery and portal vein can be clamped lightly at the porta hepatis, and released every five minutes. Another very important point is that branches of the hepatic artery and the portal vein ramify through the liver in the portal canals: each of these canals is surrounded by a prolongation of Glisson's capsule which, when encountered with a blunt instrument, gives a sense of resistance. Thus, if liver tissue is dissected with the finger, it is possible to divide liver tissue, but spare the vessels, which can be clamped and ligated, or coagulated with diathermy, before division. The resection completed, the resulting raw surfaces of the cut liver are dealt with in the same manner as described for rupture of the liver (p. 882).

Hemi-hepatectomy.—A number of cases of successful removal of the surgical right lobe of the liver for primary and secondary tumours or for carcinoma of the gall-bladder have been reported. Removal of the surgical left lobe is less frequently necessary and left (anatomical) lobectomy usually suffices (Rodney Smith).

Right Hemi-hepatectomy.—After operability has been determined through a right paramedian incision, extension into a right thoracoabdominal is achieved through the eighth interspace. The liver is mobilised and rotated forwards and upwards into the chest to expose the porta hepatis. Working from below upwards, the cystic artery and duct, the right hepatic duct, right hepatic artery, and right branch of the portal vein are successively divided between ligatures. The inferior vena cava is then exposed by mobilising the duodenum and head of pancreas and drawing them over to the left (Kocher's manœuvre). It is then possible to expose, ligate, and divide the inconstant minor hepatic veins running from the right lobe into the cava, up as high as the site of entry of the right hepatic vein; great care must be taken not to obstruct the venous return in the vena cava. The liver is then divided from in front and above between the surgical lobes by diathermy. The right hepatic vein is then exposed and divided and also the right-sided branches of the middle hepatic vein as they are encountered. The raw surface of the left lobe is covered with the falciform ligament and it is wise to drain the common bile-duct.

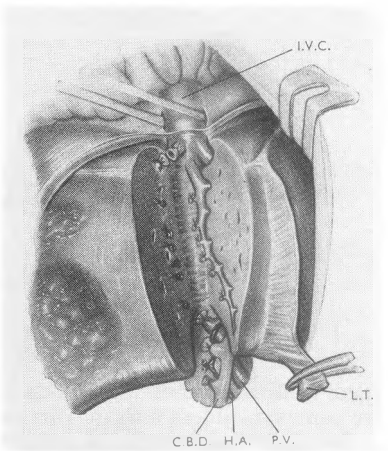


FIG. 960.—Right hemi-hepatectomy almost completed; the hepatic veins remain to be ligated (diagrammatic). I.V.C. = Inferior Vena Cava, C.B.D. = Common Bile Duct, P.V. = Portal Vein, H.A. = Hepatic Artery, L.T. = Ligamentum Teres. (*After C. Rob and R. Smith.*)

Liver Transplantation is dealt with in Chapter 61.

Francis Glisson, 1598–1677. Regius Professor of Physic, Cambridge, England.
Rodney Smith (Lord Smith of Marlow), Contemporary. Formerly Surgeon, St. George's Hospital, London.
Theodor Kocher, 1841–1917. Professor of Clinical Surgery, University of Berne, Switzerland.

CHAPTER 46

THE GALL-BLADDER AND BILE DUCTS

SURGICAL ANATOMY AND PHYSIOLOGY

The gall-bladder is pear shaped, 7·5 to 12·5 cm long, with a capacity of about 50 ml, but capable of fifty-fold distension. The anatomical subdivisions are, a fundus, a body, and a neck which terminates in the narrow infundibulum. The angulated distal part of the neck forms a pouch, called Hartmann's pouch, a common site for a solitary gall-stone to lodge. The muscle fibres in the wall of the gall-bladder are arranged in a criss-cross manner, being particularly well developed in the neck. The mucous membrane contains indentations of the mucosa that sink into the muscle coat; these are the crypts of Luschka.

The cystic duct is about 2·5 cm in length. Internal projections of circular muscle fibres account for the spiral valve of Heister, which makes the passage of calculi difficult.

The common hepatic duct is usually less than 2·5 cm long and is formed by the union of the right and left hepatic ducts.

The common bile duct, about 7·5 cm long, is formed by the junction of the cystic and the common hepatic ducts. It is divided into four parts: 1. *The supra-duodenal portion*, about 2·5 cm long, runs in the free edge of the lesser omentum. 2. *The retroduodenal portion.* 3. *The infraduodenal portion* lying in a groove, but at times in a tunnel in the posterior surface of the pancreas. 4. *The intraduodenal portion* passes obliquely through the wall of the second part of the duodenum and is surrounded by the spincter of Oddi and then forms an ampulla (Vater) before terminating with an

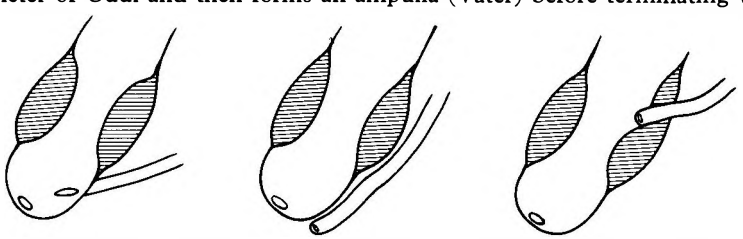


FIG. 961.—The terminal arrangement of the common bile duct. The shaded area represents the sphincter of Oddi.

opening on the summit of the duodenal papilla. The arrangements of this exit, with the pancreatic duct (Wirsung) are depicted in fig. 961.

The Arterial Supply of the Gall-bladder.—The cystic artery, a branch of the right hepatic artery is usually given off *behind* the common hepatic duct (fig. 962(*a*). Thus it makes the base of Calot's triangle, the other sides being the right hepatic artery above, and the cystic duct below. Occasionally an accessory cystic artery arises from the gastro-duodenal artery. In 15% of cases the right hepatic artery and/or the cystic artery cross in front of the common bile duct and the cystic duct (fig. 962(*b*)). The most dangerous anomaly of all is when the hepatic artery takes a tortuous course in front of the origin of the cystic duct or the right hepatic artery is tortuous, and the cystic artery is short. The tortuosity is known as the 'caterpillar turn' or Moynihan's 'hump' (fig. 962(*c*)). There is a possibility of liver infarction in accidental ligation of the common hepatic artery, but chances of recovery in this and right hepatic ligation are improved by giving neomycin or ampicillin by mouth, and ensuring a high intake of glucose. Hyperbaric oxygen (p. 85) would be useful, if available. Infarction in a jaundiced patient is probably lethal.

Henri Albert Charles Antoine Hartmann, 1860–1952. Professor of Surgery, Paris.
Hubert Luschka, 1820–1875. Professor of Anatomy, Tubingen, Germany.
Lorenz Heister, 1683–1758. Professor of Surgery and Botany, Helmstadt, Germany.
Ruggero Oddi, 1845–1906. Physiologist, Perugia, Italy.
Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg, Germany.
Johann Georg Wirsung, d. 1643. Professor of Anatomy, Padua, Italy. Murdered when entering his house at night.
Jean François Calot, 1861–1944. Surgeon, Paris.
Berkeley George Andrew Moynihan (Lord Moynihan), 1865–1936. Professor of Clinical Surgery, Leeds, England.

Lymphatics.—The lymph vessels of the gall-bladder (subserosal and submucous), drain into the *cystic lymph node of Lund* (the sentinel lymph node), which lies in the fork created by the junction of the cystic and common bile ducts. Efferent vesseles

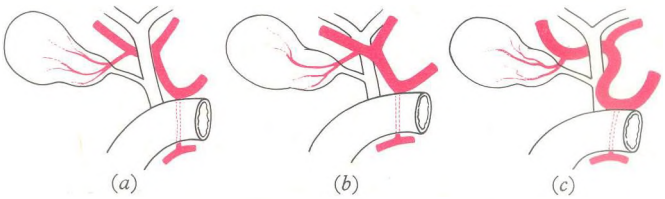


FIG. 962.—Arrangements of the arterial supply to the gall-bladder.

from this lymph node go to the hilum of the liver, and to the cœliac lymph nodes. The subserosal lymphatic vessels of the gall-bladder also connect with the subcapsular lymph channels of the liver, and this accounts for the frequent spread of carcinoma of the gall-bladder to the right lobe of the liver (p. 936).

Embryology (See p. 912).

Surgical Physiology

Bile, as it leaves the liver, is composed of 97 per cent. water, 1 to 2 per cent. of bile salts, and 1 per cent. of pigments, cholesterol, and fatty acids. The liver excretes bile at a constant rate, estimated at 40 ml. per hour. Except when active gastro-duodenal digestion is in progress, the sphincter of Oddi contracts and consequently bile leaving the liver passes into the gall-bladder.

The healthy gall-bladder has several functions:

1. **Concentration of Bile.**—By the active absorption of water, sodium, chloride, and bicarbonate by the mucous membrane of the gall-bladder into the bloodstream and to a lesser extent into the lymphatics, the hepatic bile which entered the gall-bladder becomes concentrated from 5 to 10 times, with a corresponding increase in the proportion of bile salts, bile pigments, cholesterol, and calcium it contains.

2. **Reservoir for Bile.**—Bile is stored until the gall-bladder is full, when the sphincter of Oddi partially relaxes and the overflow trickles into the duodenum. Contraction of the gall-bladder and synchronous relaxation of the sphincter of Oddi is stimulated by *cholecystokinin*, a hormone secreted by the duodenum in response to acid chyme, fats and egg yolk or magnesium sulphate in its lumen.

3. **Changing the Reaction of the Bile.**—The bile excreted by the liver is alkaline (pH 8·2), whereas that in the gall-bladder becomes acid (pH 7·6 to 7·0) as the bile becomes concentrated.

4. **Cholesterol excretion** by the gall-bladder has not been proved, but pathological evidence of the gall-bladder epithelium choked with crystalline cholesterol (p. 924) is suggestive that the gall-bladder may add cholesterol to the bile.

5. **Secretion of Mucin.**—About 20 ml. is secreted each twenty-four hours.

RADIOLOGICAL INVESTIGATIONS

Plain X-ray will show radio-opaque gall-stones in 10% of cases (fig. 963). It will also show the rare cases of calcification of the gall-bladder (porcelain gall-bladder, fig. 965, and limey (lime water) bile, fig. 966).

Oral cholecystography (Graham-Cole test) remains the most satisfactory method of visualising the gall-bladder (figs. 967 (*a*) and (*b*) and 968):

Telepaque (Bayer Products Ltd.) is the contrast medium employed most commonly in the British Commonwealth and North America, but Biloptin and Solubiloptin Schering (Germany) and Orabilix (France) are also used. At about 18.00 hrs on the day before the X-ray the patient has a meal, preferably containing some fat (to empty the gall bladder). The medium is taken by mouth, with water, at 21.00 hrs. Nothing to eat or drink is taken thereafter, the patient being X-rayed in the erect and supine position at 09.00 hrs the next day. A fatty meal or drink is then given to

Fred Bates Lund, 1865–1950. Surgeon, Boston City Hospital, Boston, Mass., U.S.A.
Evarts Ambrose Graham, 1883–1957. Bixby Professor of Surgery, Washington University, St. Louis, Missouri, U.S.A.
Warren Henry Cole, Contemporary. Emeritus Professor of Surgery, University of Illinois, Chicago, U.S.A.

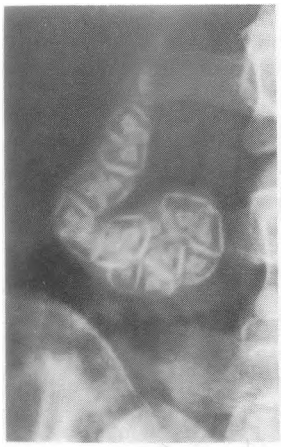


FIG. 963.—Plain radiograph showing radiopaque stones in the gall-bladder.

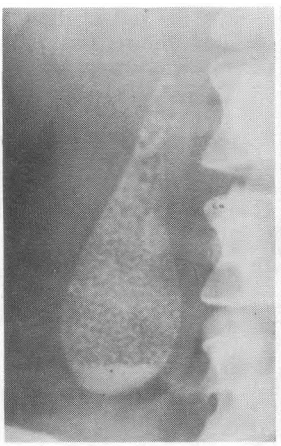


FIG. 964.—Non-opaque stones rendered visible by oral cholecystography.

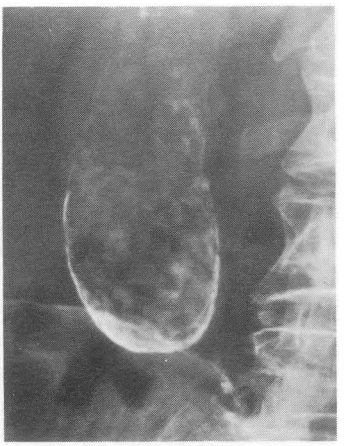


FIG. 965.—Porcelain gall-bladder.

stimulate gall bladder contraction and another X-ray is taken (fig. 967(*b*)). A double dose of contrast medium is frequently used to demonstrate the common duct and gall-bladder.

Intravenous cholangiography (Biligrafin[1]) permits radiological visualisation of the intra- and extrahepatic bile ducts (fig. 968). It may be used in conjunction with oral cholecystography. Tomography[2] is helpful in delineating stones in the ducts.

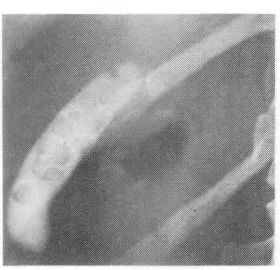
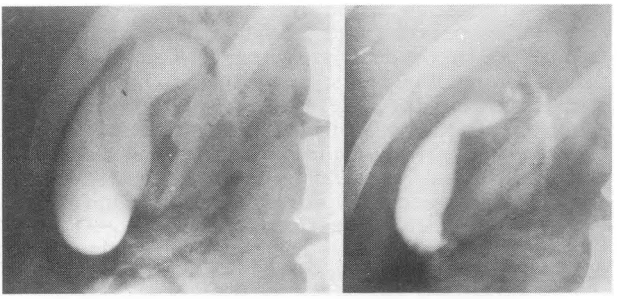


FIG. 966.—*Plain radiograph* showing a gall-bladder filled with limey bile. Gall-stones are present also.



(*a*) (*b*)

FIG. 967.—(*a*) A normal cholecystogram. (*b*) Same after a fatty meal.

Safety Precautions and Contraindications.—Substances containing iodine can give rise to severe reactions (imperceptible pulse, stertorous respirations, cyanosis). A test dose is usually given first, from a supplementary small ampoule which is supplied with each package by the manufacturers. The contents of this are injected into a vein the day before (or, in urgent cases, an hour before) the examination, in order to ascertain if any reaction is likely to occur. The treatment is to administer niketha-mide, antihistamine drugs, and oxygen.

Impaired renal function, and macroglobulinæmia are contraindications.

These investigations are not used if the patient is jaundiced with a plasma bilirubin level of over 3 mg/100 ml (51 μmol/l), for the poorly functioning hepatocytes do not excrete the medium in sufficient concentration to be shown up by X-ray. Also acute cholecystitis rather precludes their use.

[1] Iodipamide, known in the U.S.A. as cholografin.

[2] Tomography = a method of placing one given plane into sharp focus while blurring others.

Ultrasound Diagnosis, by considerable technical innovation, has given a new dimension to the diagnosis of cholelithiasis, especially in jaundiced patients where the conventional methods (above) are of no avail (figs. 969–972).

Per-operative Cholangiography (figs. 973, 974 and 975).—A cassette tunnel is placed beneath the patient (fig. 976) at a level that will include the entire biliary tract. The cystic duct is opened and a fine polythene catheter is passed through into the common bile duct for 3 cm. A ligature tied round the cystic duct and catheter prevents leakage. The catheter is filled with normal saline prior to insertion (bile should be aspirated after insertion), so that there are no air bubbles present which would have a similar X-ray appearance to a radiolucent gall-stone. Instruments and packs which might obscure the X-ray are removed, and the table is tilted 10° to the right side so that the spine and ducts are not superimposed. Two injections (3 ml and 10 ml) are given of 30% Hypaque or Urografin (sodium diatrizoate or diatrizoate salts). An X-ray exposure follows each

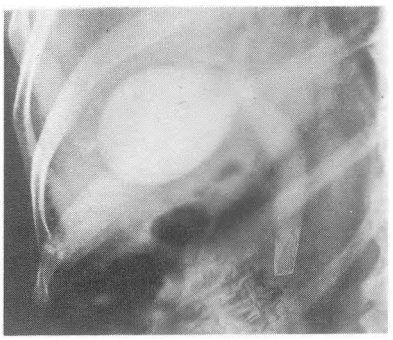


FIG. 968.—Simultaneous oral and intravenous cholecystography and cholangiography revealing a dilated gall-bladder, a distended common bile duct, and a ' cut off ' at the lower end of the duct due to a stone.

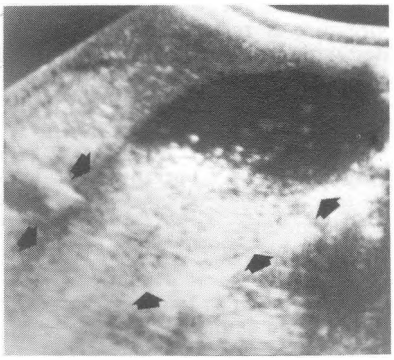


FIG. 969.—Multiple small gall stones filling part of the body and infundibulum of a large gall-bladder.

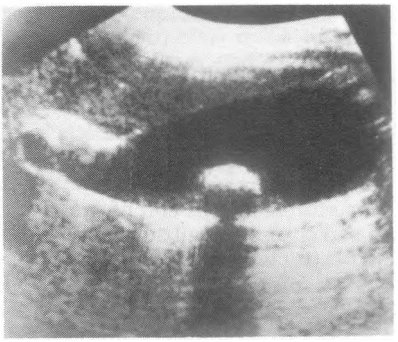


FIG. 970.—Single large gall stone casting an ' acoustic shadow '.

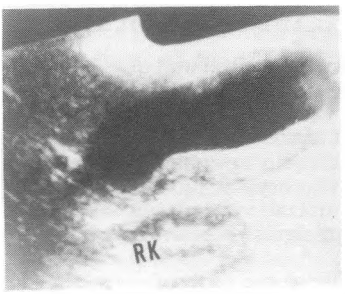


FIG. 971.—Mucocele of gall-bladder which is now larger than the right kidney (RK).

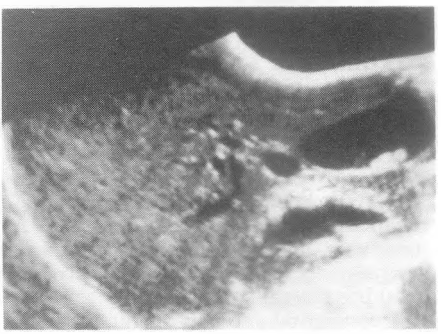


FIG. 972.—Dilatation of the intrahepatic and common bile ducts due to a calculus impacted at the sphincter of Oddi. There are calculi visible in the gall-bladder.

(Figs. 969–972 courtesy of James McIvor, F.D.S., F.R.C.R., London.)

injection; respiration being interrupted to obviate any movement which would blur the picture. An X-ray image intensifier with T.V. display (fig. 976) is a great asset as the operator can dispense with the two blind exposures, and is able to take the X-ray picture when he knows he has given a satisfactory injection. A normal cholangiogram is sufficient evidence that exploration of the common bile duct is unnecessary. If the medium, even after injection of a greater volume, fails to pass into the duodenum, and especially if the ducts concerned are dilated or if a filling defect is visualised, choledochotomy and exploration of the interior of the common bile duct is the first consideration. In about 20% of cases the medium flows along the pancreatic duct of Wirsung. This is not necessary pathological.

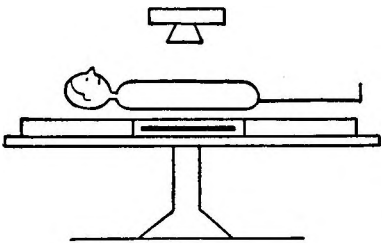


FIG. 973.—Per-operative cholangiography using a radiolucent table-top.

Radiomanometry.—The resistance to the flow of bile through the biliary sphincter in the intraduodenal part of the common duct and through the papilla can be displayed and measured. Under X-ray image intensifier with T.V. control (figs. 976 and 977), a graduated burette containing Hypaque is raised slowly against a scale until the contrast is seen to flow into the duodenum. The height of the meniscus in the burette at this point gives the yield pressure, normally up to 20 cm contrast. Alternatively, a constant flow infusion technique is employed and the pressure is recorded. High yield pressures tend to be associated with stones in the ducts, pancreatitis, a thickened or fibrosed papilla (papillitis) or a carcinoma. Occasionally an increased resistance may be due to an underlying malfunction of the sphincter and the duodenum (so-called dyskinesia[1]).

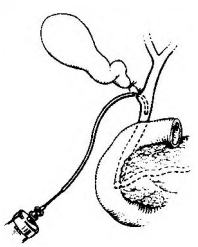


FIG. 974.—Per-operative cholangiography. Method of introducing the medium.

Per-operative Post-exploratory Cholangiography.—A cholangiogram via a catheter or the T-drainage tube (p. 931) may be performed at the end of the operation, after choledochotomy, to make sure that all stones have been removed, and that there is no obstruction to the flow of bile into the duodenum.

Post-operative Cholangiography is performed some ten to fourteen days after choledochotomy via the T-tube. The absence of stones and a normal flow of bile into the duodenum indicates that the T-tube can be removed (p. 931).

Endoscopic Retrograde Cholangio-Pancreatography (**ERCP**) is described on pages 933 and 934.

Transhepatic cholangiography is described on p. 933.

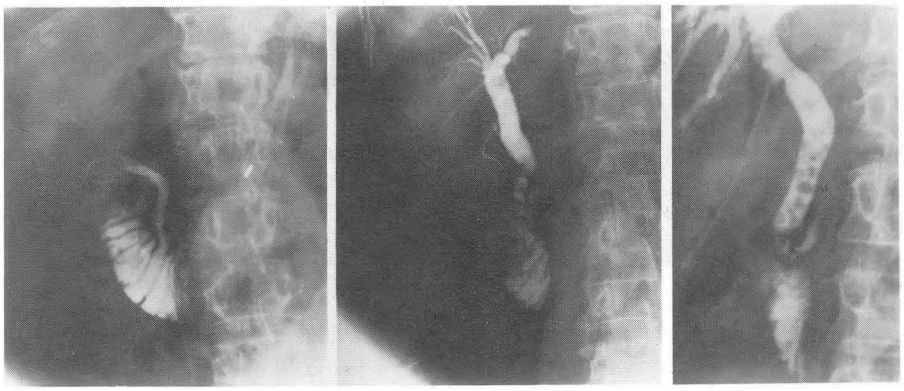
CONGENITAL ABNORMALITIES OF THE GALL-BLADDER AND BILE DUCTS

Embryology.—The hepatic diverticulum arises from the ventral wall of the foregut and elongates into a stalk to form the choledochus. A lateral bud is given off, viz.——————→ which is destined to become the gall-bladder and cystic duct. The embryonic hepatic duct sends out many branches which join up with the canaliculi attached to the liver cells. As is usual with embryonic tubular structures, hyperplasia obliterates the lumina of this ductal system, but normally recanalisation occurs subsequently and bile begins to flow. During early fœtal life the gall-bladder is entirely intrahepatic.

A congenital anomaly of the biliary tract is found in 10 per cent. of necropsies.

Absence of the Gall-bladder.—Occasionally the gall-bladder is absent in man. Stones have been found in the common bile duct in nearly 50 per cent. of cases.

[1] Dyskinesia = impairment of movement.



(*a*) (*b*) (*c*)

FIG. 975.—Per-operative cholangiography (choledochography). (*a*) Gentle infusion of contrast, passing without hindrance into the duodenum. A normal duct with no problems. (*b*) The duct is dilated and there is a slight hold-up of contrast. No stones and no real indication to operate on the sphincter of Oddi (see Sphincterotomy). (*c*) All the ducts are dilated and contain many stones. There is narrowing of the lower end of the duct with reflux of contrast into the pancreatic duct. Sphincterotomy was performed.

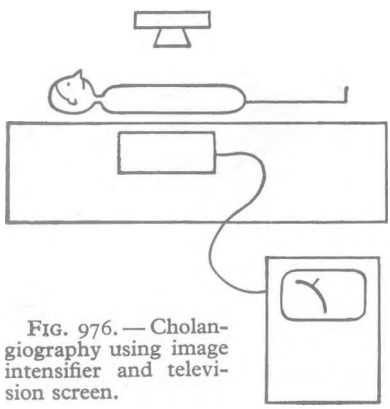


FIG. 976. — Cholangiography using image intensifier and television screen.

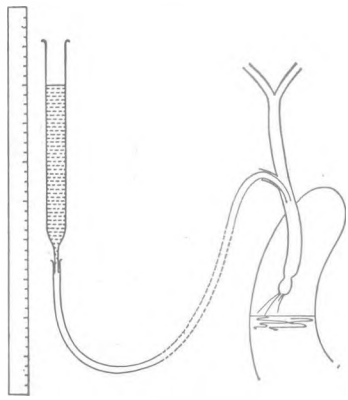


FIG. 977.—Radiomanometry.

The Phrygian cap[1] (fig. 978(*a*)) is present in 2 to 6 per cent. of cholecystographies, and may be mistaken for a pathological deformity of the organ.

Floating Gall-bladder.—The organ hangs on a mesentery which makes it liable to undergo torsion (p. 916).

Double Gall-bladder.—Rarely (fig. 978(*b*)), the gall-bladder is double. One of the twins may be intrahepatic. Cholecystography often fails to be diagnostic, because the shadows of the two gall-bladders are superimposed. Double cholecystectomy should be performed.

Absence of the Cystic Duct (fig. 978(*c*)).—Injury of the common duct is liable to occur when cholecystectomy is performed in a patient with this abnormality. Meticulous dissection is required and closure of the opening in the neck of the gall-bladder with a continuous catgut suture.

Low Insertion of Cystic Duct (fig. 978(*d*)).—The cystic duct opens into the common duct near the ampulla. This common anomaly must be sought for par-

[1] The Phrygian cap refers to hats worn by peoples of Phrygia, an ancient country of Asia Minor. The cap was rather like a liberté cap of the French Revolution.

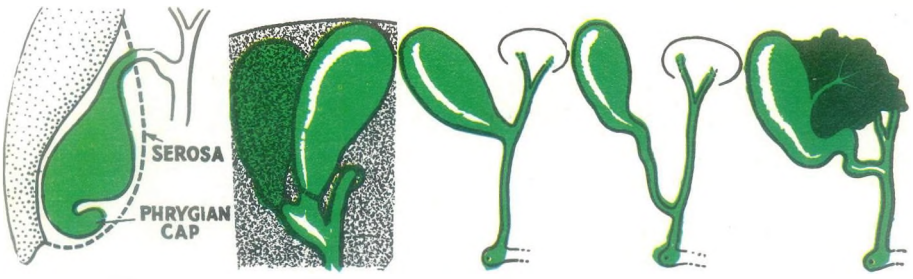


FIG. 978.—Some anatomical anomalies of the gall-bladder and bile passages.

ticularly when considering a bypass operation for jaundice due to carcinoma of the duct or the pancreas (p. 954).

An Accessory Cholecystohepatic Duct (fig. 978(*e*)) may open into the gall-bladder and cause leakage after cholecystectomy (p. 926). In the very rare anomaly where the right hepatic duct terminates in the gall-bladder, cholecystostomy or partial cholecystectomy should be performed.

CONGENITAL ATRESIA OF THE BILE DUCTS

Ætiopathology.—The probable explanation of many of the varieties is that recanalisation fails to occur in fœtal life (see above). One of the anomalies shown in fig. 979 is present. The extrahepatic bile ducts may have no lumen, or a tiny lumen blocked with epithelial debris, and perhaps biliary sand. In (*c*), a few have lumina connected with scattered bile-filled cysts. The liver, which is greatly en-

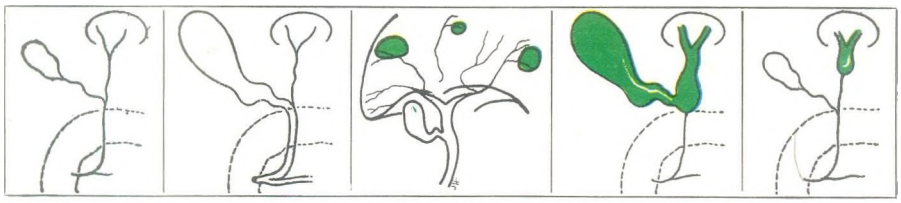


FIG. 979.—Types of congenital obstruction of the bile ducts.

larged and stained dark green, exhibits varying degrees of biliary cirrhosis. Surprisingly, a few infants have lived for several months.

Clinical Features.—Sometimes a slight icteric tinge is present at birth; more usually jaundice appears within two or three days thereof; occasionally it is delayed for up to several weeks. The urine is bile-laden and dark brown. The first meconium is often normal, i.e. greenish-black in colour; it is said to be formed during the fourth month of fœtal life. Thereafter the stools are white or clay-coloured. Gradually the liver enlarges, and on palpation feels unduly hard. Unrelieved, death usually results in three to six months.

Differential Diagnosis.—The jaundice could also be due to (*a*) erythroblastosis fœtalis (p. 894); (*b*) hereditary spherocytosis (p. 867); (*c*) jaundice of hæmolytic sepsis; (*d*) congenital syphilis; (*e*) homologous serum hepatitis (serum jaundice), if the child has had blood-transfusion or plasma infusion, and (*f*) neonatal hepatitis. The last, which is now more recognised, is the most diagnostically difficult to differentiate from atresia. In neonatal hepatitis the baby is likely to be more ill than is the case in atresia; also the stools are not without pigment. Liver-function tests need to be supported by the findings of a liver biopsy.

Treatment.—Pre-operative measures include the correction of dehydration, if present, and one or two small blood-transfusions. Vitamin K_1 is always necessary to combat prothrombin deficiency in patients with obstructive jaundice. Ampicillin should be administered before and after operation.

Operation.—The anæsthetic of choice is open ether and oxygen. Muscle relaxants must be used with caution. A midline incision affords adequate exposure, though the enlarged liver impedes dissection of the minute ducts.

Operative cholangiography (figs. 973–975) may show the site of the stricture. Even when the ducts seem to be fibrous cords it is worth finding out if a tiny lumen is present. After introducing a needle into the gall-bladder, or a duct, syringing sometimes has proved successful in clearing the ducts of inspissated material. With this exception, the operative measure should be as follows:

Gall-bladder present. Cystic and hepatic ducts distended. Atresia lower end of common bile duct.	Cholecystojejunostomy + jejuno-jejunostomy (fig. 1054).
Gall-bladder functionless or absent. Atresia lower end of common bile duct.	Choledochoduodenostomy (fig. 1001).
Common hepatic duct alone present, and contains bile.	Hepaticodochojejunostomy (Roux-en-Y).

If the atresia is of types (*a*) and (*b*) (fig. 979) it is conceded generally that the case is hopeless. In (*c*) it may be possible to unite the gall-bladder to a distended intra-hepatic duct by intubation and later, possibly, by a conduit made from jejunum.

CHOLEDOCHUS CYST

Congenital choledochus cyst is due to a specific weakness in a part, or the whole, of the wall of the common bile duct, and is classified accordingly as cystic dilatation, diverticulum, and choledochocele. Contrary to expectation, only occasionally does the distension involve the cystic and hepatic ducts and the gall-bladder is never distended, viz.:——————→ This is a rare condition affecting females four times as commonly as males, Japanese being more prone to the condition than other races. The symptoms and signs seldom become manifest before the age of six months (only half the cases present before the age of twenty). The cyst may contain as much as 1 to 2 litres. The cyst wall shows inflammatory changes and an absence of lining epithelium.

Clinical Features.—There are attacks of jaundice of the obstructive type, which

FIG. 980.—Choledochal cyst outlined by intravenous cholangiography. (*W. O. Kirwan, F.R.C.S., Edinburgh.*)

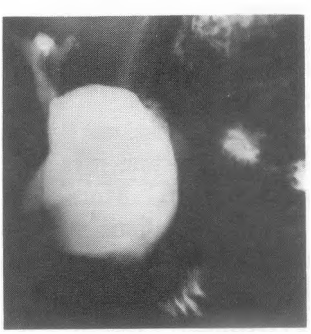


FIG. 981.—Choledochal cyst outlined by operative cholangiography.

are usually accompanied by upper abdominal pain and pyrexia due to infection. In most cases a swelling is detected in the upper abdomen. It is unusual for the cyst to be delineated by contrast X-rays (fig. 980). Untreated, the condition ultimately

César Roux, 1857–1934. Professor of Surgery and Gynæcology, Lausanne, Switzerland.

proves fatal, due to ascending cholangitis, biliary cirrhosis, or diffuse peritonitis following rupture of the cyst.

Treatment.—For cystic dilatation, choledochocystojejunostomy, and for diverticulum and choledochocele excision of the cyst, is recommended.

INJURIES

1. **Traumatic rupture of the gall-bladder or the bile passages** is a rare injury usually the result of a run-over accident. The physical signs are identical with those of rupture of the small intestine, with one notable addition—unmistakable jaundice, which appears within two or three hours of the accident in 65 per cent. of cases. When the abdomen is opened, bile is found within the peritoneal cavity.

Treatment of the various lesions is depicted in fig. 982, but when there is a small hole in the gall-bladder (such as can occur from a penetrating wound), suture of the rent with drainage of the peritoneal cavity is indicated.

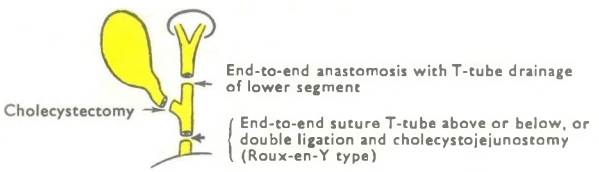

End-to-end anastomosis with T-tube drainage of lower segment
Cholecystectomy
End-to-end suture T-tube above or below, or double ligation and cholecystojejunostomy (Roux-en-Y type)

FIG. 982.—Methods of treating early complete tears of the biliary tract.

2. **Injuries of the Bile Ducts incurred during Operation** are discussed on p. 932.

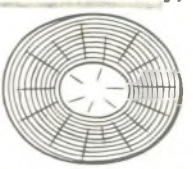
TORSION OF THE GALL-BLADDER

Torsion can occur in those rare instances where the gall-bladder has a comparatively long mesentery—the so-called 'floating' gall-bladder. There is a sudden agonising pain, with vomiting and shock. If the gall-bladder becomes gangrenous or ruptures, the pain passes off instantaneously, but signs of diffuse peritonitis soon follow. Cholecystectomy is the treatment.

GALL-STONES (Cholelithiasis) (fig. 983)

Gall-stones vary in composition. Sometimes they consist entirely of cholesterol, or of calcium bilirubinate. More often they are composed of alternating layers of cholesterol and calcium bilirubinate, or cholesterol and calcium carbonate and phosphate. Protein is another constituent.

Types of Gall-stone. (1) **A cholesterol stone** is usually solitary (the cholesterol 'solitaire'). It is oval or rounded in shape, up to 1·25 cm in diameter, or larger (fig. 983). It is light in weight as well as in colour. In its absolutely pure state it is very pale yellow in colour and, like tallow, it is feebly translucent. More often bile pigments are deposited within it. On section it has a radiating crystalline interior. Cholesterol stones, which make up some 6 per cent. of all gall-stones, are believed to be formed primarily in aseptic static bile (in a 'stasis' gall-bladder). They tend to reside in Hartmann's pouch. A brood of mixed (secondary or inflammatory) gall-stones (see below) may be formed later, or a mixed covering of pigment and cholesterol may form around the primary cholesterol stone, viz. ——————→ This is known as a combination stone.



(2) **Pigment stones** (calcium bilirubinate) comprise about 12 per cent. of the total. Black in colour, they range in shape and

Robert Hartmann, 1831–1893. Professor of Anatomy, Berlin.

size from irregular, mulberry or coral-like iridescent concretions of about 5 mm down to sludge particles. Often they are simply soft putty-like masses. On section all pigment stones seem to be amorphous.

(3) **'Mixed' stones** constitute the majority (80 per cent.) of gall-stones

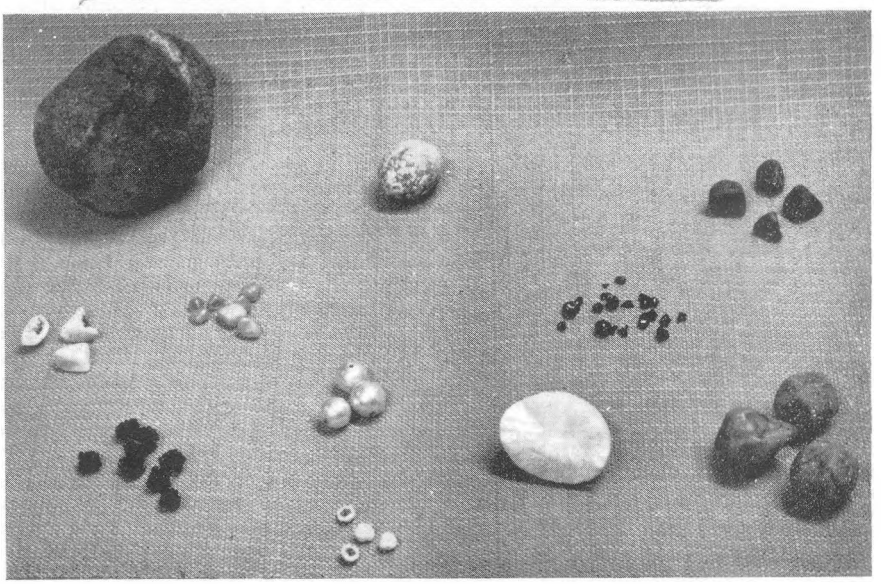


FIG. 983.—Gall-stones (see text). Note, however, the three pearls, usually formed of calcium carbonate in the oyster around a parasite or a grain of sand.

found at operation. Nearly always multiple, they become faceted by mutual pressure or by friction one against the other. Dozens or hundreds of such stones are often present, frequently the gall-bladder being packed to capacity. On section each is found to be laminated——→ The central nucleus may contain epithelial debris and bacteria, which suggests that the cause in that particular case is inflammatory. Whatever the composition of the central core, alternating layers of cholesterol and calcium carbonate and/or calcium bilirubinate are deposited upon it.

Gas in Gall-stones.—Rarely, the centre of a stone may contain radiolucent gas in a tri-radiate or bi-radiate fissure and this gives rise to characteristic dark shapes on X-ray—the *Mercedes Benz* or *Seagull* signs viz. ——————→

Limey bile.

'Lime-water' bile is revealed in a plain radiograph (fig. 966) more clearly than if the gall-bladder has been visualised by cholecystography. The opacity is the result of the gall-bladder becoming filled with a mixture of calcium carbonate and calcium phospate, usually the consistency of toothpaste. The condition tends to occur when there is a gradual obstruction of the cystic or common bile duct (e.g. chronic pancreatitis or carcinoma of the pancreas). Organisms are rarely grown from the emulsion.

Incidence of Gall-stones.—A Fat, Fertile, Flatulent Female of Fifty is the classical sufferer from gall-stones.[1] Useful as is this clinical memoran-

[1] It is a sign of the times, and a signpost to causation that gall-stones are being seen increasingly in Postpartum Primipara who were Prepregnancy 'Pill' takers (contraceptive pill).

dum, it should be tempered with the knowledge that cholelithiasis occurs in both sexes, quite often at a much earlier age—even in childhood—and is more common in old age. Stones occur in 20 per cent. of women during the child-bearing period and in 20 per cent. of both sexes in old age.

Causal Factors in Gall-stone Formation

Certain factors are likely to be responsible for the formation of gall-stones. These are: (1) Metabolic; (2) Infective; (3) Bile stasis.

(1) *The metabolic factors* are concerned with the solubility of cholesterol in bile acids. The bile acids are produced in the liver from cholesterol itself. They have an affinity for the mother substance (cholesterol) and other fats, and also for water (i.e. they are ampl[illegible]hic). This physico-chemical phenomenon is due to the aggregation of their molecules into *micelles* (fig. 984), along with phospholipids, which causes them to behave in the duodenum rather like modern fat-dissolving detergents used in the kitchen sink. The normal ratio of bile acid to cholesterol is 25:1. The critical level for precipitation of cholesterol is 13:1. The amount of bile acid in the body is called the 'bile acid pool'. Reduction of all or one of the bile acid constituents of the pool may be related to dietary factors, e.g. excessive sucrose intake, liver damage, or interference with the normal enterohepatic circulation in cases of Crohn's disease (p. 1010), after ileal resection, and also after gastrectomy. Locally, gall-bladder stasis or infection reduces the bile salt content. Such a bile is 'lithogenic' and cholesterol is likely to be thrown out of solution.

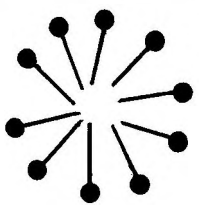


FIG. 984.—Diagram representing a spherical micelle formed by aggregation of bile-acid molecules. The rounded end of each molecule is water-loving (hydrophilic), whilst the stick-end is the hydrophobic part to which the water-insoluble cholesterol is adsorbed.

Bile pigment stones commonly follow the *excessive haemolysis* that occurs in types of hæmolytic anæmia, e.g. acholuric jaundice, septic hæmolysis and malaria.

(2) *Infection.*—Organisms are frequently found in gall-stones (streptococci, coliforms, typhoid, actinomyces), and have long been blamed as a cause. They reach the gall-bladder via the blood-stream, from a focus of infection elsewhere (tonsils, bowel, dental tartar, and caries) or from the bowel via the lymphatics, and cause an exudation from, and an exfoliation of, the mucosal cells to act as a nidus on which the stone forms. There is some truth in Moynihan's aphorism that 'a gall-stone is a tombstone erected to the memory of the organism within it'. *Infestation* with a round worm (Ascaris) is a cause of stones being formed around ova in the common bile duct. Other foreign bodies like rolled-up plum or tomato skins have been found in the centres of stones. In one case, a lady swallowed a few pins (deliberately) and one turned up in a gall-bladder stone 5 years later (Harman).

(3) *Bile Stasis.*—Without some kind of stasis, stones would not grow to the sizes commonly found. They would be voided while still in a particulate or gravel form. Stasis is usual during pregnancy and this, rather than hypercholesteræmia, may explain the increased incidence of stones in multipara. Also in the gall-bladder, the incoming bile tends to form layers owing to differences in specific gravity (fig. 964). At the interface between layers, the solubility of cholesterol is disturbed.

GALL-STONES IN RELATION TO OTHER DISORDERS

Saint's Triad.—(i) Gall-stones, (ii) Diverticulosis of the colon, and (iii) Hiatus hernia frequently coexist. Possibly obesity or deranged gastrointestinal motility is the common factor. It is important to find out which lesion is the cause of the patient's dyspeptic symptoms.

The Cardiac Link.—When the heart is the seat of a proclaimed or a latent disorder, vagal reflexes from a diseased gall-bladder may bring about decreased coronary blood

Michael John Harman, Contemporary. Radiologist, Essex County Hospital, Colchester, Essex, England.
Charles Frederick Morris Saint, 1886–1973. Emeritus Professor of Surgery, Cape Town, South Africa.

flow, arrhythmia or heart-block; the 'cholecystic heart'. Electrocardiographic abnormalities without other evidence of cardiac disease often revert to normal following removal of a diseased gall-bladder. Likewise, patients with angina pectoris, and pseudo-angina have benefited. These sufferers should be advised to undergo cholecystectomy, not only because of possible improvement of the cardiac condition, but to avoid an operation at a later date, when circumstances may be less favourable.

Previous gastric surgery and *Crohn's disease* have been mentioned in causal factors (above).

THE EFFECTS AND COMPLICATIONS OF GALL-STONES

Gall-stones are usually found in the gall-bladder, but in 20 per cent. of cases stones will also be present in the bile ducts. Nearly always it is just one stone amongst many which is responsible for the patient's suffering. The effects and complications of gall-stones can be summarised as follows:

1. **In the gall-bladder**—Silent stones.
   —Flatulent Dyspepsia.
   —Gall-stone Colic.
   —Mucocele.
   —Acute cholecystitis { perforation→peritonitis. / gangrene. / penetration→fistula. / empyema.
   —Chronic cholecystitis→acute cholecystitis.
   —Carcinoma.
2. **In the bile ducts** —Obstructive jaundice. Liver failure. White bile.
   —Acute or recurrent pancreatitis.
   —Cholangitis.
3. **In the intestine** —Acute intestinal obstruction (p. 1031).

Silent Gall-stones.—It is possible for a calculus or calculi to be present in the gall-bladder and to give rise to no symptoms during a long lifetime. At least 8 per cent. of necropsy subjects of over fifty years of age have gall-stones in the gall-bladder that have not contributed to the cause of death. As 10 per cent. of gall-stones are radiopaque, they may be discovered accidentally on X-ray for another condition, e.g. by an X-ray to determine whether a rib is fractured. Cholecystectomy should be advised on account of the likelihood of complications, particularly when a cholecystogram reveals a non-functioning gall-bladder, or the stones are large and obviously fill a gall-bladder devoid of function. A follow-up of 112 patients with symptomless gall-stones in the gall-bladder found on routine examination at the Mayo Clinic showed that 51 developed symptoms within a few years. There is also the risk, albeit small, of developing carcinoma (p. 936).

Flatulent Dyspepsia.—Apart from gall-stone colic, stones in the gall-bladder may be related to flatulent dyspepsia. This relationship is uncertain as only about 70 per cent. of patients with these symptoms are relieved by cholecystectomy. The symptoms include a feeling of fullness after food, belching and heartburn—all worse after a large or fatty meal, and they occur also in people with a normal cholecystogram. A thorough investigation of other organs is necessary, especially to rule out œsophageal hiatus hernia, and as far as possible, chronic pancreatitis (p. 946).

Gall-stone Colic.—Suddenly the patient experiences excruciating pain

across the upper part of the abdomen.[1] The pain may shoot to the back or between the shoulder-blades. In severe cases it 'doubles her up', and she rolls in agony on the floor. The attack, which lasts for upwards of two hours and is often accompanied by vomiting and retching, passes off nearly as suddenly as it came. Colic usually occurs at night, as in the horizontal position small calculi tend to congregate at the neck of the gall-bladder.

Occasionally the acute pain lasts longer, and the patient abandons hope. Sir Walter Scott, after an attack lasting many hours, 'called his family, and bidding them farewell, turned his face to the wall to await death. Nevertheless, next morning he was up betimes and in fair spirits'.

Physical Signs.—It is impossible to examine the abdomen satisfactorily during an attack of colic. Exceptionally palpation reveals an enlarged gall-bladder.[2] If continuous gentle pressure is exerted over the right hypochondrium while the patient takes a deep breath, there is a 'catch in the breath' just before the zenith of the inspiration (Murphy's sign[3]).

Jaundice follows an attack of gall-stone colic in 18 per cent. of cases, and mostly is due to a stone obstructing the common bile duct.

Radiography.—See p. 909.

Treatment:

(*a*) **Of Gall-stone Colic.**—Morphine alone is undesirable as it causes spasm of the sphincter of Oddi (p. 944). Pethidine,[4] also causes spasm. It is essential to give an antispasmodic drug (e.g. atropine or promethazine hydrochloride) before or with the analgesic drug. In most cases heat may relieve the pain, and in old-standing cases a brown pigmentation of the skin over the right hypochondrium tells of frequent hot applications.

(*b*) **Subsequent Treatment.**—Once a patient has had gall-stone colic, no question arises as to the treatment that should be recommended: it is *cholecystectomy*, unless the patient is very old or otherwise enfeebled, when *cholecystostomy* may be substituted. In either case it is essential to examine the common bile duct by all means available for the presence of additional calculi and, if such are present, to remove them (pp. 912, 925).

Mucocele of Gall-bladder.—A mucocele develops as the result of obstruction of the neck of a sterile gall-bladder by a single stone; the contained bile is absorbed and replaced by mucus secreted by the gall-bladder. The organ is thin-walled and translucent and may contain a pint of mucus (fig. 971). The treatment is cholecystectomy.

Dissolving Gall-stones.—The daily oral administration of 1 g chenodeoxycholic acid as its sodium salt may dissolve some cholesterol-containing stones, but *not* when the gall-bladder is non-functioning or the stones are too many or too big or contain

[1] The biliary apparatus is bilaterally innervated, and pain may be under the right costal margin and left costal margin. In 4 per cent. of cases it is under the left costal margin alone.

[2] Sir Zachary Cope, 1881–1975. Surgeon, St. Mary's Hospital, London, described feeling his own tense gall-bladder during colic, and before the onset of the signs of acute cholecystitis (p. 921).

[3] This sign is also named after Bernard Naunyn, 1839–1925, Professor of Medicine, Strasbourg, who described it in 1890, thirteen years before Murphy.

[4] Pethidine is known in Canada as demerol, and in the U.S.A. as demerol hydrochloride. Should the patient be taking a mono-amine oxidase-inhibitor drug for depression, pethidine is contraindicated as collapse can occur from a severe fall of blood pressure.

Sir Walter Scott, 1771–1832. Scottish poet, and master of the romantic historical novel.
John Benjamin Murphy, 1857–1916. Professor of Surgery, North-western University, Chicago, U.S.A.

much calcium (indicating infection). The treatment, which is likely to cause gastrointestinal upsets and may be hepatotoxic, takes about two years and may have to be continued for life. The stones tend to recur if treatment is stopped.

CHOLECYSTITIS

Ætiology.—Cultures from excised gall-bladders and their contents show: *Contents.*—Bacteria are present in 35 per cent. of cases. *Wall.*—Bacteria are present in 65 per cent. of cases. e.g., *Esch. coli*, *streptococci*, *klebsiella*, *Salmonella paratyphi B*, *staphylococci.*

ACUTE CHOLECYSTITIS

Acute Obstructive Cholecystitis.—If a stone impacts in Hartmann's pouch or the cystic duct (fig. 985), the gall-bladder, especially if already the seat of chronic cholecystitis, becomes intensely inflamed with its mucous membrane swollen, and the wall thickened. Occasionally gangrene occurs in patches (fig. 986). The subsequent events are :

1. When a certain degree of distension of the gall-bladder has been reached, the mucous membrane tends to be lifted away from the sides of the

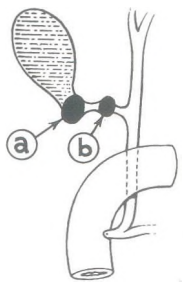


FIG. 985 — The sites of impaction of a gall-stone in acute obstructive cholecystitis: (*a*) In Hartmann's pouch. (*b*) In the cystic duct.

FIG. 986.—Acute obstructive cholecystitis in a Professor. A stone has impacted in Hartmann's pouch. Gangrene is occurring.

stone and as a consequence the stone may slip back into the body of the gall-bladder, viz. —→ and any mucoid (from a mucocele—see above) or muco-purulent contents of the gall-bladder escape by way of the cystic duct.

2. Less frequently the impaction persists and an empyema (pyocele) of the gall-bladder results (p. 924).

3. On rare occasions the distended, inflamed gall-bladder perforates. Doubtless the infrequency of perforation is due to the thickened walls of an organ that has long been the seat of chronic cholecystitis.

Perforation of the Gall-bladder.—The site of perforation is either at the fundus, which is farthest away from the blood-supply, or, less commonly, at the neck from pressure necrosis of an impacted calculus. The sequelæ are:

(*a*) **Local Abscess.**—On account of the present, and probably past, attacks of cholecystitis, there are adhesions between the gall-bladder, the greater omentum, and the parietal peritoneum. Consequently, when an infected, obstructed gall-bladder perforates the usual outcome is a local abscess.

(*b*) **Perforation into the general peritoneal cavity** occurs in only 0·5 per cent. of cases undergoing conservative treatment for acute cholecystitis, and the patient is usually a man. If the bile is infected diffuse peritonitis supervenes readily and rapidly, and the mortality is about 50 per cent. (Ellis).

Internal Fistula of the Gall-bladder (see page 937).

Air or Gas in the gall-bladder or bile ducts is caused by: (i) following operative choledocho-duodenostomy (p. 932), (ii) following cholecyst-duodenostomy, or a stone perforating into the duodenum (p. 929), and (iii) the presence of gas-forming organisms, due to *Cl. welchii*, which is rare but the danger is great and emergency operation is required (fig. 987). Gas in gall stones is described on p. 917.

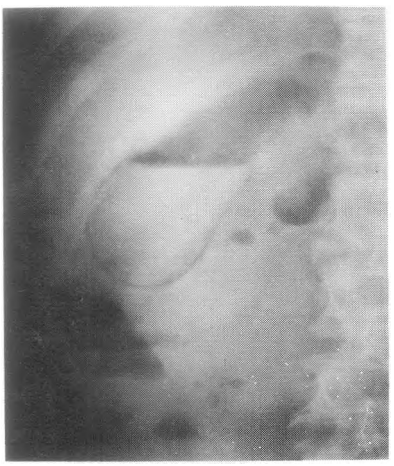


FIG. 987.—Gas in gall-bladder and gall-bladder wall (*Cl. welchii*).

Acute non-obstructive cholecystitis is less frequent than the obstructive variety. All grades of acute inflammation, from catarrhal to gangrenous cholecystitis, occur. In fulminating cases *Cl. welchii* may be implicated. Typhoid and paratyphoid cholecystitis also results in perforation.

Clinical Features of Acute Cholecystitis.—The onset is sudden, and pain—agonising in the obstructive variety, more gradual in the non-obstructive—is located mainly in the right hypochondrium. Severe nausea and vomiting are features in the early stages. Pyrexia, sometimes to 38° C. or more, is usual; occasionally fulminating cases are ushered in by rigors.

On examination tenderness and rigidity are found in the right hypochondrium. If the patient can be persuaded to relax, a mass consisting of the inflamed gall-bladder with adherent greater omentum attached may be felt, particularly in obstructive cases. In mucocele of the gall-bladder pyrexia is absent and a piriform swelling can be palpated in the right hypochondrium.

Differential Diagnosis.—The differential diagnosis between high retrocæcal acute appendicitis and acute cholecystitis and a leaking duodenal ulcer is often difficult, and when there is doubt, conservative treatment (see below) is not advised. Acute pancreatitis must be excluded by all means possible (p. 944). It is also necessary to eliminate acute pyelonephritis, and other infections of the right kidney. In relevant cases an electrocardiograph should be taken to differentiate acute cholecystitis from coronary thrombosis.

Boas's sign, if positive, is sometimes most helpful in distinguishing acute cholecystitis from other conditions. There is an area of hyperæsthesia between the ninth and the eleventh ribs posteriorly on the right side.

Radiography.—*A plain radiograph* may be negative (p. 909). In children the calculi are usually opaque (Forshall). *Oral cholecystography* is usually carried out when the acute phase is subsiding. A non-filling gall-bladder supports the diagnosis of cholelithiasis. Ultrasound may be helpful (p. 911). Gas or air, see above.

Harold Ellis, Contemporary. Professor of Surgery, Westminster Hospital, London.
Ismar Isador Boas, 1858–1938. Gastroenterologist, Augusta Hospital, Berlin.
Isabella Forshall, Contemporary. Formerly Surgeon, Royal Liverpool Children's Hospital, Liverpool, England.

Treatment:

(*a*) **Conservative Treatment followed by Cholecystectomy.**—Experience shows that in more than 90 per cent. of cases the symptoms of acute cholecystitis subside with conservative measures. Non-operative treatment is based upon four principles:

1. *Rest to the inflamed gall-bladder* and the biliary and pancreatic systems by gastric aspiration for three to five days. Nothing is given by mouth. Fluid and electrolyte balance are maintained by continuous intravenous dextrose-saline solution, with the usual precautions (p. 94).

2. *Analgesics.*—The recommendations are given on p. 920.

3. *An anticholinergic drug* is given to reduce gastric and pancreatic secretion, and therefore loss of electrolytes by aspiration, and to relax the sphincter of Oddi. Propantheline is probably the most satisfactory for this purpose.

4. *Antibiotics* of the tetracycline group reach the interior of the gall-bladder *via* the blood-stream even in the presence of occlusion of the cystic duct. These drugs may be given intramuscularly, but oral administration should be commenced as soon as the patient is permitted to take fluids by mouth.

5. *Subsequent Management.*—When the temperature, pulse, and other physical signs show that the inflammation is subsiding (usually by the third day), after the bowels have been emptied, e.g. by a suppository, the gastric aspiration tube is removed, and, commencing with flavoured dextrose drinks, fat-free clear fluids are given for one day, and a soft fat-free diet on the next. A full fat-free diet is then given. A cholecystogram is performed about two weeks later (see Radiography, above). Eight to ten weeks after the acute symptoms have subsided *cholecystectomy* is carried out.

Conservative treatment is not advised (*a*) when there is uncertainty about the diagnosis, e.g. when early high retrocæcal appendicitis or a leaking duodenal ulcer cannot be excluded, (*b*) in typhoidal cholecystitis, because of the frequency of perforation in these cases.

Conservative treatment must be abandoned: If the pain and tenderness spreads across the abdomen and the pulse-rate rises. Cholecystectomy should be undertaken forthwith. In the very ill and the elderly patient it may be advisable to limit the operation to *cholecystostomy*[1]—usually a dependable, safe operative procedure (p. 927).

(*b*) **Routine Early Operation.**—Some surgeons advocate urgent operation as a routine measure in cases of acute cholecystitis. Provided the operation is undertaken within forty-eight hours of the onset of the attack, the surgeon is experienced and excellent operating facilities are available, good results are claimed, but no better than those that accrue from the delayed method described above which is associated with far fewer operative injuries to the main ducts.

It is not proposed here to enter into a controversy on the merits and demerits of early and delayed operation for acute cholecystitis, but to state categorically that the most dangerous period for operation is between the seventh and fourteenth days of the attack. Should operation become imperative during this period, when the ducts are often obscured by œdema and immature fibrous tissue and the liver function is at a

[1] Elective cholecystectomy is still indicated in due course, in suitable patients. Cholecystostomy may relieve all symptoms, but some patients continue to suffer, and they may have an intermittent mucopurulent discharge from the drainage wound.

low ebb, it is often prudent to perform a simple cholecystostomy (p. 927) rather than a difficult cholecystectomy (see footnote[1] p. 930).

Empyema of the Gall-bladder.—This is a curious condition in which an inflamed, obstructed gall-bladder becomes filled with creamy 'pus'. This 'pus' is really a cream of calcium carbonate, cholesterol crystals, and epithelial cells, and may be sterile on culture. The treatment is cholecystectomy.

Chronic Cholecystitis.—The gall-bladder wall is thickened, with loss of the normal mucous membrane pattern. Stones may or may not be present. Many of these gall-bladders are examples of the cholecystoses (see below). Calcification of the wall of the gall-bladder (porcelain gall-bladder fig. 965) is uncommon. Patients with chronic cholecystitis may present with biliary dyspepsia and be diagnosed by cholecystography, or they develop acute cholecystitis.

Typhoid Gall-bladder.—Typhoid carriers 'harbour' living *Salmonella typhi*[1] in their gall-bladders and their gall-stones.[2] The organisms are discharged into the alimentary tract from time to time, and are present in the fæces. Cholecystectomy in such cases may rid the patient of the infection, and the community of a potential danger. The patient must be 'barrier nursed', and given a course of ampicillin. In conjunction with the Public Health Authorities, the patient is kept in an isolation hospital until it has been proved over a sufficiently long period that the fæces are *Salmonella typhi* free.

FIG. 988.—The interior of the strawberry gall-bladder.

THE CHOLECYSTOSES

(Cholesterosis, Polyposis, Adenomyomatosis, Cholecystitis Glandularis Proliferans)

This is a not uncommon group of conditions affecting the gall-bladder in which there are chronic inflammatory changes with hyperplasia of all the tissue elements. Endocrine, metabolic, and dyskinetic factors have been suggested as the cause of the hyperplasia. Malignant change does not occur.

Cholesterosis (*syn.* **Strawberry Gall-bladder**).—In the fresh state the interior of the gall-bladder looks something like a strawberry; the yellow specks (submucous aggregations of cholesterol crystals and cholesterol esters) correspond to the seeds (fig. 988). The presence of macrophages stuffed with the esters (foam cells) indicates that the condition is inflammatory in nature and not, as previously thought, an expression of hyper-

[1] 'Typhoid Mary', a cook-general who passed *Salmonella typhi* in her fæces and urine, was responsible for nearly a score of epidemics of typhoid in and around New York City.

[2] Surgeons should not give patients their stones after operation if there is any suspicion of typhoid!

Daniel Elmer Salmon, 1850–1914. Veterinary Pathologist, Chief of the Bureau of Animal Industry, Washington, D.C., U.S.A.

cholesteræmia and cholesterol excretion. Cholesterosis *per se* is symptomless. Symptoms, if any, are generally due to associated gall-stones or cholecystitis. Cholesterosis is often accompanied by one or more cholesterol stones, supposedly derived from the deposits in the mucous membrane. On cholecystography the concentration of the contrast medium is frequently very dense and the edge of the gall-bladder may appear slightly blurred.

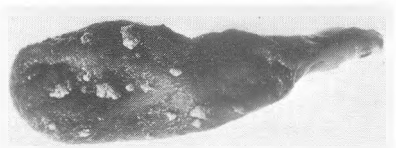


FIG. 989.—Cholesterol polyposis.
(D. J. Oakland, F.R.C.S., Hereford.)

Cholesterol Polyposis of the Gall-bladder (fig. 989).—Cholecystography shows negative shadows in a functioning gall-bladder. The shadows, which are adjacent to the wall of the gall-bladder, remain constant in position and in relation to one another in all films of the series. Histologically, cholesterol polyposis is similar to the cholesterol-laden projections of the strawberry gall-bladder, but the lesions are much less numerous and are relatively gigantic. The treatment is cholecystectomy.

Cholecystitis glandularis proliferans (polyp, adenomyomatosis, and intramural diverticulosis). Fig. 990 summarises the varieties of this condition. A polyp of the mucous membrane is fleshy and granulomatous. All layers of the gall-bladder wall may be thickened, but sometimes an incomplete septum forms which separates the hyperplastic from the normal. Intraparietal 'mixed' calculi may be present which can be complicated by an intramural and, later, an extramural abscess. *Diverticulosis of the gall-bladder* is usually manifest as black pigment stones impacted in the outpouchings of the lacunæ of Luschka (p. 908). Diverticulosis of the gall-bladder may be demonstrated by cholecystography, especially after the gall-bladder contracts after a fatty meal: there are small dots of contrast medium just outside the gall-bladder (fig. 991). A septum may also be present (to be distinguished from the Phrygian cap—fig. 978). The treatment is cholecystectomy.

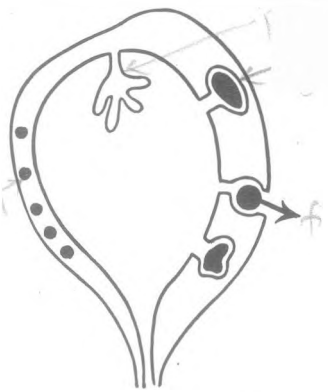


FIG. 990.—Types of cholecystitis glandularis proliferans (polypus, intramural or diverticular stones, and fistula).

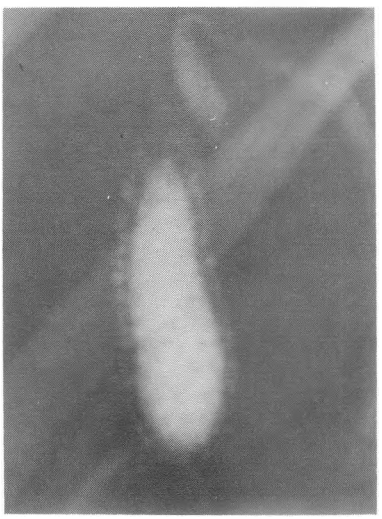


FIG. 991.—Cholecystogram showing cholecystitis glandularis proliferans.

CHOLECYSTECTOMY AND CHOLECYSTOSTOMY

1. **Preparation for Cholangiography** (p. 910).—Provision is made for per-operative cholangiography by placing the patient on an operating table

equipped with a radiolucent top, so that X-ray exposures may be made without disturbance (fig. 973, p. 912). If the table is not so equipped, a cassette holder and film is placed beneath the patient at the appropriate level. Some tables are designed for the use of an X-ray image-intensifier and television control (fig. 976 and p. 912).

Routine cholangiography is preferable to haphazard periodic attempts. It may be omitted in acute cholecystitis and if the cystic duct is too narrow to admit a fine catheter, or indeed to have allowed the passage of stones from the gall-bladder into the common bile duct.

2. **Laparotomy.**—A right paramedian incision is satisfactory. It has the advantage of extensibility.[1] After the peritoneum has been opened, the gall-bladder and the common bile duct are inspected and palpated.

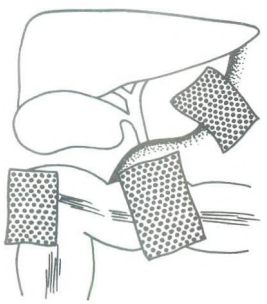


FIG. 992.—Cholecystectomy. Positions of the packs after identifying the colon, the duodenum and the stomach.

N.B.—It is essential before commencing a standard cholecystectomy to examine the other abdominal organs. By so doing the writer has discovered an early carcinoma of the stomach, and on two occasions one in the colon. Naturally resection of the growth takes precedence to cholecystectomy, although in favourable cases this may also be possible.

3. **Cholecystectomy.**—The area is now isolated with packs (fig. 992). If the gall-bladder is greatly distended, it is aspirated through the fundus by means of a trochar and cannula attached to a suction apparatus. The fundus is then grasped with sponge-holding forceps, thereby sealing the puncture. These forceps, and another pair applied to the neck of the gall-bladder, serve as tractors.

Then the very important dissection to display the junction of the cystic, the common hepatic, and the common bile ducts is commenced.[2] During the course of this dissection the cystic artery is found, and its relation to the common hepatic artery verified (p. 909). The artery is then divided between ligatures at least 5 mm away from the hepatic duct (fig. 993). Cholangiography (p. 910) is performed at this stage. The cystic duct is then ligated. Forceps are applied to the gall-bladder side and cystic duct is divided. From below, upwards, the gall-bladder is dissected from its bed, dividing the peritoneum on the gall-bladder (fig. 994). The gall-bladder having been removed and hæmostasis assured, the abdominal wall is closed with drainage of the operation instituted via a separate ' stab ' wound.

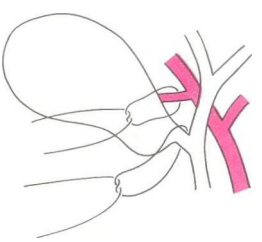


FIG. 993.—Ligatures are passed and tied round the cystic artery and cystic duct.

Some Golden Rules in case of difficulty:

(1) Clear identification of the colon, the pylorus and duodenum is a prerequisite for a proper approach to the ductal and arterial system.

(2) A common duct hidden in fibrous tissue may be located by means of an aspirating syringe and fine needle.

(3) With severe inflammation in Calot's triangle (p. 908) it may be wise to open the gall-bladder, extract all the stones and bile, and as much of the wall of the gall-bladder as possible. The cystic duct opening is closed by a catgut suture from ' within '. Any mucous membrane remaining on the hepatic side is diathermied. An alternative is cholecystostomy.

[1] Big surgeons make big incisions.

[2] ' It is the left hand of my assistant that does all the work ' (Moynihan). By splaying the fingers to depress the stomach and duodenum, he displays the biliary ducts, aided also by retraction of the quadrate lobe of the liver.

(4) A clear indication of the position of the common and right hepatic duct in the danger area can be given by inserting a sound upwards through a choledochotomy below.

A cholecystohepatic duct (fig. 978(*e*)), is present in about 0·5 per cent of cases, and is usually the size and colour of No. 60 thread. Its possible presence must be remembered when separating the gall-bladder from its bed, and, if recognised in time, it can be ligated. If avulsed from the liver, a persistent ooze of bile occurs from the ruptured end, which should be sutured.

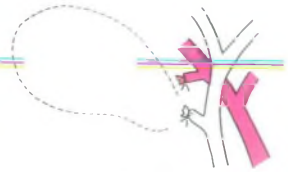


FIG. 994.—The gall bladder has been removed.

Cholecystostomy.—Two stay sutures are inserted on either side of the fundus, in order to steady the organ, the fluid contents of which are aspirated. The fundus is opened and stones are removed from the interior by Desjardins' forceps, aided, always, by a finger milking up a stone or stones from Hartmann's pouch. Minute calculi are often dislodged by strips of dry gauze passed into the interior. A 6-mm drainage tube is passed into the gall-bladder, and there retained by a transfixion stitch. The opening in the gall-bladder is closed about the tube. The tube is brought through a portion of greater omentum, which is anchored to the gall-bladder by the original stay sutures. The tube is then brought to the surface through a separate stab incision. The abdominal incision is closed, and the tube is joined by a connection to more tubing, which leads to a sterile polythene bag. Seven to ten days later Hypaque injected down the tube may be seen on X-ray to flow easily into common bile duct and duodenum if no obstruction exists, in which case removal of tube will be followed by closure of the biliary fistula within a week (see footnote p. 923).

Bacteriology at Operation.—At every biliary operation cultures are taken of the bile and, if removed, from the gall-bladder wall (p. 921). In acute inflammatory disease a broad spectrum antibiotic will be needed immediately and changed subsequently according to the bacterial sensitivity report (see Sepsis, Chapter 3).

Indications for Choledochotomy following Cholecystectomy (above).—In the absence of peroperative cholangiography, opening and exploring the common bile duct is indicated when: (1) Stones can be felt in the ducts. (2) There is jaundice or a history of jaundice, or rigors. (3) A dilated common bile duct (10 mm diameter or more) is present. (4) If the cystic duct is dilated (implying that stones may have migrated from the gall-bladder to the common duct). (5) Intravenous cholangiogram has shown the presence of a stone. Choledochotomy is described on p. 930.

SYMPTOMS PERSISTING AFTER CHOLECYSTECTOMY

In 15% of all cases, cholecystectomy fails to relieve the symptoms for which the operation was performed. The term 'post-cholecystectomy syndrome' should be abandoned. The symptoms are most commonly due to disease of organs other than the biliary tract, such as hiatus hernia, duodenal ulcer, pancreatitis or diverticulitis. If these can be eliminated, one of the following lesions of the biliary tract must be considered:

1. A stone in the common bile duct escaped detection at the original operation.
2. When a comparatively long stump of a cystic duct has been left behind as frequently happens where the cystic duct joins the common bile duct lower than usual (fig. 978(*d*)). This diverticulum may enlarge considerably and may harbour infection. Calculi may form therein.
3. Operative damage to the common bile duct occurred, resulting in stricture of that duct (p. 932).
4. The cause of the original symptoms was biliary dyskinesia (p. 912).

Treatment.—Intravenous cholangiography (p. 910) is valuable in determining the cause which, if possible, is removed.

Abel Desjardins. Surgeon, Dispensaire Henri de Rothschild, Paris.

GALL-STONES IN THE BILE DUCTS

In addition to those in the gall-bladder and cystic duct (p. 884), stones may be present in the common bile duct, the common hepatic and the intrahepatic ducts. They may originate from the gall-bladder, having negotiated their way by a process of dilatation of the cystic duct. Those formed primarily in the hepatic or common ducts may range from large concretions down to sludge. A number may contain a nucleus around which bile pigment and cholesterol have been deposited (e.g. rolled-up fruit skins, ascaris ova or flukes (p. 916)).

Clinical Features.—Usually there is a long history of flatulent dyspepsia, not infrequently punctuated by episodes of biliary colic.

Pain.—There is no great difference between the biliary colic described already (p. 919) and that occasioned by a stone becoming impacted in the common bile duct. In the latter instance the pain is likely to be prolonged, severe, and difficult to relieve, and is followed by jaundice. A parous woman will often say that the pains of childbirth are preferable.

Jaundice.—Within forty-eight hours the pain is followed by jaundice, the tinge of which ranges from pale lemon to bright orange. The urine becomes dark with bile, and the fæces become pale. The skin commences to itch, sometimes intolerably. The patient loses weight.

Abdominal Examination.—Usually, soon after the attack of colic, tenderness can be elicited in the epigastrium. As a rule the gall-bladder is impalpable.

Courvoisier's 'Law' [1] states that if in a jaundiced patient the gall-bladder is palpably enlarged, it is probably *not* a case of stone impacted in the common bile duct, because in that case previous cholecystitis has already made the gall-bladder fibrotic.

Sometimes it happens that after an attack of biliary colic, followed by fleeting jaundice, the patient remains well for many months. More often than not, after a varying interval another similar attack occurs, and it is sometimes presumed that another gall-stone has entered the common bile duct. But this is not necessarily the case; it is likely that the same stone has again caused obstruction.

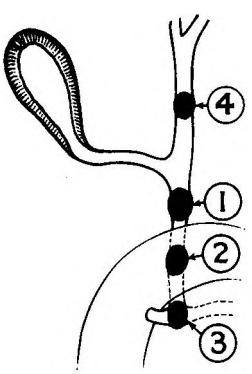


FIG. 995.—Sites of stone impaction. 1. Stone impacted in the supraduodenal portion of the common bile duct (commonest site).
2. Stone impacted in the retroduodenal portion of the common bile duct (second most common site).
3. Stone impacted in the ampulla of Vater (about 10 per cent. of cases).
4. Stone impacted in the common hepatic duct (rare).
Note that the gall-bladder is usually thick-walled and contracted because of chronic cholecystitis (Courvoisier's Law, above).

[1] Courvoisier only made a statement of probability (approximately in 75% of cases). Posterity unwisely considered it a 'law'.

Ludwig Courvoisier, 1843–1918. Professor of Surgery, Basle, Switzerland.

Charcot's Biliary Triad.—Fluctuating jaundice, recurrent pain, and intermittent fever with rigors[1] constitute this triad. A stone becomes impacted but floats free when the duct dilates, and subsequently impacts again.

Atypical Cases:

1. **Jaundice from Pressure of a Stone on the Duct from Without.**—Jaundice can arise from a large calculus in Hartmann's pouch or extrabiliary pathology pressing upon the common bile duct.

2. **Jaundice is the First Symptom.**—In about 3 per cent. of individuals jaundice is the first and only symptom.

3. **Stone in the Common Bile Duct without Jaundice.**—Stones may lie dormant for many years in the common bile duct, giving rise only to vague indigestion.

Differential Diagnosis.—Calculous biliary obstruction is a very frequent cause of jaundice encountered in surgical practice (fig. 996). It is important to differentiate a stone in the common duct from virus hepatitis (p. 883) and carcinoma of the pancreas (p. 954).

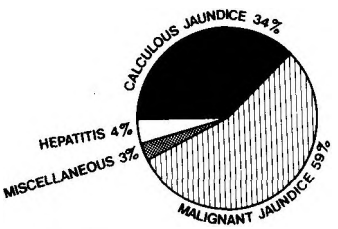


FIG. 996.—Incidence of jaundice in surgical practice (The London Hospital Surgical Unit, 1964–1973, 197 cases).

Ultrasound Diagnosis and **Laboratory Investigations** are described on p. 911 and p. 880.

Complications.—If the obstruction is not relieved, one of four (three dangerous) sequelæ ensues:

1. **Liver Function becomes increasingly impaired.**—When liver function becomes seriously depressed, as evinced by the finding at operation of 'white bile' in the bile passages above the site of an impacted calculus, the outlook is serious, but not hopeless, as a brisk recovery often takes place. However, jaundice due to stone lasting for over four weeks tends to cause liver damage, and the operative mortality rises.

2. **Suppurative Cholangitis Supervenes.**—This is a dangerous complication, causing liver failure and death and requires timely surgical intervention and antibiotic therapy (p. 17).

3. **The gall-stone ulcerates through the wall of the common bile duct** and diffuse bile peritonitis supervenes. The perforation may be minute and difficult to find and sometimes occurs into the retroperitoneal tissues. The treatment is to drain the common bile duct, and also the general peritoneal cavity or the retroperitoneal tissues, as the case may be.

4. On rare occasions (as subsequently revealed at necropsy) a stone in the common bile duct ulcerates into the duodenum, and a natural cure follows.

'White Bile'.—The phenomenon occurs following obstruction of the common bile duct or the common hepatic duct, usually (but not always) when the gall-bladder is absent or not functioning. White bile is mostly mucus derived from the glands lining the ducts, to which has been added a minute amount of cholesterol, and either only traces of bile salts, or none. The term 'white bile' is a misnomer, since it is not bile derived from the liver but mucus, for the liver fails to excrete due to cellular damage or mounting pressure within the bile ducts; also it is not white but opalescent.

[1] In Britain the two common causes for rigors are urinary and biliary obstruction with infection. In equatorial regions, malaria is the commonest cause.

Jean Martin Charcot, 1825–1893. Physician, Hopital Salpêtrière, Paris.

MANAGEMENT OF BILIARY OBSTRUCTION DUE TO STONE

To commence with, a high intake of dextrose is essential to build up the store of liver glycogen, and also to protect the liver (Chap. 45).

If the Jaundice Abates.—The depth of the jaundice may diminish, the raised bilirubin level becomes constantly lower, and the stools become increasingly coloured. Such a train of events implies disimpaction of the stone, or possibly the passage of a small stone into the duodenum. In such circumstances diagnostic intravenous cholangiography can be undertaken and laparotomy performed if stones are still present.

If the Jaundice does not Abate.—If the jaundice is found to be increasing rather than decreasing, and particularly when any degree of jaundice is accompanied by pyrexia, operation should be planned to take place within two or three days.

Jaundiced patients tend to ooze from a cut surface. This tendency to bleed may be due to a diminution of prothrombin in the blood, resulting from failure of absorption of vitamin K_1 which is consequent upon diminution of bile salts in the alimentary canal. To raise the prothrombin level in the blood, the patient is given vitamin K analogue (menapthone B.P.) 10 mg twice daily i.m., or vitamin K_1 (phytomenadione) 10 mg, i.m. or i.v. (in an emergency). Blood transfusion is another important ancillary. Antibiotics are given (see p. 927). *Mannitol* (p. 82) may be given intravenously to promote a diuresis and to prevent the renal failure which is a cause of mortality.

In patients who are deeply jaundiced, or who show signs of suppurative cholangitis, operation may be limited in the first instance to draining the common bile duct (choledochotomy), and removing a stone or stones only if they are readily accessible. When the patient has recovered[1] the subsequent elective procedure can be planned.

Choledochotomy.—If a stone (or stones) is present in the common bile duct, its removal should have priority over cholecystectomy. Should the patient be unfit for cholecystectomy, or even cholecystostomy, the gall-bladder should be removed on a future occasion.

Supraduodenal Choledochotomy.—Most stones in the common bile duct can be removed by this route. If, as is often the case, a stone can be felt, an attempt is made to manœuvre it into a position midway between the entrance of the cystic duct and the superior border of the duodenum. The stone is steadied between the finger and thumb. The duct is opened longitudinally directly on to the stone, enabling it to be removed by a malleable scoop or Desjardins' gall-stone forceps. The interior of the duct is then explored upwards and downwards with the scoop for further stones.

When the stone cannot be felt, or cannot be manipulated into the optimum position just described, after incising the peritoneum overlying the common bile duct, a length of the underlying structure is displayed. (See also ' golden rules ' p. 926.) As soon as about 2 cm of the common bile duct has been exposed, the duct is transfixed by two stay sutures and a longitudinal incision into the duct is made between them. Escaping bile is mopped up or removed by suction. Through this opening it may be possible to identify the stone and remove it with a scoop or forceps (fig. 910). After removal of the stone or stones by either of the above methods the common bile duct is cleared of any mud or grit by ' milking ' the duct, followed by irrigation with saline solution. The patency of the sphincter of Oddi is tested either by the passage of a sound (3 to 5 mm diameter), or by a confirmatory cholangiogram (p. 910).[2] Usually drainage of the common bile duct is carried out by means of a T-tube (fig.

[1] A living problem is better than a dead ' cert '. (Grey Turner).

[2] It is estimated that 7 per cent. of patients with cholelithiasis have intrahepatic calculi in addition.

George Grey Turner, 1877–1951. Professor of Surgery, Postgraduate Medical School, London.

998).[1] The transverse limb, shortened if necessary to about 5 cm long, is inserted in the duct which is closed snugly about the vertical limb, using fine catgut on an atraumatic needle. The long limb is brought out through a separate stab wound and securely anchored to the skin. The bile draining from the tube is collected in a plastic bag by the side of the bed, its amount and character being noted. After 10 days the tube may be clamped for increasing periods, and the absence of pain and jaundice, the presence of bile in the stools indicate satisfactory flow into the duodenum. Hypaque is injected down the tube to obtain a cholangiogram (p. 912), and if there are not filling defects in a well-outlined duct, and the ' dye ' enters the duodenum freely, the T-tube can be removed by a sharp pull. Subsequent bile drainage is minimal and does not usually persist for more than a day.

Closure of the Common Duct without a T-*tube.*—If this procedure is attempted, it is most necessary to provide drainage placed in apposition to the common duct.

Transduodenal Sphincterotomy is indicated when a stone is found to be impacted near the ampulla of Vater (fig. 999) and it cannot be retrieved from above. Some surgeons prefer the method to supraduodenal choledochotomy.

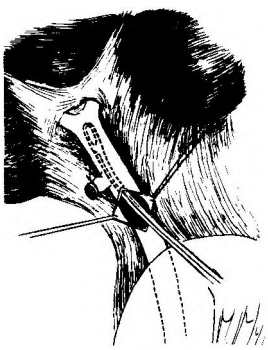


FIG. 997.—Choledochotomy. The stone is seized with Desjardins' forceps.

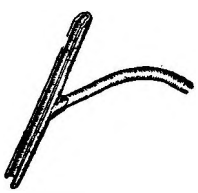


FIG. 998.—T-tube for draining the common bile duct.

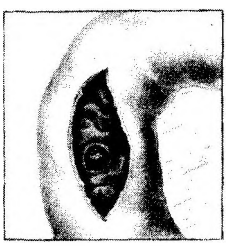


FIG. 999.—The transduodenal approach to a stone impacted in the ampulla of Vater.

The duodenum is opened in its second part by a transverse or longitudinal incision between stay sutures, and the region of the ampulla brought into the opening by traction using tissue forceps. Removal of the stone is either easy or requires the division of the duodenal papilla and the sphincter (fig. 1000).

Endoscopic Sphincterotomy and Gall-stone Removal.—Using diathermy, a

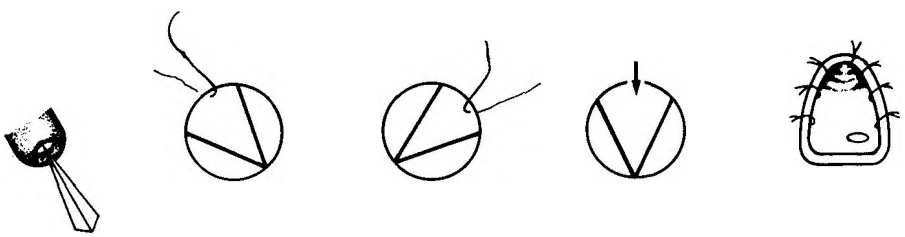


FIG. 1000.—Sphincterotomy.—A fine-pointed grooved director is introduced into the papillary opening. It is turned to 10 o'clock and a catgut suture traverses the duodenal and ampullary mucosæ. It is then turned to 2 o'clock and another suture is introduced. The director is turned to ' 12 noon ' and an incision is made. This routine is repeated until a satisfactory opening is obtained. Care is taken to resite the director to avoid the pancreatic duct. The operation is concluded by tying the sutures (mucosa to mucosa).

(*after R. Hunter, F.R.C.S.*)

[1] T-tubes should be of latex or rubber and only used once. Plastic tubes are ' hardened ' by the bile and are difficult to remove. Latex (and rubber) stimulate the fibrinous adhesion of omentum to liver and colon to form a safe track. There is little reaction to a plastic tube and therefore the risk of biliary peritonitis is greater.

sphincterotomy can be performed under endoscopic control in lightly sedated patients. Any gall-stones remaining in the bile duct when the patient is re-examined with E.R.C.P. (p. 933), 7 to 10 days later may be removed under screening control, using a long Dormier type stone basket passed down the biopsy channel of the duodenoscope. This procedure may be of value in a small selected group of patients who are a poor operative risk due to age, medical illness or previous biliary surgery.

Choledochoduodenostomy is indicated when the common duct is grossly dilated because of an unresolved problem in the region of the biliary sphincter and papilla, e.g. due to stone, sludge, pancreatitis and papillary fibrosis, in the older patient where imprecise operative trauma to this delicate region is likely to be followed by acute pancreatitis. Also in overt carcinoma of the lower end of the bile ducts where palliation only is possible this operation is to be preferred to cholecystenterostomy when involvement of the cystic duct is imminent (see low insertion of cystic duct p. 913). The operation is contraindicated if the common duct is not 1 cm or more in diameter or it is impossible to make a stoma of 2–3 cm. The stoma is fashioned to extend as low as possible in order to reduce the collection of sludge in the 'sump' necessarily left in the retroduodenal portion of the duct. It must permit free entry and egress of fluid, for if it is large enough, regurgitant cholangitis does not occur and liver function is not impaired. The two openings are approximated with catgut, with several reinforcing fine silk sutures. A drain is inserted (fig. 1001). The convalescence is usually surprisingly placid.

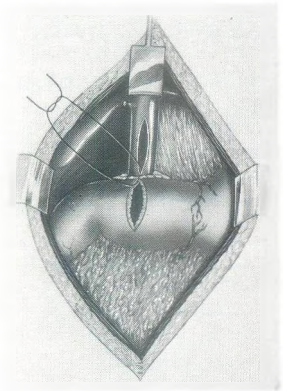


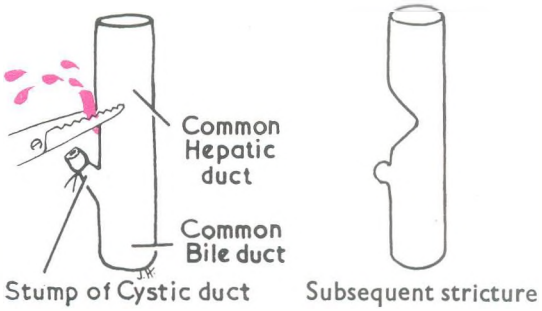
FIG. 1001.—Choledochoduodenostomy. The stoma must be 2–3 cm long.

STRICTURE OF THE COMMON BILE DUCT

- BENIGN
  - Post-operative 80%
  - Inflammatory 20%
- MALIGNANT (p. 933)

Post-operative stricture concerns either the common bile duct or the common hepatic duct. In a few cases only the right hepatic duct is implicated. The stricture is the result of a preventable error in technique, during the performance of cholecystectomy:

1. Blind plunge application of a hæmostat to a bleeding cystic or accessory cystic artery, or to the right hepatic artery, is likely to damage the common hepatic duct, viz.——→

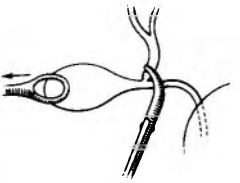


The prevention of this tragic happening is standardised. All unexpected hæmorrhage in this region should be controlled initially by inserting an index finger into the foramen of Winslow, and pinching the free edge of the gastro-hepatic omentum between the finger and thumb. Temporary compression of the hepatic artery in this way allows the bleeding-point to be visualised and ligated accurately (Hogarth Pringle's manœuvre).

2. Should cholecystectomy be performed by dissecting from the fundus (the fundus-first operation) too much traction applied to the freed gall-

Jacob Benignus Winslow, 1669–1760. Professor of Anatomy, Physic and Surgery, Paris.
James Hogarth Pringle, 1863–1941. Surgeon, Royal Infirmary, Glasgow, Scotland.

bladder may so tent the common bile duct that any forceps intended for the cystic duct grasp the angulated main channel,[1] viz. ——————→



3. Ignorance of the anatomical anomalies of the bile ducts (p. 912).

4. Laceration of the common bile duct while exploring it for stones or contracture of Oddi's sphincter.

5. In 3 per cent. of cases of stricture of the common bile duct, injury occurs during partial gastrectomy for a penetrating duodenal ulcer.

About 15 per cent. of injuries to the bile ducts are recognised at the time of the operation. In 85 per cent. of cases the injury declares itself post-operatively by: (*a*) a profuse and persistent discharge of bile if drainage has been provided, or bile peritonitis if such drainage has not been provided; (*b*) by deepening obstructive jaundice. When the obstruction is incomplete, jaundice is delayed until subsequent fibrosis renders the lumen of the duct inadequate.

Inflammatory strictures may result:

(1) From pooling of bile around the common bile duct, such as occurs after insecure ligation of the cystic duct during cholecystectomy or when the duct is improperly drained.

(2) Stenosis can also follow cholecystectomy or choledochostomy when the mucous membrane of the ducts is acutely inflamed (' red-hot ' duct).

(3) Occasionally stricture of one or more ducts (usually the hepatic ducts) occurs as a result of what is known as stenosing cholangitis, the ætiology of which is obscure.

(4) Asiatic cholangio-hepatitis (p. 935).

Radiological Investigation of Biliary Strictures

(1) Intravenous cholangiography (p. 910) with tomography may be helpful providing the serum bilirubin is less than 3 g/100 ml.

(2) Cholangiography *via* a T-tube if present.

(3) E.R.C.P. (see below and p. 957).

(4) Transhepatic cholangiography (figs. 1004 and 1009).

(5) Ultrasound (p. 911).

E.R.C.P.—Endoscopic Retrograde Cholangio-Pancreatography will demonstrate the entire biliary tree (fig. 1002) and will help to distinguish between a medical and a surgical cause of obstructive jaundice.

Malignant bile duct strictures.—E.R.C.P. will show the lower extent of obstruction, and a percutaneous transhepatic cholangiogram immediately pre-operatively determines the upper extent (fig. 1004). A pancreatogram will differentiate between duct cancer and pancreatic cancer (figs. 1005 and 1006).

Gall-stones are well shown, in patients who have previously had biliary surgery (fig. 1007) and in obstructive jaundice. If the bile duct is dilated it is prudent to inject a more dilute contrast (Hypaque 25%) than is used for pancreatography, otherwise small multiple stones may be obscured (fig. 1008).

Acute cholangitis may follow E.R.C.P. when a dilated obstructed duct is filled. Antibiotics and expeditious surgery are then required.

Transhepatic Cholangiography (fig. 1009).—The prothrombin level should be normal or raised to normal by i.m. injection of Vitamin K_1, and an intravenous test dose of 1 ml. of the medium given to exclude hypersensitivity (p. 910). Either the percutaneous (general anæsthetic) or the per-operative laparotomy approach is used. In the former case the needle is introduced just to the right of the xiphisternum. The needle is 15 cm long, gauge 20, ensheathed by a flexible polypropylene tube, and

[1] Forceps should not in fact ever be used for grasping the cystic duct prior to ligature (fig. 993).

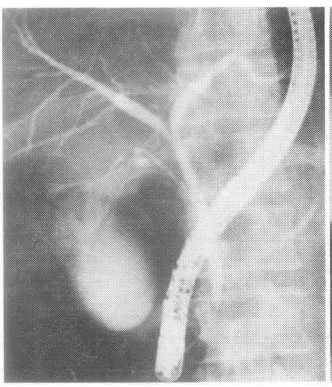


FIG. 1002.—E.R.C.P.: Normal cholangiogram.

FIG. 1003.—E.R.C.P.: Complete occlusion of common hepatic duct due to cholangiocarcinoma.

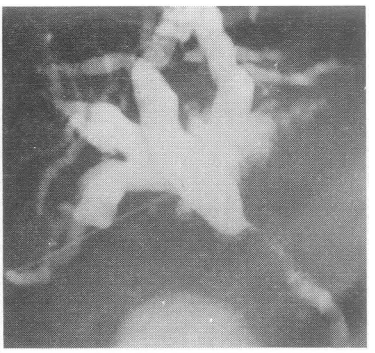


FIG. 1004.—Percutaneous transhepatic cholangiogram in the same patient as fig. 1003. Demonstrates the upper extent of the malignant stricture.

FIG. 1005.—E.R.C.P.: Partial occlusion of bile duct by malignant stricture.

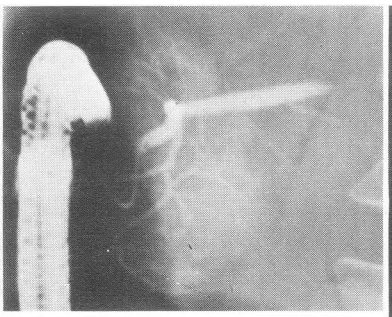


FIG. 1006.—E.R.C.P.: Same patient as fig. 1005. Complete block to main pancreatic duct indicates a pancreatic carcinoma.

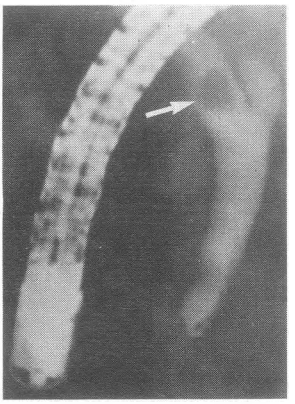


FIG. 1007.—E.R.C.P.: Small solitary gall-stone in bile duct following cholecystectomy.

it is passed into the substance of the right, and, if necessary, the left lobe of the liver. The needle is withdrawn and trial-and-error aspiration is performed until bile (usually white bile, p. 929) enters the syringe, whereupon 20 to 40 ml of 45 per cent Hypaque (sodium diatrizoate) is injected and an X-ray exposure made. Visualisation by using an X-ray image intensifier is helpful. Several attempts are made to find bile before it is presumed that no dilatation of the intrahepatic bile ducts exists (as in hepatitis). A sample of the bile is always examined for bacteria—coliform organisms are often present (George).

Treatment.—As these patients with strictures or biliary fistulæ are debilitated, efforts should be made to improve their poor condition (Chap. 8). Various operative procedures are employed, according to the site of the stricture:

1. **Choledochoduodenostomy** (p. 932) is applicable if the duct protrudes more than 2 cm below the porta hepatis. The duodenum can easily be mobilised upwards after mobilisation. A wide stoma is essential, otherwise stricture will occur.

Phyllis Ann George, Contemporary. Surgeon, Royal Free Hospital, London.

2. **Hepaticojejunostomy,** using a jejunal conduit (Roux loop), is performed when choledochoduodenostomy cannot be undertaken. A mucosa-to-mucosa anastomosis (Smith) is best but an effective alternative is to sew the open end of the jejunum

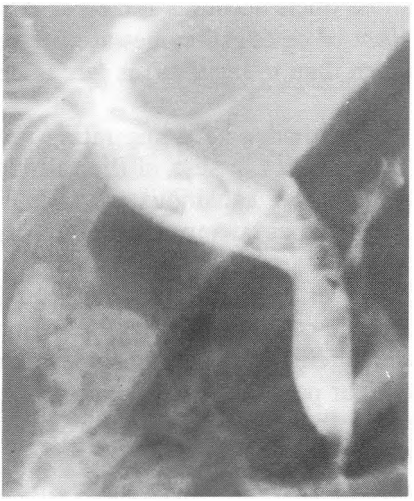


FIG. 1008.—E.R.C.P.: Small multiple gall-stones in dilated bile duct following cholecystectomy. (See operative cholangiography, p. 911.)

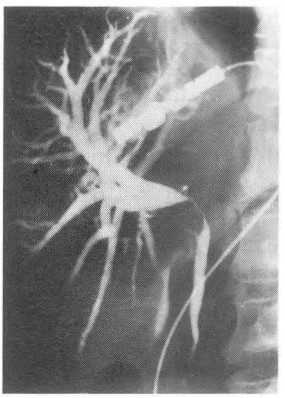


FIG. 1009.—Transhepatic cholangiography (see text), showing a stricture of the common hepatic duct. (*Miss Phyllis George, F.R.C.S., London.*)

to the liver around openings made in the ducts. Any temporary post-operative drainage of the duct, and any splintage of the anastomosis, is obtained by suitably fenestrated latex tubes emerging from the jejunum, the liver or from both.

3. Should the gall-bladder by chance still be in situ it can be used as an intermediate conduit between the ducts above the obstruction and the duodenum or jejunum below.

ASIATIC CHOLANGIO-HEPATITIS[1]

Obstruction to the common bile duct due initially to infection by liver flukes (*clonorchis sinensis*), which live in the biliary passages of a mammalian host, is frequently encountered in the Far East. The patient presents with pain, jaundice, and intermittent pyrexia. The urine contains bile. Clay-coloured stools are less frequently observed. Secondary infection of the obstructed ducts has almost always occurred by the time the patient seeks relief. Laparotomy should be undertaken as soon as the patient can be rendered fit to undergo the operation.

Operation.—The gall-bladder is frequently distended but is not often acutely inflamed. Palpation of the common bile duct reveals the presence of calculi and it is not unusual for the whole of the accessible system to feel solid with impacted stones. On opening the common bile duct, thick purulent bile, often containing adult clonorchis, gushes out and is removed by suction. The common bile duct and hepatic ducts are cleared of stones and mud by scoop and forceps, aided by irrigation with saline. Strictures frequently exist in the intrahepatic bile ducts which may render complete evacuation difficult or impossible. Choledochoduodenostomy at the initial exploratory operation gives better results than T-tube drainage, but if the duct is not very dilated transduodenal sphincterotomy may be adequate. A wide stoma allows stones to pass to the duodenum and helps to prevent a recurrence, although if intrahepatic biliary strictures are present multiple liver abscesses may form and the prognosis is then poor. Cholecystectomy should always be combined with choledochoduodenostomy or sphincterotomy, for if it is not done the accumulation of stagnant

[1] This condition may occur in all countries where watercress, especially if wild, is eaten. Sheep and cattle are prone to the condition.

Rodney Smith (Lord Smith of Marlow), Contemporary. Formerly Surgeon, St. George's Hospital, London.

and infected bile within the gall-bladder frequently results in a secondary cholecystitis (Stock).

CARCINOMA OF THE GALL-BLADDER AND BILE DUCTS

Carcinoma of the gall-bladder and bile ducts accounts for 1 per cent.[1] of all malignant neoplasms. In 90 per cent of cases gall-stones are present, so this condition is more common in women than in men. It is rare under the age of forty.

The usual type is a scirrhous adenocarcinoma, a hard infiltrating tumour, usually starting in the fundus. If it arises near the neck of the gall-bladder a mucocele may follow from obstruction. A squamous carcinoma may follow the squamous metaplasia of the mucous membrane due to chronic irritation by gall-stones.

Malignant spread occurs early to the liver either directly or through the cystic lymph node and lymph nodes in the portal fissure. Involvement of the latter results in obstructive jaundice and, later, ascites. In many cases a past history of cholecystitis can be obtained, but pain, if present, tends to be continuous, with nausea, loss of appetite and weight. In over a third of cases a firm non-tender tumour is palpable in the gall-bladder area.

Treatment and Prognosis.—In view of the relationship with chronic cholecystitis and gall-stones, all gall-bladders on removal should be sectioned. In cases where a very early lesion is found, good results may be expected. Otherwise the prognosis is dismal (2 per cent. surviving five years). For the comparatively early definite cases, cholecystectomy with excision of adjoining liver is performed, but in many cases, especially where deep jaundice is present, no surgical treatment is possible.

Carcinoma of the major bile ducts (**cholangiocarcinoma**) is less common than carcinoma of the gall-bladder. Gall-stones co-exist in 38 per cent. of cases. The primary lesion can be intrahepatic or extrahepatic. When occurring in the portahepatis it obstructs the left and right hepatic ducts. In such a case and in others situated above entry of the cystic into the common bile duct the gall-bladder is flaccid. The diagnosis may be confirmed by hepatic scintiscanning (a hilar defect), percutaneous transhepatic cholangiography (p. 933) and E.R.C.P. (p. 933). In most cases the state of affairs is only recognised at laparotomy for jaundice.

This cancer may grow slowly, but eventually spread throughout the liver occurs and hepatic coma heralds the termination.

Treatment.—Early laparotomy affords the patient a slender chance of prolonged survival. Rarely is the growth found to be resectable. Sometimes palliative procedures such as choledochoduodenostomy (p. 932) or hepaticojejunostomy (p. 935) or the insertion of a tubular stenton (fig. 1010) are possible, and symptomatic relief of the jaundice is afforded thereby.

Dacron Jump Graft.—This is a useful addition to the palliative methods of relieving biliary obstruction. A length of woven graft is inserted end-to-the-side of the common hepatic duct in the portahepatis and end-in-side to the duodenum below, well away from any direct duodenal invasion by the tumour. It is quick and easy to perform and the postoperative morbidity and mortality is low (Reynolds).

Carcinoma of the Ampulla of Vater is discussed on page 954.

BILIARY FISTULÆ

(*a*) **External Biliary Fistula**—In days gone by, gall-stones and bile were sometimes discharged with the pus after a superficial abscess of the abdominal wall had been incised, or had ruptured. Even spontaneous extrusion of gall-stones through the

[1] In Great Britain.

Francis Edgar Stock, Contemporary. Formerly Professor of Surgery, Liverpool, England.
Keith William Reynolds, Contemporary. Surgeon, Charing Cross Hospital, London.

umbilicus has been reported. Today nearly all external biliary fistulæ follow operations upon the biliary tract. When the neck of the gall-bladder or the cystic duct is occluded by a calculus (fig. 1011A), the discharge is mucus. Should a stone be

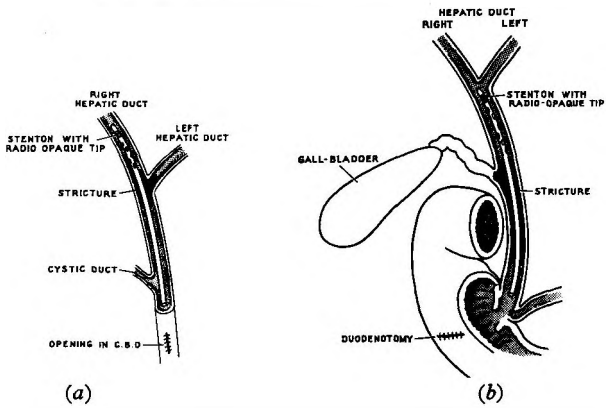


FIG. 1010.—Insertion of a stenton for (*a*) a high lesion and (*b*) a low lesion (*after Ritchie and MacLean*).
(*from Annals of the Royal College of Surgeons*)

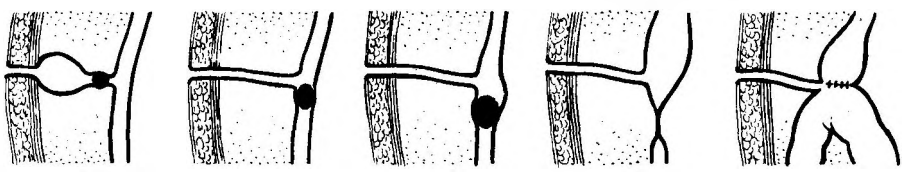


FIG. 1011.—Underlying causes of an external fistula of the gall-bladder or the bile ducts.
(*After J. J. Byrne.*)

occluding the common bile duct, and cholecystostomy is performed, bile will continue to be discharged externally (fig. 1011B). If cholecystectomy has been performed in these circumstances, again a biliary fistula (fig. 1011C) will result. By far the most common cause of a biliary fistula is injury to the common bile duct during cholecystectomy (fig. 1011D). A leakage after cholecysto-jejunostomy (fig. 1011E) or cholecystduodenostomy also will give rise to a fistula discharging bile. Other causes of fistula are division of an aberrant right hepatic duct or a hepaticocystic duct (p. 914).

After investigating by cholangiography the origin of the leak, the continuity of the main bile duct must be restored (p. 934) or, in the case of a mucous fistula, the gall-bladder or its remains must be removed.

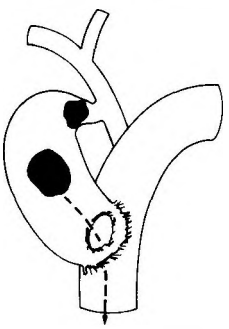


FIG. 1012.—Fistulation of gall-stone into duodenum.

(*b*) **Internal biliary fistula** is due to a stone ulcerating through the wall of the gall-bladder or the common duct into a hollow viscus, viz. :

1. *Into the duodenum*—a large and usually single gall-stone sometimes ulcerates into the duodenum (fig. 1012); this is the commonest internal biliary fistula. The stone may impact there causing duodenal obstruction. If smaller, it may pass on to obstruct in the lower ileum (gall-stone 'ileus', p. 1050). There is evidence that such a fistula will close spontaneously. Later, if symptoms persist, the gall-bladder can be removed.

2. *Into the colon*—as a rule no untoward complications ensue, but a large stone occasionally becomes impacted in the pelvic colon or the rectum. A hitherto silent carcinoma may be brought to light in this way.

3. *Into the stomach*—the patient may vomit the stones.

Alexander David Willard MacLean, Contemporary. Surgeon, The London Hospital.

CHAPTER 47

THE PANCREAS

Surgical Anatomy.—Every student is familiar with the head, neck, body, and tail of the pancreas. From the lower and left part of the head projects the uncinate process upon which rest the superior mesenteric vessels. The posterior surface of the neck is grooved deeply by these vessels, and more superiorly by the portal vein. In spite of the intimate contact of the portal vein with the pancreas, except in the case where it is bound down by inflammatory adhesions or in neoplastic infiltration, it can be separated from the pancreas with comparative ease. The arterial supply of the head comes from the pancreaticoduodenal arteries while the body and the tail are nourished mainly by branches of the splenic artery. The lower part of the common bile duct lies between the second part of the duodenum and the head of the pancreas.

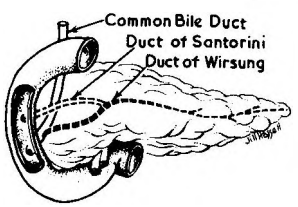


FIG. 1013. — The usual arrangement of the ducts.

The ducts of the pancreas (fig. 1013). The main duct (Wirsung) discharges its contents usually through the duodenal papilla (fig. 961). In 10 per cent of persons the accessory duct (Santorini) becomes the main secretory duct of the pancreas, in which event the bulk of the secretion of the gland is ejected into the duodenum through a superior papilla (fig. 1013).

Innervation of the Pancreas.—Apparently all the pain fibres are limited to the greater splanchnic nerves (T5–T10) (the sympathetic). Parasympathetic fibres reach the gland through the vagus nerves and terminate in the intrinsic pancreatic ganglia.

Surgical Physiology.—The external secretion of the pancreas is controlled by nervous and humoral mechanisms of which the latter is the more important:

(*a*) *Nervous.*—Vagal stimulation evokes a viscid secretion rich in enzymes. Vagotomy, however, does not appear to affect it.

(*b*) *Humoral.*—This mechanism involves two hormones—*secretin* and *pancreozymin*. Both are liberated from the mucous membrane of the duodenum and proximal jejunum chiefly by acid but also by food. The hormones are carried via the bloodstream to the pancreas. *Secretin* alone produces a thin watery alkaline secretion poor in enzymes but rich in sodium bicarbonate. *Pancreozymin*, probably the same hormone as cholecystokinin (Jorpes), evokes a juice rich in enzymes. Gastrin may also play a part.

Pancreatic juice, which contains the enzymes amylase, lipase, trypsinogen, chymotrypsinogen, proelastase, procarboxypeptidase, prophospholipase and ribonuclease, is strongly alkaline. The alkalinity, due to the sodium bicarbonate it contains, is about pH 8.3, and no less than 3 to 4 g sodium is secreted daily in this way. It is therefore not surprising that sodium deficiency develops if the juice is lost to the exterior. The normal adult secretes about 1 to 2 litres of pancreatic juice daily. It contains up to 40 g of protein mainly in the form of these enzymes.

Tests of pancreatic function are described on p. 947.

CONGENITAL ABNORMALITIES

Annular pancreas is due to failure of complete rotation of the ventral segment during development so that a collar of pancreatic tissue surrounds the second part of the duodenum. Usually it does not cause symptoms. In a minority it causes duodenal obstruction (fig. 1014), which is characterised by a typical gas shadow seen on a plain X-ray film, and known as the 'double bubble', viz.: ——————————→

One-third of those with symptoms are infants—the remainder are adults.

Johann Georg Wirsung, d. 1643. Professor of Anatomy, Padua, Italy. (*See footnote p. 908.*)
Giovanni Domenico Santorini, 1681–1737. Professor of Anatomy and Medicine, Venice, Italy.
Erik Jorpes, ?1895–1973. Biochemist, Kemiska Institutionen II, Karolinska Institutet, Stockholm.

Treatment.—Since excision may be followed by pancreatic fistula the usual treatment is duodeno-jejunostomy, anastomosing the jejunum to the dilated first part of the duodenum. Sometimes, in adults, duodeno-duodenostomy is possible and this is ideal.

FIG. 1014.—Annular lobe of the pancreas causing duodenal obstruction.

An ectopic pancreas is found in the submucosa of some part of the stomach, duodenum, or small intestine (including Meckel's diverticulum), the gall-bladder, adjoining the pancreas (e.g. in the hilum of the spleen) or, (in 2 per cent. of carefully conducted necropsies) within the liver. Ectopic pancreas in the wall of the intestine is apt to be the starting-point of an intussusception. Ectopic pancreas in the wall of the stomach is liable to undergo cystic degeneration.

Congenital cystic disease of the pancreas sometimes accompanies congenital disease of the kidneys and liver.

Mucoviscidosis (*syn.* fibrocystic disease) of the pancreas

This condition is but one manifestation of a hereditary congenital abnormality of mucus secretion. Mucoviscidosis affects all mucus-secreting glands, in particular those of the pancreas and bronchioles. Viscid mucus obstructs the pancreatic ducts, and retention of pancreatic secretions results; some of the alveoli rupture, pancreatic enzymes escape and become activated by tissue juice (Bodian). Thus, from birth or before it, the infant suffers from pancreatitis with subsequent fibrosis. Intestinal obstruction, often in the ileum, is another manifestation. The meconium sets into a sticky mass (see below). The secretion of abnormally viscid mucus into the bronchial tree predisposes to respiratory infection. The sweat glands produce sweat containing two or four times more sodium chloride than normal. Thus, this remarkable disease affects not only mucus-secreting glands but all exocrine glands.

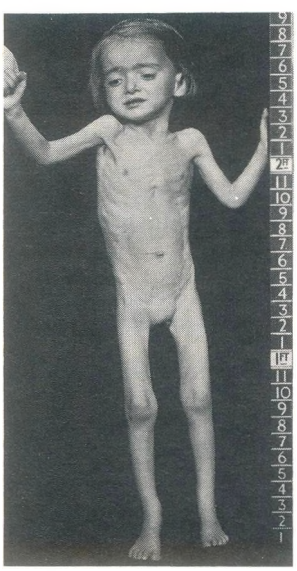


FIG. 1015.—Fibrocystic disease of the pancreas in a girl two years of age. (*Dr. A. P. Norman, London.*)

Clinical Features.—The life of one afflicted with fibrocystic disease of the pancreas is fraught with dangers.

At Birth.—In 10 per cent. of cases the infant is born with meconium intestinal obstruction (p. 1040), or more rarely with meconium peritonitis (p. 975).

During Infancy.—In most cases a few weeks or months after birth bronchiolitis develops. There is dyspnœa, with inspiratory indrawing of the lower chest and the suprasternal notch, and a distressing spasmodic cough. In a number of instances the chest becomes barrel-shaped, and a radiograph shows bronchiectasis. In 90 per cent. of cases steatorrhœa is present, and usually the stools are unduly frequent, greasy, and offensive. In the absence of severe respiratory distress the appetite is voracious, in spite of which the infant is marasmic, except for a distended abdomen.

In Older Children.—Steatorrhœa with wasting (fig. 1015) continues, and is distinguished from cœliac disease by the early onset, the excellent appetite, the almost constant accompaniment of attacks of respiratory infection, and, possibly, by a history of death in infancy from intestinal obstruction or pneumonia of siblings. In hot weather excessive loss of electrolytes in sweat sometimes results in severe dehydration and even death. Cirrhosis of the liver and hypertension frequently develop; indeed, one-third of all cases of portal hypertension at this time of life occur in subjects with this disease. If the patient reaches late childhood without gross pulmonary or hepatic damage, improvement is remarkable and often sustained.

In Adults : (*a*) Middle-aged patients with long-standing pulmonary suppuration and steatorrhœa should always be suspected of suffering from mucoviscidosis.

Johann Friedrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle, Germany.
Martin Bodian, 1910–1963. Pathologist, Hospital for Sick Children, Great Ormond Street, London.

Opacities in the vitreous humour, choroiditis, and bilateral enlargement of the submaxillary salivary glands are frequent accompaniments.

(*b*) Staphylococcal infections are frequent and difficult to eradicate. This organism thrives in a medium containing a high concentration of sodium chloride.

(*c*) The maintenance of fluid balance following any operation on a patient with mucoviscidosis is a severe test of applied physiology. The amount of sodium, potassium, and chloride generally allowed in making up fluid balance charts is so much exceeded (because of loss of these ions in the sweat) as to call for "courage lest we falter."

Confirmatory Tests:

1. The amount of trypsin in the duodenal content is very low or absent.

2. *Sweat Tests:* Neither in health nor in any other disease does sweat contain more than 70 mEq/l (70 mmol/l) sodium, 60 mEq/l (60 mmol/l) chloride, or 20 mEq/l (20 mmol/l) potassium. In 99 per cent. of patients suffering from mucoviscidosis these figures are (often greatly) exceeded. Because of the dangers of heat stroke, the method of sweat collection whereby the patient was encased in a polythene bag has been largely superseded by pilocarpine iontophoresis. An anode is placed on a pad soaked with pilocarpine nitrate on the anterior surface of the forearm and a cathode soaked with a suitable conducting solution is placed on the posterior surface. A current of 4 mA at 15–20 v D.C. is passed for a few minutes. The area under the anode is washed with distilled water and dried. Sweat is then collected on a filter paper placed over the treated area which is covered by a polythene 'bandage'. Analysis for sodium concentration is then performed.

3. The electric conductivity of saliva secreted by healthy persons declines after stimulation. In mucoviscidosis alone the value remains constant, or even increases.

Treatment should aim at controlling respiratory infection, which is nearly always staphylococcal. A major problem is development of resistance by the organisms to one antibiotic after another.

Much can be done for fibrocystic disease of the pancreas by symptomatic treatment. Extra proteins should be given. The sugar intake should be high, but that of starch must be moderate. Fat is curtailed drastically, whereby the frequency, bulk, and foulness of the stools are diminished. Provided this diet is adhered to and enteric-coated capsules of pancreatin, 5 to 10 g are taken before feeds, and 200 units of vitamin D and 10,000 units of vitamin A are given daily, there is nearly always a substantial gain in weight.

Meconium intestinal obstruction is discussed on p. 1040.

Lastly, there should be liberal salting of food. In hot weather extra salt, 2 g per day, should be supplied. The dietetic regimen must be followed throughout life.

INJURIES TO THE PANCREAS

Injuries to the pancreas are rarely seen, because the trauma severe enough to produce this usually kills the patient. Traffic accidents are often the cause; but stabbing is a cause in some countries.

There are two types: (*a*) Laceration, with symptoms due to bleeding, and (*b*) Direct trauma, with symptoms due to the release of enzymes (e.g. high serum amylase). This type may present later as a pseudocyst (p. 950).

Treatment.—Excise the distal part if the duct is divided or repair with drainage.

PANCREATITIS

Classification.—The classification of pancreatitis has always been a problem. Two useful classifications are as follows:

A. **Classification by clinical presentation** (Marseilles[1]):—

1. Acute Pancreatitis	*These return to normal when the primary cause is removed.*
2. Relapsing Acute Pancreatitis	
3. Chronic Pancreatitis	*In these, even if the cause is removed, functional or structural damage still remains.*
4. Relapsing Chronic Pancreatitis	

[1] The Marseilles classification was adopted in 1963 in order to standardise analysis of this condition.

B. **Classification according to ætiology**[1]:—

1. **Biliary Tract Disease**	55%	*Ætiology is very important in relating the natural history of the disease to prognosis, long term treatment, and prophylaxis.*
2. **Alcoholism**	2%	
3. **Post-operative**	5%	
4. **Traumatic**	1%	
5. **Rare causes** (e.g. mumps, hyperparathyroidism, vascular disease)	2%	
6. **Idiopathic**	35%	

ACUTE AND RELAPSING ACUTE PANCREATITIS

Acute pancreatitis is a serious disorder and carries a mortality of 20 per cent. Fortunately only about 5 per 100,000 suffer from it per year. The term relapsing acute pancreatitis does not convey the point that the patient recovers and remains well for months or years between attacks.

Causal theories.—The basic process in acute pancreatitis is one of autodigestion following activation of trypsinogen. There are many hypotheses as to the mechanism of this. Some postulate reflux of bile or of duodenal juice into the pancreatic duct system due to anatomical or mechanical causes, or pressure differences between the pancreatic and biliary system. Others claim to find pancreatic duct obstruction or infection, in addition to the vascular and metabolic conditions mentioned already. It is important to add, however, that evidence is accumulating that the process may start as an intracellular derangement. Recently enzymes have been cited as ætiological factors. Amongst those which have been incriminated are activated phospholipase A and elastase. Viral theories are also gaining popularity and there is some evidence that minor epidemics may occur. There is no evidence that it is an auto-immune disease. None of these theories gives adequate explanation of the disease and the actual cause remains unknown. Hereditary factors appear to be important in a few cases. Drugs have been reported to be responsible, steroids ACTH, diuretics, œstrogen and chemotherapeutic agents being mentioned.

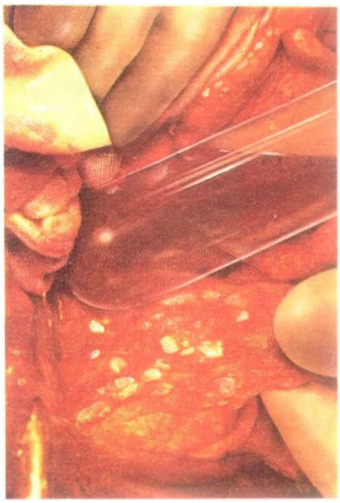


FIG. 1016. — Widespread fat necroses of omentum. A test-tube has been filled with the blood-stained peritoneal fluid. This specimen was rich in amylase. *(G. D. Adhia, F.R.C.S., Bombay.)*

Pathology.—Hitherto, the terms œdematous pancreatitis and hæmorrhagic pancreatitis have been used. These are not really separate conditions, but different stages of one process.

(*a*) **The peritoneal cavity** contains a blood-stained exudate; the omentum and subperitoneal fat are studded with areas of fat necrosis (fig. 1016).

Fat necroses are dull, opaque, yellow-white areas suggestive of drops of wax. They

[1] Based on an analysis of 520 cases (Trapnell). Note the size of the group with no known cause (idiopathic).

John Eliot Trapnell, Contemporary. Surgeon, Royal Victoria Hospital, Bournemouth, England.

are most abundant in the vicinity of the pancreas, but are widespread in the greater omentum and the mesentery. At necropsy they can sometimes be demonstrated beneath the pleura and pericardium, and even in the subsynovial fat of the knee joint. Fat necroses consist of small islands of saponification caused by the liberation of lipase, which splits fat into glycerol and fatty acids. Free fatty acids combine with calcium to form soaps = fat necrosis.

(*b*) **The Pancreas.**—In the mildest form the whole or part of the organ is œdematous, but if severe, it may be grossly hæmorrhagic and sometimes necrotic. The retroperitoneal tissues around the pancreas may then be engorged with blood-stained fluid.

(*c*) **Associated Pathology.**—Gall-stones and infected bile are frequently found. When the lesser sac has been drained and the patient survives, portions of necrotic, pancreatic, and peri-pancreatic tissue may be discharged through the wound as putty-like material. On analysis this material has been shown to be composed largely of calcium stearate.

Clinical Features.—Unlike acute cholecystitis, acute pancreatitis shows no predilection for obese persons, no sex discrimination, and while the age of the patient is extraordinarily variable, the maximum incidence is between fifty and sixty years. The salient features of acute pancreatitis are as below, but it must be pointed out that in the early stages there may be little in the way of physical signs (tenderness and guarding) in contrast to the severity of the symptoms. This state of affairs should suggest the diagnosis.

Onset.—The attacks come on at any time but frequently follow the ingestion of a heavy meal and/or alcohol.

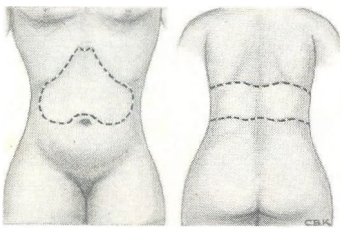


FIG. 1017.—The usual distribution of the pain of acute pancreatitis.

Pain is agonising, mainly epigastric, radiates to the back (fig. 1017) and slowly rises to a crescendo over several hours (in contradistinction to the pain of perforation). Moynihan described it as 'illimitable agony'.

Vomiting and retching, repeated and noisy, are a marked feature.

Rigidity, like pain, develops slowly and may become generalised.

Tenderness may be elicited by firm and deep palpation over the pancreas and occasionally in the left renal angle where the tail of the pancreas is comparatively near the surface.

Profound shock as a *presenting* symptom is uncommon (10 per cent. of cases), but when it occurs the patient is sweating and collapsed, the nose is cold, and the blood-pressure depressed. This feature calls for urgent infusion of plasma. The shock may be due partly to circulating 'kinins' which are formed by the action of trypsin on the plasma proteins.

Cyanosis.—Cyanosis is not as common as currently believed.

Faint jaundice may sometimes be seen especially during the second day. Probably it is obstructive, and due to œdema of the head of the pancreas.

Ileus in the early stages is limited to the duodenum and proximal jejunum. The gas-filled solitary loop may show on plain X-ray. Unless a severe paralytic ileus is prevented by gastro-intestinal aspiration, peristalsis diminishes after twelve hours and abdominal distension supervenes.

A tender, palpable mass may appear in the epigastrium towards the end of

Berkeley George Andrew Moynihan (Lord Moynihan of Leeds), 1865–1936. Professor of Clinical Surgery, Leeds, England.

the second week. This is usually a pseudo-cyst (p. 950). If the mass appears later—after the third week—it is due to abscess formation.

Discoloration of the skin is a rare manifestation of acute pancreatitis seen in cases of two or three days' standing. Grey Turner first described it in the loins, and likened it in appearance to that of late extravasation of urine. Cullen described a similar discoloration around the umbilicus. The cause of the phenomenon is the action on the subcutaneous fat of pancreatic ferments that have escaped from the retroperitoneal tissues, either directly or via the round ligament of the liver.

Laboratory and Other Investigations: (fig. 1018).

Leucocyte Count.—A leucocytosis above 10,000 per cu mm (10×10^9/l) occurs frequently, and it may rise as high as 30,000 (30×10^9l).

The Serum-amylase Estimation.—The normal value is 80 to 150 Somogyi units; 400 units is suggestive of abnormality. A level of 1,000 units is the minimum necessary to support a diagnosis of acute pancreatitis. The highest level is attained within an hour after the onset of symptoms. Subsequently there is a fall to normal level within 3–5 days.

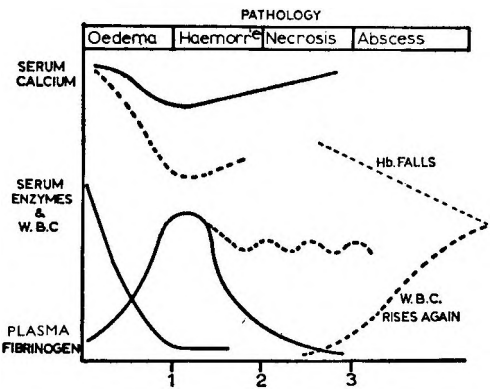


FIG. 1018.—Relation of the natural history of acute pancreatitis to laboratory investigations. 1, 2, 3 = weeks. The broken lines are intended to show the variations from normal in complicated cases and indicate a worsened prognosis associated with the appearance of methæmalbumen. (*John Trapnell F.R.C.S., Bournemouth.*)

Amylase: creatinine clearance ratio (abbreviated to Cam/Ccr or ACCR). This simply involves taking simultaneous blood and urine samples, measuring amylase and creatinine in each, and calculating the Cam/Ccr from the expression:—

$$\frac{\text{urine amylase}}{\text{serum amylase}} \times \frac{\text{serum creatinine}}{\text{urine creatinine}} \times 100$$

A normal value is less than 3 per cent; the mean value in acute pancreatitis is about 6 per cent.

The diastatic index of the urine is normal (below 50 units) during the early hours of the attack. After twelve to twenty-four hours it often rises to 100 units or more; sometimes it is as high as 500 and, rarely, 1,000 units.

Glycosuria may be present.

The serum calcium (normal—10 mg/100 ml (2·5 mmol/l)) undergoes a delayed fall about 5–8 days after the attack. Concentrations below 7 mg/100 ml (1·75 mmol/l) are often followed by the death of the patient, in spite of the administration of calcium. The test is of some prognostic value as is the appearance of tetany. The serum calcium level may be an indication of the amount of calcium fixed in the formation of calcium soaps.

The serum bilirubin (normal—not exceeding 0·2 mg/100 ml (3·4 mmol/l)) is nearly always raised, even when no icteric tint of the sclera is apparent.

The plasma fibrinogen is raised at the end of the first week and should return to normal in the third. This is of considerable prognostic value (fig. 1018).

George Grey Turner, 1877–1951. Professor of Surgery, Postgraduate Medical School, London.
Thomas Stephen Cullen, 1868–1953. Professor of Gynæcology, Johns Hopkins University, Baltimore, U.S.A.
Michael Somogyi, 1883–1971. Biochemist, Jewish Hospital, St. Louis, Missouri, U.S.A.

Methæmalbumen—the presence of this in the blood indicates a hæmorrhagic necrosis and is associated with a bad prognosis.

Electrocardiographic Changes.—It is by no means uncommon to find abnormal electrocardiographic recordings in patients with acute pancreatitis; in a few the record is so abnormal as to support a provisional diagnosis of myocardial infarction. These electrocardiographic changes disappear with recovery of the patient, but may return during subsequent attacks (Pollock).

Radiography.—A gas shadow and fluid level is seen in the region of the duodeno-jejunal junction. Curiously, this is most often clearly seen *after* the diagnosis has been made.

Differential Diagnosis.—The most important point in diagnosis is to remember it as a possible cause in every case of upper abdominal pain and to request a serum amylase. Coronary occlusion, high intestinal obstruction, perforated peptic ulcer, leaking dissecting aortic aneurysm, and gall-stone colic are the conditions that may have similar features. The absence of gas beneath the diaphragm helps to exclude a perforated peptic ulcer.

Abdominal pain with raised serum amylase

The significance of a serum amylase above 1,000 units associated with abdominal pain is of great importance. The following conditions should be considered:

1. *Afferent loop obstruction* following gastrectomy (p. 832). Back pressure in the distended loop up the pancreatic duct is presumed to raise the serum amylase (sometimes up to 3,000 units or more).
2. *Absorption through the peritoneum* as in biliary peritonitis—due to a perforated duodenal ulcer, leaking duodenal stump, a leak following biliary surgery or perforation of the gall bladder.
3. *Damage to the Pancreas or its blood supply*—as in direct trauma, thrombosis of the aorta or the inferior vena cava or a ruptured aortic aneurysm.
4. *Spasm* at the sphincter of Oddi.

Raised serum amylase has also been reported in ectopic gestation and after head injuries.

Treatment of Acute Pancreatitis.—This should be conservative providing that there is no doubt about the diagnosis. Contrary to past teaching, if there is any doubt it is better to perform a laparotomy and see if there is pancreatitis. This procedure may save a patient's life if, for example, he has a perforated peptic ulcer, a leaking aneurysm, a stone in the common bile duct, or a mesenteric thrombosis.

1. **Conservative treatment** is as follows:—*Relief of Pain and Rest for the Pancreas.*—Morphine or Pethidine[1] should *not* be used alone as they cause spasm of the sphincter of Oddi (Boulter). Hormonal stimulation (secretin and pancreozymin) is best subdued by continuous and efficient gastric suction and nothing must be given by mouth. These measures, by resting the pancreas, contribute greatly to the relief of pain.

Replacement of Fluid and Electrolytes.—The patient needs large quantities of fluid (saline, plasma and blood). Rapid correction is urgent and life-saving. In bad cases a central venous pressure monitoring catheter should be introduced (p. 73). Calcium gluconate 10 ml. of a 10 per cent. solution i.v. should be given daily.

Antibiotics.—Large doses of an antibiotic such as a tetracycline should be used to prevent secondary infection.

[1] Pethidine is known as demerol in Canada, and as demerol hydrochloride in the U.S.A.

Alan Victor Pollock, Contemporary. Surgeon, Scarborough Hospital, Scarborough, Yorkshire, England.
Ruggero Oddi, 1845–1906. Physiologist, Perugia, Italy.
Patrick Stewart Boulter, Contemporary. Surgeon, Royal Surrey County Hospital, Guildford, Surrey, England.

Other therapy.—*Low-molecular weight* (40,000) *Dextran*, i.v. has been shown to be helpful (p. 81), and besides maintaining proper perfusion and oxygenation of the pancreas it has similar effects upon such vital organs as the kidneys and the adrenals which can be damaged by vasoconstriction (p. 81).

Hydrocortisone, 100 mg. i.v. is used in repeated doses for persistent hypotension.

Dorso-lumbar paravertebral block (cf. p. 143) with local anæsthetic interrupts the afferent pain fibres of the sympathetic splanchnics and has long been advocated.

Trasylol (aprotinin—an antitryptic substance) i.v. is not proven as an effective treatment.

Glucagon which tends to inhibit pancreatic secretion has been said to improve prognosis if given early in the disease, in a dosage of 1 mg i.v. stat and 1–1·5 mg four-hourly for three days. Recent evidence is not encouraging.

Closed Peritoneal Lavage may be helpful in severe cases.

2. **Procedure if the diagnosis is made at laparotomy.**—The pancreas must not be manipulated in any way, but an assessment can be made of the extent of the condition. If, and only if, there is evidence of gallstones, or there is jaundice, the gall-bladder or the common bile duct may be drained (cholecystostomy, p. 927, or choledochotomy, p. 930). The latter procedure is rarely feasible on account of the extensive bile-stained œdema and hæmorrhagic extravasation. No attempt should be made to manipulate anywhere near the ampulla of Vater.

Nothing else is done and the abdomen is closed with non-absorbable sutures.

Complications of Acute Pancreatitis.—While the treatment of acute pancreatitis is preferably non-operative, the treatment of most of its complications requires surgical intervention. In epitome the complications are:

1. *The development of a pseudo-cyst* occurs in about 12 per cent. of cases (p. 950).
2. *Abscess formation* (rare), which gives rise to a swelling most commonly in the left flank, a swinging temperature (sometimes masked by antibiotics), and a raised plasma fibrinogen level (fig. 1018). An abscess should be drained extraperitoneally.
3. *A collection of blood in the lesser sac* (rare), characterised by the very early and sudden appearance of an epigastric swelling, is best treated by evacuation of the clot —a measure which slightly improves the very poor prognosis.
4. *Hæmatemesis and melæna* is uncommon and carries a bad prognosis.
5. *Renal Failure.*
6. *Toxic Psychosis.*

Convalescence and Subsequent Management.—The diet should be bland, consisting essentially of small frequent meals avoiding fried foods, spices, and thick soups. Alcohol is forbidden. Food is best eaten dry, fluids being taken between meals. An antispasmodic, i.e. tinct. belladonna, just before meals may be beneficial. Some patients derive considerable benefit from the regular use of the slow-release glyceryl-trinitrate preparation 'Sustac'. By far the most important feature of convalescence is to determine those cases likely to have recurrences, in particular those with associated biliary disease. I.V. cholangiography is mandatory but should be delayed for a month. In those cases with stones, cholecystectomy and choledocholithotomy should be done as a safeguard against recurrence. If biliary tract disease is fully eradicated the attacks should cease. In the other cases ætiological factors should be attended to where possible (e.g. the alcoholic should stop drinking, a parathyroid tumour should be removed). *The prognosis* with regard to recurrence is now clearer. Of the idiopathic group, 20 per cent. have another attack within 1–2 years of the first attack and it is usually milder than the initial episode. Death from a recurrence is very rare. If a patient lives 6 years without recurrence he may be considered 'cured'. In general, however, acute pancreatitis or relapsing acute pancreatitis does not proceed to chronic pancreatitis.

Post-operative acute pancreatitis.—In this condition, the mortality is greater than 50 per cent. It occurs most commonly after operations on the biliary tree or

Abraham Vater, 1684–1751. Professor of Anatomy and Botany, Wittenberg, Germany.

stomach. Manipulation in and around the sphincter of Oddi such as E.R.C.P. (p. 957) or the insertion of long-arm T-tubes is sometimes a cause of this condition. The essential treatment lies in early diagnosis and conservative management as described.

CHRONIC AND RELAPSING CHRONIC PANCREATITIS

These conditions are separate entities from acute pancreatitis. The essential change in the pancreas is slowly progressive with destruction of acini, increasing fibrosis, and often with corresponding functional failure. In some, there may be calcification in the gland—this is seen especially if there is a background of alcoholism or where hereditary or endocrine factors are prominent.

In *chronic pancreatitis* there are five main types of presentation:

(1) Upper abdominal *pain* passing through to the back (p. 942). (2) As a cause of obstructive *jaundice*. (3) As a *malabsorption* disorder—i.e. failure of external secretion, with steatorrhœa, weight loss, and vitamin deficiencies. (4) As *diabetes*—i.e. failure of internal secretion. (5) Sometimes the diagnosis is made solely by palpation of the pancreas during *laparotomy*. The features may include nodularity, irregularity, fixity, induration, or the presence of a mass. It is unwise to make the diagnosis on nodularity alone (the normal pancreas may be nodular when it is turgid with secretion).

Age, Sex, and Habitude.—Unlike cholecystitis, chronic pancreatitis occurs much more frequently among men than among women. There is no predilection for fat persons, and the age incidence, while having a more restricted ambit, is not dissimilar from that of carcinoma of the pancreas.

The hall-mark of *relapsing chronic pancreatitis* is recurrent attacks of *pain* in the epigastrium and in the back which may exceptionally last for only an hour but usually continue for two or three days. As opposed to recurrent acute pancreatitis the patients are not well between the attacks.

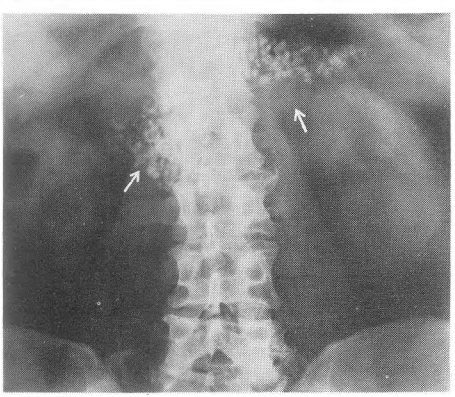


FIG. 1019.—Diffuse pancreatic calcinosis.

Periodicity.—The attacks vary in frequency; they may occur as often as once a week or as seldom as three times a year. As a rule, with the passage of time the attacks become more frequent.

Vomiting occurs in some 50 per cent. of cases.

Weakness or fatigue is common, and it is not unusual for this to herald an oncoming attack by two or three days.

Jaundice is exceptional in under 15 per cent. of cases. In the more advanced cases it is the result of pressure upon the terminal part of the common bile duct.

Atherosclerosis and chronic relapsing pancreatitis often go hand in hand (Saint).

Alcoholism or drug addiction is so often present (40 to 50 per cent.) that either is to be

Eric Galton Saint, Contemporary. Professor of Medicine, University of Western Australia, Perth.

regarded as important in diagnosis. The habit is often acquired in an endeavour to seek relief from the intolerable pain.

Radiography.—It is important to have a plain film before any barium series to ascertain whether there is any pancreatic calcification (fig. 1024). When this is present it is usually para- or intra-ductal. The barium series may show widening and indentation of the duodenal circuit. Routine cholecystography should always be done. If the gall-bladder has been removed, an intravenous cholangiogram is necessary. E.R.C.P. may be diagnostic (p. 957 and fig. 1045).

Special Tests of Function

Serum Amylase.[1]—This may be of use in the relapsing cases. It is increased during the attack. The elevation may not be great but it must be related to the base-line level obtained between attacks.

Stools.—Steatorrhœa is present when chemical examination (to measure the fatty acids) shows the fat content to be greater than 18 g in three days.

The stools become bulky, pale, frothy, offensive, and float on water. Malabsorption occurs late in the disease and only appears when some 80 to 90 per cent. of the gland has been destroyed. Having recognised the presence of steatorrhœa, pancreatic disease can only be identified for certainty by duodenal intubation tests to determine the presence or absence of pancreatic enzymes.

Duodenal Intubation (Howat)—*The Secretin-Pancreozymin Test.*

Pancreatic secretory capacity can be measured by collecting duodenal contents quantitatively after stimulating the pancreas with secretin and pancreozymin (1 unit/kg body wt)—i.v. A double-lumen radio-opaque tube (Dreiling) is passed and so sited

	Volume (ml)		HCO_3^- (mmol)		Amylase (Units × 1000)		Lipase (Units)	
	Mean	Lower Limit	Mean	Lower Limit	Mean	Lower Limit	Mean	Lower Limit
Post Secretin (30 minutes)	111	48	6·1	1·7	49·6	13·8	1427	436
Post Pancreozymin (30 minutes)	141	51	7·5	1·7	65·4	10·3	2133	631
Post Secretin and Post Pancreozymin (60 minutes)	252	124	13·6	5·4	114·9	23·5	3560	1552

FIG. 1020.—Normal Values for Exocrine Pancreatic Function Studies (Boot's Secretin and Pancreozymin 1 unit/kg i.v.) Amylase in Somogyi units per litre; lipase measured by the method of Cherry and Randel.

Volume	Bicarbonate	Enzyme	Diagnosis
Low	Low	Low	Carcinoma of the head of pancreas
Low	Normal	Normal	Carcinoma of the pancreas
Normal	Low	Normal or low	Chronic pancreatitis
Normal	Normal	Low	Nutritional or *metabolic* pancreatic fibrosis
Increased	Low	Normal	Hæmachromatosis

FIG. 1021.—Diagnostic Significance of Altered Exocrine Pancreatic Function.

[1] The test is now in tablet form (Phadebas® Amylase test—Pharmacia Ltd.).

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David A. Dreiling, Contemporary. Associate Professor of Surgery, Mount Sinai Hospital, New York.

that one lumen drains the stomach, the other the duodenum; in this way duodenal contents can be collected free of gastric contamination. Continuous suction with negative pressure not greater than 50 mmHg (6·7 kPa) is applied and the duodenal contents collected in fractions. After requisite control periods, secretin is given and the duodenal contents collected in 10-minute fractions for 30 minutes. Then pancreozymin is given and the collection is continued for a further 30 minutes. Normal values are shown in fig. 1020.

The diagnostic help which can be obtained is summarised in fig. 1021.

The feature which distinguishes chronic pancreatitis from an attack of relapsing pancreatitis in which restitution to normal takes place, is a permanently diminished concentration and output of bicarbonate after secretin. The output of enzymes after pancreozymin is a more sensitive index of pancreatic damage than a reduction of bicarbonate, particularly in carcinoma of the head of the pancreas. Since pancreozymin is probably identical with cholecystokinin, which contracts the gall-bladder, the secretin-pancreozymin test in early obstructive jaundice is a useful guide to the site of the primary obstruction either in the pancreatic or bile ducts. Impaired tests are always found in steatorrhœa of pancreatic origin. The duodenal contents obtained in these tests are particularly suited to examination for exfoliated carcinoma cells in both biliary duct and pancreatic growths (Triple test (p. 957)).

Lundh Test.—The trypsin activity in the duodenum is measured after a test meal (consisting of corn oil 18 g, Casilan 15 g, glucose 40 g, syrup 15 g and hot water to 300 ml.). The duodenal juice is collected for 2 hours after the meal by syphonage or gentle suction through a carefully positioned tube. The tryptic *activity* is normally more than 9 μEq/min/ml, but it is less than 2 μEq/min/ml in pancreatic steatorrhœa (Lennard-Jones).

Glucose Tolerance Test.—With progressive pancreatitis, glycosuria appears and a diabetic type of blood sugar curve is obtained. This is in contradistinction to intestinal causes of malabsorption, e.g. idiopathic steatorrhœa, which give flat curves.

Occult blood in stools is usually absent in chronic pancreatitis, but may be present in carcinoma of the pancreas (p. 954).

Differential Diagnosis from Carcinoma of Pancreas.—It can be difficult to be certain about this: The duodenal intubation tests or E.R.C.P. give a diagnostic accuracy in the region of 90%. If a swelling found at laparotomy is confined to the pancreas alone, then it is still impossible to be certain. Biopsy is of limited value and it may be dangerous. Birnstingl recorded ten cases with obstructive jaundice declared on pancreatic biopsy to be chronic pancreatitis, yet all were dead with carcinoma of the pancreas within three years. Chronic pancreatitis predisposes to the development of carcinoma in the organ.

Prognosis.—A patient is unlikely to die of chronic pancreatitis, or even from one of the complications of this disease such as steatorrhœa, diabetes mellitus, or gastro-intestinal hæmorrhage, but is likely to succumb eventually to some unrelated disorder such as cerebrovascular disease or to cancer developing in the organ itself. In late onset diabetes cancer of the pancreas should always be suspected and the appropriate investigations undertaken.

Treatment

In general those with diabetes and malabsorption phenomena (i.e. chronic pancreatitis) are best treated conservatively and rarely need surgery. When, however, much pain or jaundice is present surgery is usually necessary.

All cases need a high-protein, high-calorie diet. Sustac (p. 945) should be tried. Alcohol is completely forbidden. A concentrated triple-strength

Göran Lundh, Contemporary. Department of Surgery, University of Lund, Lund, Sweden.
John Edward Lennard-Jones, Contemporary. Professor of Gastro-enterology, London Hospital, London.
Martin Avigdor Birnstingl, Contemporary. Surgeon, St. Bartholomew's Hospital, London.

enteric-coated tablet of pancreatic enzyme is given with each meal, together with a multivitamin capsule. Anæmia should be corrected.

Operations for Pain.—This may be so severe that it leads to drug addiction. The variety of procedures recommended is an index of the unsatisfactory treatment of this condition:—

Biliary Surgery.—*Pathology in the biliary tree* must be thoroughly eliminated.

Operative Pancreatography.—The planning of an appropriate operation depends upon the state of the main duct of Wirsung, as demonstrated by a contrast X-ray if this has not already been outlined by endoscopic cannulation (p. 957). Two ml. of 60% diatrizoate (Hypaque) are injected into the duct by a needle transduodenally. If the duct is dilated above a stricture at the ampulla a sphincterotomy is indicated. If the duct is stenosed over a distance, partial excision and drainage of the remnant into a Roux loop is done.

Transduodenal Sphincterotomy.—The second part of the duodenum and the head of the pancreas are mobilised and the region of the duodenal papilla is located by palpation. A transverse incision is then made through the duodenal wall exactly opposite the papilla. A grooved director is insinuated into the orifice and passed up into the common bile duct. The sphincter is now divided with the knife in the groove (at 12 o'clock). The full thickness of the duodenal wall should not be severed (see page 931).

Sphincterotomy performed in this way gives free access to the terminal portions of both the common bile duct and the duct of Wirsung only. The duodenum is closed meticulously. The subhepatic space is drained.

Partial Excision and Drainage—Retrograde Pancreato-jejunostomy.—If the major pancreatic duct is strictured proximal to the sphincter the tail of the pancreas is mobilised and amputated obliquely. The duct is located and slit for a short distance, the cut edges being held apart by sutures. Any calculi are removed. The divided distal end of the jejunum is then brought over the stump of pancreas like the finger of a glove, and sutured into place. The operation is completed as shown in fig. 1022.

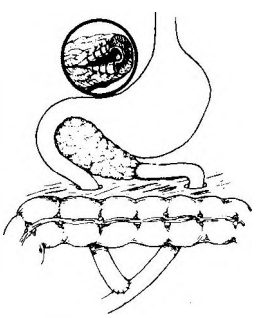


FIG. 1022.—Retrograde pancreatojejunostomy. (*After M. K. Du Val, Jnr.*)

Where these changes are extensive the duct is dilated and contains multiple stones. The body can be 'filletted' and anastomosed to a Roux loop of jejunum (the Puestow operation).

Interruption of Nerve Pathways—Splanchnicectomy—Resection of 3 cm of the right great splanchnic nerve may be used in the treatment of intractable pain, but the long-term results are uncertain.

By and large, if any of these operations is undertaken, a good result may be expected in about half the cases. Splanchnic block may also, on occasion, prove useful.

For Jaundice.—If this is the presenting feature, biliary drainage is essential.

Cholecyst-jejunostomy-en-Y (fig. 1023) is the oldest operation for this condition, and, for a patient in poor condition, is inferior to none, provided the gall-bladder is thin-walled and distended with bile (i.e. that there is no obstruction to the cystic duct). Simple cholecyst-jejunostomy with entero-enterostomy is equally efficacious. This stoma must be at least 3 cm.

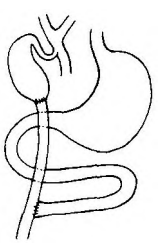


FIG. 1023.—Cholecyst-jejunostomy-en-Y (Roux).

PANCREATIC CALCULI

These are intraductal and consist of calcium carbonate. The usual picture is one of multiple radio-opaque stones. Parathyroid disease should be excluded. They are the *result* of the disease and not the cause—but if surgery is performed on the pancreatic duct (see above), the stones should be removed. Surgery is *not* mandatory for stones alone.

PANCREATIC CYSTS — Pseudo-cyst 80%. True 20%.

Charles B. Puestow, Contemporary. Professor of Surgery Emeritus, The Abraham Lincoln School of Medicine, University of Illinois, U.S.A.

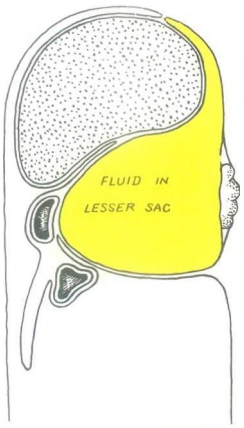


FIG. 1024.—Pseudo-pancreatic cyst.

Pseudo-cyst of the Pancreas.—This is essentially a collection of fluid in the lesser sac (fig. 1024) and occurs most frequently as a result of acute pancreatitis, or sometimes after injury. In order of frequency the pseudo-cyst projects (*a*) between the stomach and the transverse colon; (*b*) between the stomach and the liver; (*c*) behind or below the transverse colon.

Clinical Features.—An epigastric swelling appears during the conservative treatment of pancreatitis, or in traumatic cases during or after convalescence for an injury that did not merit laparotomy. Exceptionally the patient is admitted with a pseudo-cyst of the pancreas and gives a history of an attack of severe abdominal pain that occurred a week or more previously.

On examination there is a swelling, sometimes as large as a melon, placed centrally above the umbilicus: the swelling is fixed, and in many instances it is so tense that fluctuation cannot be elicited. As a rule, transmitted pulsation from the abdominal aorta is very noticeable and the diagnosis of an aneurysm may be considered, although in the latter case the pulsation is expansile and, in the knee-elbow position the pulsation is much diminished. Moreover, in the case of a pancreatic cyst, a bruit is absent and the swelling, which is the presenting feature, is much broader. A Ryle's tube passed into the stomach may be palpable over the swelling if the patient is thin (Baid). Mesenteric cysts are rare and very mobile. A barium meal shows projection of the cyst into hollow viscera, especially the stomach (fig. 1025). Every case of acute pancreatitis must be observed for the development of a pseudo-cyst. With this in mind, the width of the vertebrogastric interval as seen on lateral X-ray should be assessed on about the tenth day of the disease. Ultrasound and CT scanning[1] (p. 961) are proving valuable diagnostic advances. The earlier a pseudo-cyst appears the worse the prognosis.

Treatment.—It is necessary to distinguish between acute and chronic pseudocyst formation. Acute pseudocysts commonly arise after severe attacks of pancreatitis. About 50% of these will resolve without treatment and so no action should be taken during the first weeks after they have been recognised unless there is clinical evidence that the cyst has become infected. Serial ultrasonic measurements will give a good indication of how the cyst is progressing and should show spontaneous resolution if this is occurring. Where, however, infection has supervened or when the cyst has been present for 2–3 months or is over 6 cm in diameter, operative treatment is recommended because of the risk to life from spontaneous rupture. The treatment is cysto-gastrostomy. The stomach is opened through its anterior wall and an incision of at least 6 cm made through the posterior wall into the cyst. Hæmostasis is secured with a circumferential running suture and the anterior wall of the stomach is closed (fig. 1026). The contents of the cyst then drain into the stomach. Convalescence is shortened thereby and the complications referred to are obviated. Curiously, food does not enter the sac, as might be expected. The opening must be large enough to allow for subsequent contraction. Bleeding from the

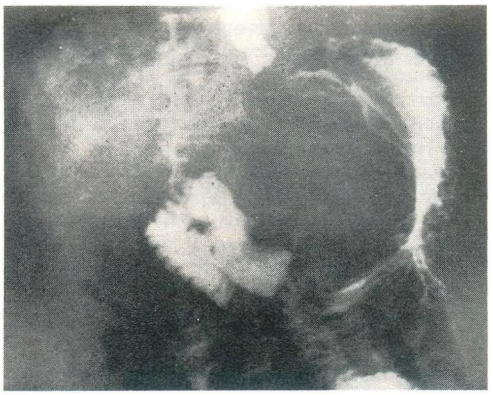


FIG. 1025.—Barium meal—Pseudo-pancreatic cyst displacing stomach. (*Lord Smith, London, in* Trans. med. Soc. Lond.)

[1] Originated in 1968 in the EMI Central Research Laboratories.

J. C. Baid, M.S., Contemporary. Surgeon, Jodhpur, India.

incised edge may follow and this is the major post-operative hazard. Cysto-jejunostomy is equally effective.

True Cysts of the Pancreas.—In 20 per cent. of cases a cyst of the pancreas is a true cyst, and falls into one of the following categories :

(*a*) *Cyst-adenoma.*—Most true cysts belong to this group. Paradoxically these usually present as a solid swelling. The cysts are multiple and lie in an adenomatous mass. They may move on respiration and often have a narrow attachment to the pancreas. Removal is therefore accomplished without undue difficulty.

(*b*) *Mucoviscidosis of the Pancreas* (p. 939).

(*c*) *Congenital Cystic Disease.*—Possibly associated with polycystic disease of the kidneys and liver.

(*d*) *Retention Cyst.*—This is part of the pathological change in chronic pancreatitis and is due to ductal obstruction.

(*e*) *Hydatid Cyst.*—The ubiquitous hydatid completes the list.

Clinical Features. — Epigastric discomfort and the presence of a swelling are the usual reasons that cause the patient to seek advice. When the swelling arises in the tail of the pancreas it may be impossible to differentiate it from a renal lesion until pyelography has been performed. When the cyst arises in the head of the pancreas, the symptoms are identical with those of a choledochal cyst (p. 915).

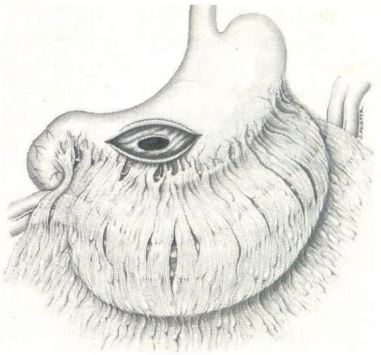


FIG. 1026.—Cysto-gastrostomy for pseudo-pancreatic cyst.

Treatment.—A comparatively small cyst can be treated expectantly. A large cyst of the head of the pancreas is suitable for cystoduodenostomy. A similar cyst of the body of the organ can be treated by cysto-gastrostomy. When the cyst (or cysts) are situated towards the tail of the pancreas (this is the site of election for cystadenoma) the affected portion of the pancreas can be amputated.

PANCREATIC FISTULA

This may be external or internal.

External Fistula.—This usually follows trauma to the gland either by closed external injury or operations for splenectomy or rarely gastrectomy or open biopsy. In most cases following operation, the injury was not suspected at the time.

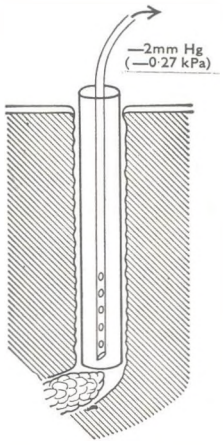


FIG. 1027.—Continuous suction sump drainage for pancreatic fistula.

Treatment.—The problems in the management of these fistulæ are:

(1) *Fluid and Electrolyte Loss.*—In the presence of these fistula the pancreas tends to hypersecretion and up to 2 litres may be lost in twenty-four hours. Such a loss of fluid and electrolytes may constitute a menace to life; they must therefore be replaced either by mouth or intravenously. In large losses, daily fluid and electrolyte balance must be maintained. If there is much loss, pancreatic extract should be given by mouth. Pancreatic secretion can be partially suppressed by diamox and atropine. The diet should consist of high protein and low carbohydrate.

(2) *Autodigestion of the Parietes.*—The skin should be protected by an ointment composed of 1 per cent. HCl in zinc oxide cream.

(3) '*Pooling*' *of the discharge* deeply in the wound. If possible, completely *dependent* drainage must be instituted; if necessary, the patient must lie prone. If this is not possible, continuous motor suction sump drainage must be carried out (fig. 1027). In any case, the daily loss must be measured.

Spontaneous closure occurs in 90 per cent. of cases,

though this may take several months. Failure is usually due to inadequate dependent drainage.

If in spite of all these measures the fistula fails to close, it should be dissected to its source in the pancreas and drained into the stomach or into a Roux-en-Y jejunal loop.

Internal Fistula.—This is often associated with chronic pancreatitis, pancreatic ascites or pleural effusion may result. Diagnosis is made by finding a high serum amylase level in the aspirate. The conservative regime as described above may cure half the patients, but nasogastric suction must be included. Surgery is necessary for the remainder, either resection or Roux-en-Y drainage may be indicated.

ISLET-CELL (HORMONE SECRETING) TUMOURS OF THE PANCREAS

The islets of Langerhans contain three types of cells: alpha, beta, and gamma, and of these the beta-cell is most common.

Pathology.—The actual islet-cell lesion may be one of the following:

(1) Generalised hyperplasia; (2) discrete adenomas; (3) generalised adenomatosis; (4) carcinomas. Two-thirds of islet lesions occur in the body or tail of the pancreas. Approximately in one-third of cases they are multiple and in one-third they are malignant.

Such lesions may be associated with similar states in other endocrine glands, especially the anterior pituitary, the parathyroids, and the adrenal cortex (multiple endocrine adenomatosis).

The condition is usually familial. Such cells are now believed to comprise an APUD cell system (Amine Precursor Uptake and Decarboxylation). An apudoma is a tumour derived from these cells. It is neuroectodermal in type and secretes its normal peptide hormone, or variants, and sometimes others as well. Gastrinoma, Vipoma, Glucagonoma, Insulinoma, Residioblastoma, Somatostatinoma, and Pancreatic Polypepidoma are described. All have similar morphologic characteristics but can be distinguished by radioimmunoassay.

Clinically, in overt cases, there may be: (1) Hyperinsulinism. (2) Features of other endocrine lesions, e.g. enlarged sella turcica, altered phosphorus and calcium blood levels, and increased keto-steroids in the urine. (3) Peptic ulceration. (4) Zollinger-Ellison syndrome (p. 953).

Of these it is only possible here to discuss (1) and (4).

HYPERINSULINISM (BETA-CELL TUMOUR). INSULINOMA (fig. 1028)

Clinical features are not stereotyped, and unless the attending clinician is alive to the bizarre symptoms to which it gives rise, insulinoma of the pancreas, still somewhat of an enigma, will often continue to be displayed only at that final Court of Appeal—the post-mortem room, for so often the patient is diagnosed as psychoneurotic, epileptic, a malingerer, or a sufferer from organic nervous disease.

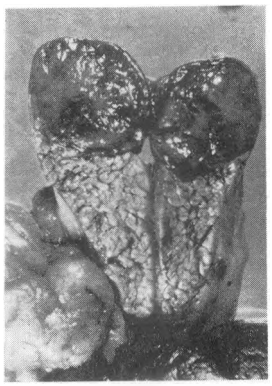


FIG. 1028.—Beta-cell islet tumour of the pancreas.

The attacks, due to hypoglycæmia, come on at irregular intervals and often with progressively increasing frequency and severity. Four stages are recognised, and in the beginning the attacks do not necessarily progress beyond the first or second stages.

Stage 1.—Often the symptoms simulate duodenal ulcer, awakening the patient in the early hours of the morning with vague abdominal discomfort, relieved by carbohydrates. At other times nervous symptoms predominate. There is a vague feeling of being unwell, a disinclination for exertion, and possibly some disorientation.

Stage 2.—Trembling, sweating, dizziness, blurring of vision, and great hunger.

Stage 3.—Sluggish mind, inarticulate speech, incoordinated movements, diplopia, sometimes hallucinations.

Stage 4.—Fits indistinguishable from epilepsy[1] passing into semi-consciousness or coma, with dilated pupils

[1] The brain cannot store dextrose, which it requires in large amounts. Deprivation of this essential brings about a burst of nervous energy; the higher centres are affected first.

Paul Langerhans, 1847–1888. Professor of Pathological Anatomy, Freiburg, Germany.

and extreme spasticity. In course of time intellectual deterioration is liable to follow.

Usually the patient is an adult under forty years of age, but a few cases have occurred in children. Because eating relieves the symptoms, over-weight not infrequently results. Pain is not a feature of benign insulinoma, but is present in most cases where a malignant change has occurred.

In a few instances the tumour contains sufficient calcium to show in a radiograph.

Diagnosis.—The three criteria (Whipple's triad), which, if present, suggest the diagnosis, are:

1. An attack, as described above, occurs in the fasting state (i.e. in the morning) or with exercise.
2. During the height of the attack there is a hypoglycæmia below 45 mg per 100 ml (2·5 mmol/l) of blood.
3. The symptoms are relieved by glucose.

Confirmation depends on the identification of fasting hypoglycæmia (fig. 1029), associated with elevated human insulin levels as measured by radioimmunoassay. Further confirmation may be sought by more elaborate insulin stimulation and suppression tests.

Treatment.—The only curative treatment is extirpation of the tumour or tumours. An intravenous drip infusion of isotonic dextrose solution is started, laparotomy is performed and the pancreas is displayed. When the tumour (fig. 1030) is not located

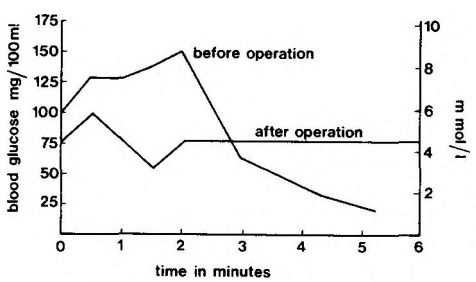


FIG. 1029. — The hypoglycæmia caused by the islet-cell tumour is cured by operation.

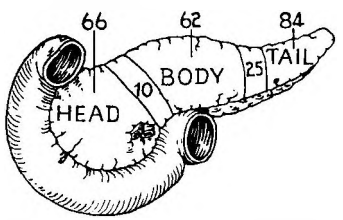


FIG. 1030.—Site of the insulin-secreting adenoma in 254 cases. Seven were situated in an ectopic pancreas. Twenty were overlooked at operation. (*J. M. Howard et al., Philadelphia.*)

at once, the pancreas should be mobilised to enhance thorough palpation. In 12 per cent. of cases more than one tumour is present. Once found by palpation, an incision is made in the pancreas over the tumour. As a rule complete enucleation is possible. Drainage of the area is essential. Undue hardness of the tumour calls for partial pancreatectomy, but this measure is required most often because of failure to locate the tumour, in which event more than half the specimens removed reveal no abnormality. Consequently, the result in these instances is not always what is hoped for. In cases where the tumour is removed, 87 per cent. of patients are cured; in 4 per cent. there is slight or no relief. The mortality is about 8 per cent.

Zollinger-Ellison Syndrome.—This diagnosis should be suspected in every case of recurrent peptic ulceration particularly where multiple ulcers are present or where they occur in unusual sites in the gastrointestinal tract. Two types are recognised—

Z.E.S. Type I (rare). These have antral G-cell hyperplasia, hypergastrinæmia and chronic peptic ulceration.

Z.E.S. Type II. This may be due to an ulcerogenic, non-β-islet-cell tumour[1], or sometimes to diffuse hyperplasia of the islet cells. The tumour secretes gastrin and is usually found in the tail of the pancreas. About half are malignant. There is (1) intractable peptic ulceration, often in the jejunum (2) hypergastrinæmia with massive acid hypersecretion of up to 500 ml per hour (3) diarrhœa and sometimes steatorrhœa or hypokalæmia.

Diagnosis.—Acid secretion studies (p. 816) and estimation of circulating gastrin

[1] So-called because it is likely, but not absolutely certain, that alpha-cells are involved.

Allen Oldfather Whipple, 1881–1963. Valentine Mott Professor of Surgery, Columbia University College of Physicians and Surgeons, New York, U.S.A.
Robert Milton Zollinger, Contemporary. Professor of Surgery, Ohio State University, Columbus, Ohio, U.S.A.
Edwin Homer Ellison, Contemporary. Professor of Surgery, Marquette University, Milwaukee, Wisconsin, U.S.A.

(p. 816) will usually establish this. In Type I the plasma gastrin levels are further increased by meat extracts. In Type II calcium infusions may do the same.

Treatment.—Type I—partial gastrectomy to remove the G-cell bearing area.
Type II—total gastrectomy with removal of the tumour if present.

This classification is the subject of current dispute.

H_2 receptor antagonists have recently been found to be effective in some cases (p. 829).

CARCINOMA OF THE PANCREAS

of the Head 70% — The head proper ($\frac{2}{3}$).
of the Head 70% — The periampullary region ($\frac{1}{3}$).
of the Body and Tail 30%.

Primary carcinoma of the pancreas accounts for some 4,000–5,000 deaths in England and Wales every year. Both here and in North America it is thought to be on the increase. Most of the patients are past the meridian of life, the average age being 57; sometimes this condition is seen in patients who are in their thirties. Contrary to general belief men are attacked only a little more than women.

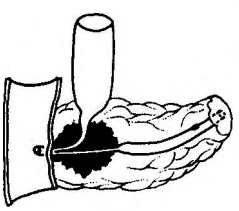


FIG. 1031.—Carcinoma of the head of the pancreas causing obstruction to the common bile duct and the duct of Wirsung.

Pathology.—Usually the lesion is scirrhous, almost gritty to the knife edge, and fibrous in appearance. Occasionally it is medullary; exceptionally it is a cystadenocarcinoma. It is thought to arise from the duct epithelium.

(*a*) **Carcinoma of the head proper** is sometimes small in size (diameter 2–4 cm). The reason for this is that while still comparatively small it compresses the common bile duct (fig. 1031) and unless the obstruction is relieved, death from cholæmia results. As a small growth always lies deeply in the pancreas, obtaining a reliable specimen for biopsy is problematical (p. 963). This group includes a small number of carcinomas of the distal portion of the common bile duct which are much less invasive.

(*b*) **Periampullary Carcinoma.**—In less than one-third of cases of carcinoma of the head of the pancreas the neoplasm arises in the duodenal papilla, in the ampulla of Vater, or in the duodenal mucosa adjacent to the papilla. When a specimen is examined at operation, it is often impossible to decide which of the three was the seat of origin—hence they are grouped together under the comprehensive term ' periampullary '.

(*c*) **Carcinoma of the body and tail** tends to attain a greater size than the foregoing varieties; it also metastasises more widely.

Spread: *Local Spread.*—Carcinomas belonging to categories (*a*) and (*c*) above tend to adhere to, and invade, adjacent structures, particularly the duodenum, occasionally the pyloric antrum, and the transverse mesocolon in the region of the superior mesenteric vessels.

Lymphatic spread from the head is first to the lymph nodes along the hepatic artery; that from the body and tail is to the gastric, cœliac, mesenteric, and

para-aortic lymph nodes. The supraclavicular lymph nodes are involved less commonly than in cases of carcinoma of the stomach.

Spread by the blood-stream occurs somewhat later. Metastases are carried especially to the liver; usually the hepatic metastases are small and numerous.

Peritoneal implantation resulting in ascites occurs in 10% of cases.

Clinical Presentation

Primary carcinoma of the pancreas presents in two distinct ways:

(1) **As Painless progressive Obstructive Jaundice.**—This group includes the vast majority of cases, and pruritus due to bile salts in the blood coincides with the onset of jaundice and may be severe. The jaundice usually progresses steadily until the patient is almost green in colour.

An exception to this rule sometimes occurs in cases of periampullary carcinoma. After about a month of progressive (usually painless) jaundice, necrosis of a portion of the growth sometimes occurs, and pent-up bile escapes into the duodenum. Variations in the depth of the jaundice and pyrexia heighten the similarity of the symptoms caused by this neoplasm to those of obstruction of the common bile duct by a gall-stone.

Jaundice may occur when the growth is in the body and tail, but then it is due either to malignant lymph nodes in the portal fissure or metastases in the liver.

Pain.—This must be carefully enquired for, for though it is present in some of these patients it may not be a marked feature. It is usually a steady dull ache in the epigastrium radiating through to the back; it may be colicky and simulate gall-stone pain; sometimes it is paroxysmal radiating from the umbilicus and through to the back. *A distended gall-bladder* is palpable in 60% of cases (Courvoisier's law, p. 928).

Anorexia is present in about 65% of cases. It is usually not severe.

Diarrhœa may be troublesome, and foul smelling. The pale stools in the jaundiced patient with a periampullary tumour may take on a glistening watery appearance (like *aluminium or lead paint*)[1] due to the presence of small amounts of blood. Steatorrhœa from enzyme deficiency, is the explanation.

Loss of weight occurs in 90% of cases. Often the patient may not have noticed it. A feeling of profound weakness or depression is not uncommon.

Enlargement of the Liver.—The liver is palpably enlarged in 65% of cases of carcinoma of the head of the pancreas. As a rule this enlargement is due to biliary obstruction, but it may be due to hepatic metastases.

Acute cholecystitis and/or acute pancreatitis is occasionally the first indication of carcinoma of the pancreas, and in patients of the older age group this possibility should come to mind.

(2) **As Intractable Pain without Jaundice.**—It must be remembered that any middle-aged patient who complains of intractable pain in the back at the level of the lumbar vertebræ 1 and 2, and especially on the left side, may be suffering from carcinoma of the tail of the pancreas. The usual

[1] Sir Heneage Ogilvie, 1887–1971. Surgeon, Guy's Hospital, London, described them as 'silvery stools'.

picture is that of a persistent epigastric or back pain with a normal barium series. The patient usually says that the pain is worse when he lies down and it tends to keep him awake at night. As with most cases of retroperitoneal inflammatory or neoplastic disease of the posterior abdominal wall, the pain is eased by sitting up and leaning forward in bed, and this is the position the patient usually adopts. The pain is not related to food, but a marked feature is that it persists, week in, week out. Unfortunately many of these cases are regarded as having a backache of functional origin. Weakness, loss of weight, anorexia, and diarrhœa are commonly present, and indeed, the disease may present in just this way.

Thrombophlebitis migrans may be an indication of the presence of pancreatic carcinoma of either type. The thrombophlebitis appears spontaneously in almost any superficial vein; it resolves, only to re-appear elsewhere. The explanation of this phenomenon is still obscure. It was first described by Trousseau and also occurs in other types of abdominal carcinoma and sometimes in chronic pancreatitis.

Diabetes and Carcinoma of the Pancreas.—(*a*) Diabetes mellitus is occasionally the first sign of carcinoma of the pancreas. Typical pain may be a pointer. The condition should be suspected when a middle-aged person develops diabetes and, in spite of adequate diabetic treatment, continues to lose weight. In these patients the carcinoma may run a galloping course.

(*b*) Carcinoma of the pancreas is about ten times more frequent in diabetics than in the population at large.

Calcinosis and Carcinoma of the Pancreas.—While pancreatic calcinosis is relatively rare among patients with pancreatic carcinoma, carcinoma of the pancreas develops in patients with pancreatic calcinosis with such frequency as to warrant the assumption that pancreatic calcinosis is a pre-carcinomatous condition.

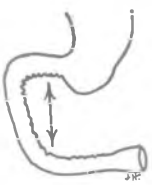


FIG. 1032.—The pad sign.

Investigations:

Radiography.—In the case of carcinoma of the head of the organ sometimes the ⊂ of the barium-filled duodenum is widened—the ' pad ' sign (so-called because it appears as if the ⊂ is crammed with padding) (fig. 1032). On infrequent occasions a periampullary carcinoma provides a filling defect which may give the duodenum the appearance of a reversed 3→Ɛ (the reversed Ɛ sign, fig. 1033). Cœliac angiography may show an abnormal vascular pattern in the gland.

Hypotonic Duodenography.—A Dreiling double lumen tube is screened into the duodenum (fig. 1034) 20–40 ml. of liquid barium solution is then run in and 4 mg of oxyphenonium bromide (Antrenyl) given intravenously to render the bowel atonic. Air is then carefully insufflated to distend the duodenum. This gives a clear outline of the viscus (fig. 1035). Where carcinoma has invaded the duodenal wall spiculation or ' rose-thorn ' distortion of the contour of the medial border may be seen (fig. 1036).

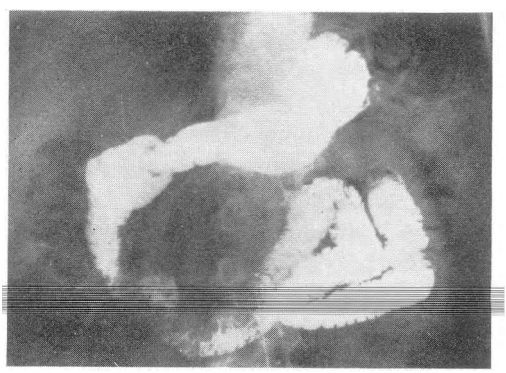


FIG. 1033.—Barium meal showing widened duodenal loop and the reversed 3 sign. (*Rodney Smith, P.P.R.C.S., London,* Trans. med. Soc. Lond.)

Armand Trousseau, 1801–1867. Physician, Hôtel-Dieu, Paris.

Chronic pancreatitis by contrast appears to straighten the medial duodenal wall.

Ampullary carcinomas may be well shown as a filling defect (fig. 1037).

Laboratory Tests.—*The stercobilinogen content of the fæces* is often lower than that found in chronic pancreatitis—less than 10 mg. per day.

Occult blood in the stools is a valuable finding. It is especially frequent in cases of periampullary carcinoma (see aluminium paint stool, p. 955).

The Triple Test.—In this useful test, 1. the tube is inserted, pancreatic function tested (p. 947) and 2. some of the aspirate centrifuged as soon as possible, at 3,000

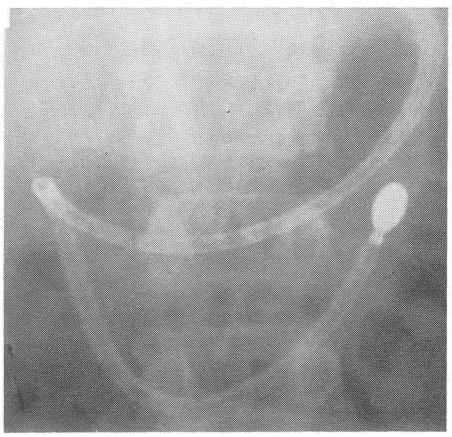


FIG. 1034.—Dreiling tube in duodenum.

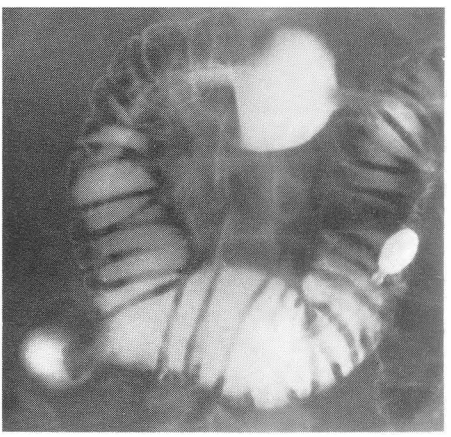


FIG. 1035.—Normal hypotonic duodenogram.

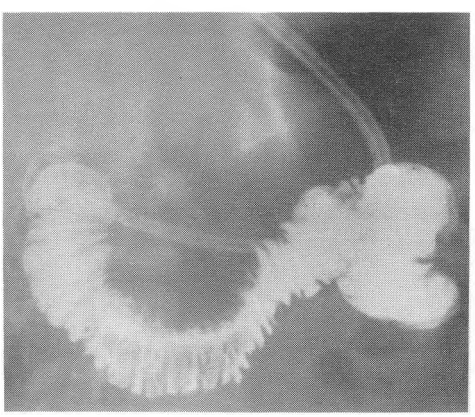


FIG. 1036.—Carcinoma of head of pancreas. 'Rose thorning' of medial wall of duodenum.

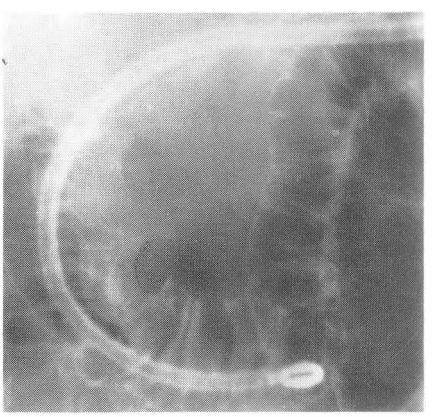


FIG. 1037.—Carcinoma of ampulla. Filling defect in region of ampulla.

rev/min for 10 minutes. 3. The deposit is then fixed and stained by Papanicolaou's method. At the conclusion of this hypotonic duodenography is performed. In a recent series all but one of 29 carcinomas of the head of the pancreas and all ampullary tumours were diagnosed by this combination of tests. This method will not, however, show stones in the common bile duct or high bile duct carcinomas. It can be carried out with modest hospital facilities.

Endoscopic Retrograde Cholangio-Pancreatography (**E.R.C.P.**).—The ampulla of Vater (fig. 1038) can now be cannulated using a side-viewing fibre-optic duodenoscope and the biliary and pancreatic ducts outlined with 65% Angiografin. This technique can be of value in confirming the presence or absence of a surgical cause of jaundice (fig. 1039).

George Nicholas Papanicolaou, 1883–1962. Professor of Clinical Anatomy, Cornell University, New York.

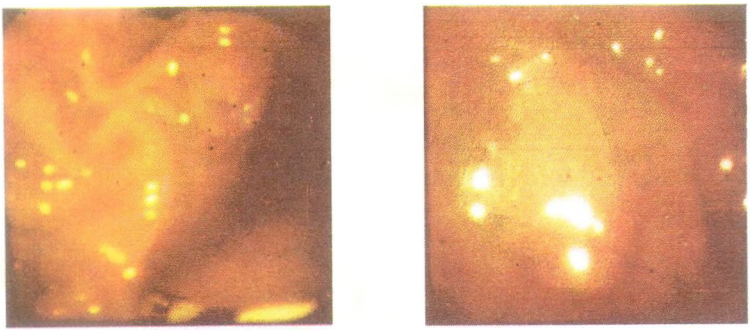


FIG. 1038.—Normal ampulla seen at duodenoscopy.

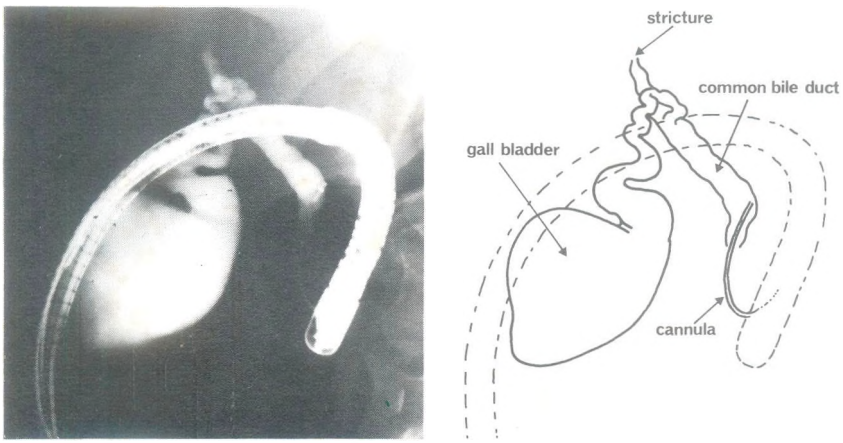


FIG. 1039.—E.R.C.P. Malignant stricture of common hepatic duct. No contrast passes upwards beyond it. *(Dr. P. Cotton ,London and The Lancet, 1972.)*

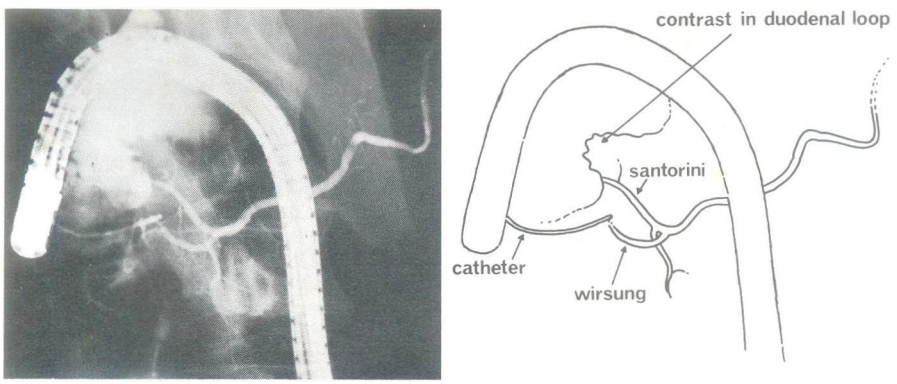


FIG. 1040.—E.R.C.P.: Normal pancreatic duct with filling of duct of Santorini from the

The retrograde pancreatogram will demonstrate the morphology of the pancreatic ducts. The normal anatomy is shown in fig. 1040. Changes seen in chronic pancreatitis include strictures, dilated main ducts with stones (figs. 1041 and 1042) and first order branches and pancreatic cysts (fig. 1043). The ' chain ' of lakes appearance

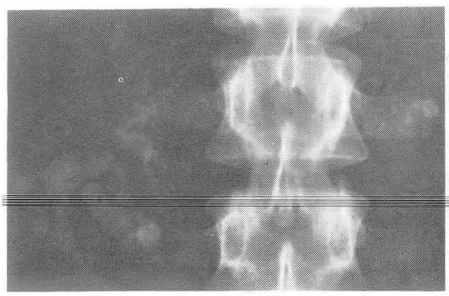


FIG. 1041.—Plain abdominal X-ray: Chronic pancreatitis. Multiple opacities in the region of head and tail of pancreas.

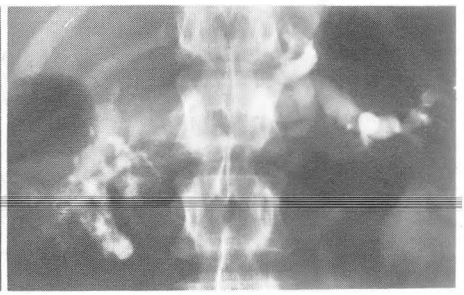
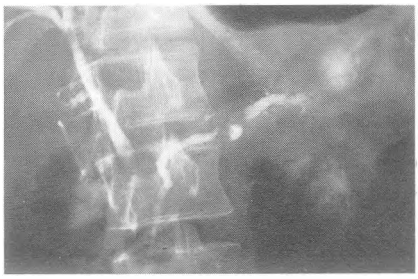


FIG. 1042.—E.R.C.P.: Same patient as in fig. 1041. Most of opacities lie within the duct system and are stones. Gross dilatation of ducts in body and tail due to obstruction by stones in head of pancreas.



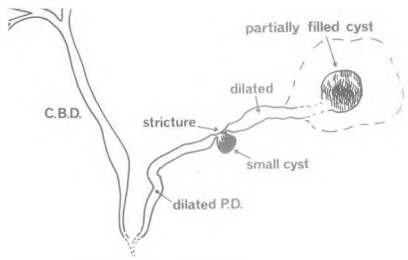
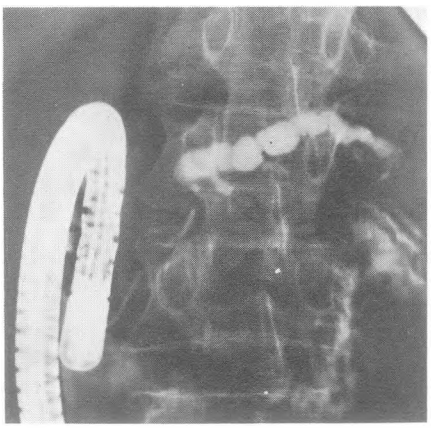


FIG. 1043.—E.R.C.P.: Relapsing acute pancreatitis. Normal biliary tree. Pancreatogram shows stricture of main duct in the body with distal dilatation and cyst formation. (*Dr. P. Cotton, London, and Gut, 1972.*)



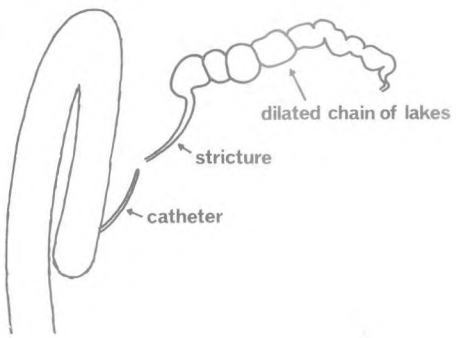


FIG 1044.—E.R.C.P.: Chronic pancreatitis. Long stricture of pancreatic duct in the head; distal pancreatic duct shows sacculation with intervening short strictures, 'chain of lakes'. (*Dr. P. Cotton, London.*)

due to alternating strictures and duct sacculation is seen in fig. 1044. The technique is of great value in both the diagnosis of chronic pancreatitis and in determining whether surgical intervention is indicated and what therapeutic procedure should be used.

In pancreatic carcinoma the main pancreatic duct is narrowed or completely obstructed at the site of the tumour with dilatation upstream of the obstruction but with a normal duct system downstream of the obstruction (fig. 1045).

In cases where the changes are difficult to distinguish from pancreatitis, cytological

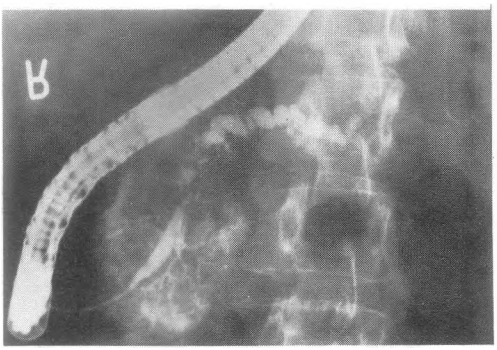


FIG. 1045.—E.R.C.P.: Pancreatic carcinoma. Irregular stricture of main pancreatic duct with dilatation upstream of obstruction.

FIG. 1046.—Group of adenocarcinoma cells from the pancreatic juice, collected at the time of E.R.C.P. of the patient illustrated in fig. 1045.

examination of pancreatic juice obtained directly from the cannulated pancreas can confirm the radiological diagnosis (fig. 1046).

The investigation is not free of side effects; cholangitis, amylasæmia and acute pancreatitis have occasionally been encountered.

Although difficult to master, the technique has much to offer and seems less risky than liver biopsy or percutaneous transhepatic cholangiography (p. 933) in jaundiced cases where the diagnosis remains in doubt. The *triple test* by contrast requires no special equipment or skill but has the limitations mentioned above (p. 957).

METHODS OF SCANNING THE PANCREAS

There are at present three quite different methods for imaging the pancreas, namely; isotope scanning, ultrasonography and computed axial tomography (CAT or CT scan). All three methods are non-invasive and therefore more acceptable to patients than selective arteriography or endoscopic retrograde pancreatography, but differ considerably in availability, cost and in their ability to detect disease.

Isotope Scanning.—Radioactive Seleno-Methionine (^{75}Se-Selenomethionine) is concentrated in the pancreas, but is also taken up by the liver, and through γ-ray emission is detected by a gamma camera after a dose of approximately 250 mCi given intravenously. Scanning takes place 20 to 30 minutes later. If the liver hides part of the pancreas a subtraction scan can be performed after giving 2–3 mCi ^{99}mTc sulphur/sulphide colloid which is taken up by the liver, but not by the pancreas. Thus the normal pancreas can be imaged using isotope scanning.

In clinical practice, however, isotope imaging of the pancreas has proved disappointing. Its main value was in demonstrating a normal pancreas to exclude pancreatic disease, but is of limited value in diagnosing pancreatic carcinoma because the false positive rate is so high. Non-uptake of ^{75}Se-Selenomethionine also occurs in chronic pancreatitis, old age and various other conditions. There is frequently an area of non-uptake at the junction of the head and body of the pancreas where the superior mesenteric artery indents its posterior margin. Isotope imaging of the pancreas has thus largely been abandoned in favour of ultrasonography and computed tomography.

Ultrasonography uses mechanical vibrations from a transducer which by the Piezo-effect, both produces and receives the ultrasound waves. The reflections can be recorded as a series of individual deflections A-scope, or be formed into 2 dimensional images in the B-scope. With the development of grey-scale the sectional

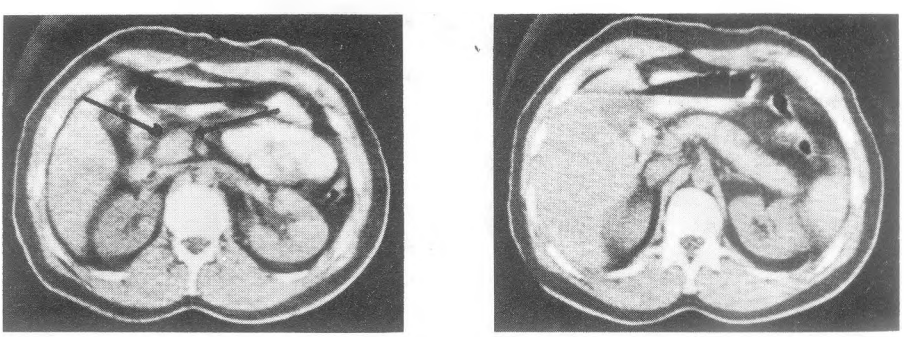
images are no longer presented as only white dots on a black background, but with shades of grey forming sections in which the anatomy is more easily recognized. The further introduction of ' real-time ' scanning shows the images almost instantaneously as they are received. Movements become visible, and therefore fluid bowel and pulsating vessels can be identified. Ultrasonography offers major advantages as it is relatively inexpensive, requires no patient preparation, and sections are not limited to any one plane. However, gas and bone are impenetrable barriers for ultrasound which limits its use in the region of the tail of the pancreas. It is also more difficult to perform and interpret than isotope or computed tomography, and the results are therefore rather more subjective, being dependent on the skill of the performer.

In scanning the pancreas, the adjacent vessels particularly the aorta, superior mesenteric artery and portal vein are identified in longitudinal sections and then the pancreas is examined with axial or oblique sections 2–5 mm apart. The normal pancreas is moderately reflective. The best images are produced in thin subjects, but the images are poor in the obese with well developed surrounding fat planes.

Carcinoma of the pancreas in the head and body can be identified in most patients when they present clinically but tumours less than 2 cm in diameter are not usually detected. Liver metastases are usually visible but not pulmonary or bone metastases. Pseudocysts are easily recognized and pancreatic calcification produces highly reflective echoes with acoustic shadowing beyond. With real expertise a dilated pancreatic duct is recognizable. Jaundice is one of the major indications for ultrasonography. Obstructive jaundice is shown by the dilated intrahepatic ducts produced and the actual site of obstruction can often be identified particularly if a tumour is in the head of the pancreas. Gallstones in the gall-bladder and even intrahepatic cholelithiasis can be shown.

Ultrasonography has thus become an important primary imaging technique in pancreatic disease, but its limitations must be appreciated particularly in identifying lesions in the tail of the pancreas or in obese subjects.

Computed Tomography.—Figs. 1047–1052, uses x-radiation together with a



(*a*) (*b*)

FIG. 1047.—Normal pancreas on computed tomography. (All CAT scans are viewed from the foot of the patient.) (*a*) The head of the pancreas (arrows) lies adjacent to the superior mesenteric artery and anterior to the inferior vena cava at the entry of the left renal vein which curves over the aorta. (*b*) The body and tail of the pancreas curves over the superior mesenteric artery with the tail of the pancreas lying in the angle between the left kidney and the spleen.

reconstructive algorithm programme to form axial sections of the body. The major advantage of CT scanning is the detailed anatomical displays produced, but these are limited to the transverse axial plane and the equipment and running costs are considerably more expensive than either ultrasonography or isotope scanning.

Pancreatic carcinoma larger than 3 cm is usually demonstrable whether in the head (fig. 1048), body or tail, but the adjacent duodenum and jejunum must be clearly labelled with an oral contrast agent, otherwise they may be mistaken for a tumour. Pseudocysts (figs. 1051 and 1052) are easily identified as well as pancreatic calcifications, gallstones, dilated intrahepatic ducts, and a distended gall-bladder. In acute pancreatitis the

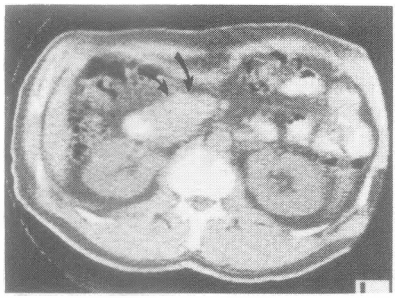


FIG. 1048.—Carcinoma of the head of the pancreas (curved arrows) causing enlargement and a lobulated margin.

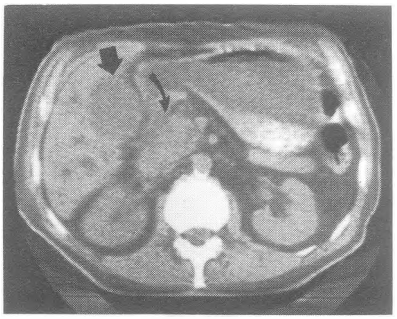


FIG. 1049.—Carcinoma of the head of the pancreas (curved arrow) with an enlarged gall-bladder (broad arrow) and dilated ducts in the liver.

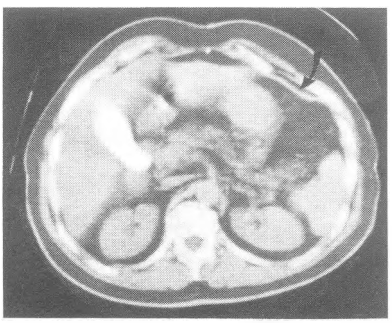


FIG. 1050.—Chronic pancreatitis causing peritoneal thickening (curved arrow).

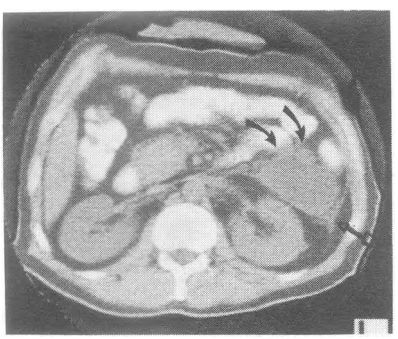


FIG. 1051.—Pseudocyst in the region of the tail of the pancreas (curved arrows) associated with thickening of the transversalis fascia and anterior and posterior pararenal fascia (open arrow).

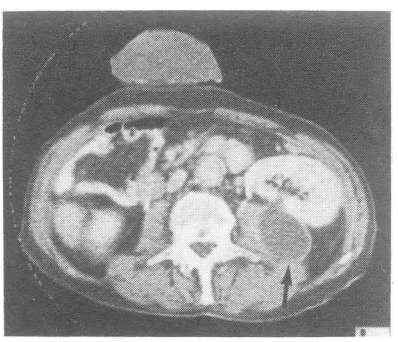


FIG. 1052.—Retroperitoneal pseudocyst displacing the kidney. A dense rim has appeared after intravenous contrast enhancement.

surrounding inflammatory changes are frequently visible as well as the late thickening of the peritoneal reflections, particularly the anterior peri-anal fascia of Gerota. Contrary to ultrasonography, the best results are obtained with patients who have well developed fat planes. Abscesses and pseudocysts of the pancreas have similar appear-

of cases the clinical findings are sufficiently distinctive allowing correct interpretation of the scanning images. While the scanning methods have considerably enlarged the scope of diagnostic imaging there are still significant deficiencies. Endocrine tumours such as insulinomas and gastrinomas are not visualised when small, and are best demonstrated by selective arteriography. E.R.C.P. (fig. 1045) is more effective in diagnosing tumours which obstruct or narrow the pancreatic duct even when small. However, the clear 3-dimensional view obtained with ultrasonography and computed tomography allows accurate placements of needles and catheters. Percutaneous cytology using these scanning methods has now become accepted practice and percutaneous drainage of pseudocysts and abscesses can save an otherwise inevitable laparotomy.

In the future other medical imaging systems will almost certainly be available. *Nuclear magnetic resonance computed tomography* (Zeugmatography) is non-ionizing and apparently harmless. Imaging in any plane is possible and blood vessels show particularly well.

Scanning methods are now accepted as a valuable diagnostic method in the pancreas and may have an even more significant role in the not too distant future.

Management:

Exploratory laparotomy is called for under the following circumstances: (1) Patients presenting with upper abdominal pain and loss of weight which might be pancreatic in origin, where none of the above special investigative back-up is available, (2) where undiagnosed jaundice persists and the laboratory tests remain equivocal but not until three weeks have passed. Preliminary vitamin K injections are necessary. If the serum bilirubin is over 15 mg/100 ml (255 mol/l) and rising or the blood urea is elevated Mannitol (p. 82) should be given at the start of the operation. If resection is judged to be feasible and there is no evidence of metastases or extra-pancreatic spread, histological evidence should, if possible, be obtained. Suction needle biopsy is emerging as the most safe technique for this but all methods are limited by histological dubieties. Ideally studies of pancreatic juice obtained with the Triple Test or with E.R.C.P. will already have shown malignant cells and this renders biopsy, which can be hazardous, unnecessary.

Operative Treatment.—The problem of obtaining histological evidence is now, however, of limited relevance. Many series from all over the world have shown that pancreaticoduodenectomy for growths in the head of the pancreas itself have given five-year survival rates which are no better than is achieved by palliative operations. These carry an immediate operative mortality rate which is much lower. In most centres resection is now advised only in cases where the tumour is likely to be slow growing, that is, in tumours of the distal bile duct, ampulla or duodenal papilla.

PANCREATICODUODENECTOMY

Procedure.—A long right paramedian incision is made. Explore the abdomen and the area thoroughly. Involved lymph nodes, fixity of the tumour, and liver secondaries rule out radical surgery.

Expose the duodenum by separating the right half of the transverse colon and the hepatic flexure from their peritoneal attachments and dissect out the common bile duct

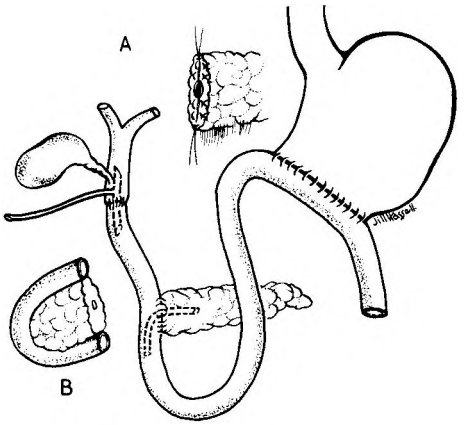


FIG. 1053.—Reconstruction after pancreato-duodenectomy. Inset A, the stump of the body of the pancreas prepared for anastomosis with the jejunum. Inset B, the portion removed. (*After H. A. Zintel.*)

above the duodenum. Mobilise the duodenum by dividing the peritoneum along its right border so that it can be lifted off the inferior vena cava. Clear the superior mesenteric vessels and make sure that these and the portal vein are not involved in growth.

If the growth is operable the operation may proceed. Divide the common bile duct above the duodenum, mobilise the duodenum and pyloric antrum and ligate and divide the right gastric, right gastro-epiploic, and gastro-duodenal vessels. The duodenojejunal flexure is exposed and divided and the inferior pancreaticoduodenal vessels are ligated. The superior mesenteric vessels in the region of the uncinate process must be carefully exposed. The pancreatic venous radicles there are very short and tenuous. They may be torn easily. Reconstruction is now carried out as in fig. 1053. Alternatively, the pancreas may be anastomosed end-to-end with the jejunum and the bile duct end-to-side.

The abdomen is now closed, but the areas of the biliary and pancreatic anastomoses must be drained.

Post-operative Treatment.—Transfusion of blood should be continued after the operation, as necessary, and vitamin K injections given for several days.

Mortality and Results.—The general mortality of this extensive procedure is about 20%. The complications are principally those of bleeding from numerous unidentified sources or leakage from the pancreatic or common bile duct anastomoses. The five-year survival rate of this operation is disappointingly low for carcinoma of the pancreas proper (4–6%); when performed for carcinoma of the ampulla the rate is much better (> 30%).

PALLIATIVE OPERATION

In the presence of jaundice when resection is not indicated, cholecystjejunostomy (fig. 1054) is performed. This measure, by short-circuiting the obstruction of the common bile duct, relieves the jaundice and gives good palliation. It should be combined with gastroenterostomy if there is any danger of the tumour later obstructing the duodenum. Where the cystic duct is inserted low and may come to be occluded, the operation is unsuitable. Here choledocho-jejunostomy is indicated. Perhaps the major blessing it confers is relief from the intolerable itching. Back pain from continued infiltration of the growth is its most serious sequel. Operative mortality is 3–5%.

FIG. 1054.—Cholecystjejunostomy.

From time to time, to the astonishment of all concerned, the patient continues to improve and recovers completely, which shows how closely chronic pancreatitis of the head of the organ can mimic a neoplasm in this situation (fig. 1055).

Treatment of Carcinoma of the Body and Tail of the Pancreas.—When laparotomy is performed before a growth situated towards the tail of the organ has metastasised, it can be resected with comparative ease, and some lasting cures have been reported. The outlook in rare cases of papillary cystadenocarcinoma is particularly favourable.

Total pancreatectomy is a formidable procedure but has been successful for both carcinoma and chronic pancreatitis. The patients require insulin—30 to 70 units daily. Pancreatin, 10 to 12 g daily, is given to improve fat absorption.

Treatment of Papillary Carcinoma.—There is some evidence that transduodenal amputation of such lesions, with re-implantation of the bile and pancreatic ducts

into the duodenum, may afford good palliation in debilitated subjects. The procedure should be combined with cholecystjejunostomy, to anticipate local recurrence. It should not be attempted without full mobilization of the duodenal loop.

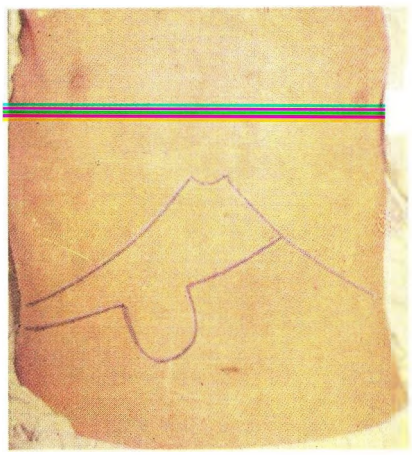


FIG. 1055.—Obstructive jaundice with markedly distended gall-bladder (Courvoisier's Law, p. 928). ? carcinoma of pancreas. ? chronic pancreatitis.

Treatment of Intractable Back Pain.—Both Splanchnic Block and Percutaneous Cordotomy have been used successfully for this.

Chemotherapy 5-Alpha-Fluoro-Uracil (5 FU) reduces pancreatic secretion and may reduce the speed of growth of the tumour and relieve pain in some cases.

CHAPTER 48

THE PERITONEUM. OMENTUM. MESENTERY AND RETROPERITONEAL SPACE

THE peritoneum is conveniently divided into two parts—the *visceral* surrounding the viscera, the *parietal* lining the rest of the cavity. The parietal portion is richly supplied with nerves and, when irritated, causes severe pain accurately localised to the affected area. The visceral peritoneum, on the other hand, is poorly supplied with nerves and pain arising therefrom is vague and poorly localised (p. 990).

Surgical Physiology.—The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane being nearly equal to that of the skin. This veil-like serous membrane is composed of flattened polyhedral cells, one layer thick, resting upon a thin layer of fibro-elastic tissue, the two layers constituting the peritoneum. Beneath the peritoneum, supported by a small amount of areolar tissue, lies a network of lymphatic vessels and rich plexuses of capillary blood-vessels from which all absorption and exudation must occur. In health, only sufficient peritoneal fluid, which is a pale yellow, somewhat viscid fluid containing lymphocytes and leucocytes, is secreted to ensure that the more mobile viscera glide easily. The specific gravity of the intra-abdominal contents is approximately 1·000. Mobile gas-filled structures float upwards. It is not possible to measure pressure in a potential space but indirect evidence suggests that intraperitoneal pressure may be lower than atmospheric, and is unrelated to intravisceral pressure.

In the erect position, when free fluid is present in the peritoneal cavity, pressure is reduced in the upper abdomen compared with the lower abdomen. When air is introduced it rises, allowing all the abdominal contents to sink.

Under the influence of decreased intra-abdominal pressure, occasioned by the upward movement of the diaphragm during expiration, aided by capillary attraction, any secretion travels continuously in an upward direction, where experimental evidence shows that fine coloured particulate matter and bacteria are absorbed rapidly into the peritoneal lymphatic network. In a matter of minutes these coloured particles reach the lymph vessels above the diaphragm.

When there is an outpouring of many ounces of peritoneal fluid, e.g. when the contents of the stomach or duodenum escape into the peritoneal cavity through a perforation, in all probability, excess of free fluid within the peritoneal cavity unobstructed by adhesions runs downwards.

When parietal peritoneal defects are created, healing occurs not from the edges, but by metamorphosis of *in situ* mesenchymal cells. Large defects heal as rapidly as small defects.

ACUTE PERITONITIS

Nearly all varieties of peritonitis are due to an invasion of the peritoneal cavity by bacteria. To such an extent is this true that when the term ' peritonitis ' is used without qualification, bacterial peritonitis is implied.

Bacteriology

Bacteria from the Alimentary Canal.—Usually the infection is caused by two or more strains. The commonest invaders are *Escherichia coli*, ærobic and anærobic streptococci, and the bacteroides. Less frequently the *Clostridium welchii* is found; still less frequently staphylococci or *Klebsiella*

Theodor Albrecht Edwin Klebs, 1834–1913. Professor of Bacteriology successively at Prague, Zurich and Rush Medical College, Chicago, Illinois, U.S.A.

pneumoniæ (Friedländer's bacillus), and so on. Many of the strains of *Esch. coli*, bacteroides, and *Cl. welchii* produce toxins which cause severe illness or death when they invade a large absorptive area (endotoxin shock, p. 83).

The Bacteroides.—Only recently has the frequency and importance of the presence of bacteroides in the causation of peritonitis been realised. These Gram-negative, non-sporing organisms though predominant in the lower intestine often escape detection because they are strictly anærobic, and slow to grow on culture media unless there is an adequate carbon-dioxide tension in the anærobic apparatus (Gillespie). In many laboratories, the culture is discarded if there is no growth in forty-eight hours. These organisms are resistant to penicillin and streptomycin but sensitive to metronidazole, clindamycin and lincomycin.

Bacteria NOT *from the Alimentary Canal.*—Examples are peritonitis due to the gonococcus, beta-hæmolytic streptococcus, pneumococcus, and the *M. tuberculosis*. Since the advent of antibiotics hæmolytic streptococcal peritonitis has lost many of its dreaded lethal properties.

PATHS OF BACTERIAL INVASION

Direct infection	Via perforation of some part of the gastro-intestinal canal. Through a penetrating wound of the abdominal wall. Post-operative.
Local extension	From an inflamed organ, e.g. appendicitis, cholecystitis. Migration through gut wall, e.g. strangulated hernia. From or via the Fallopian tubes.
Blood-stream	Part of general septicæmia.

Even in non-bacterial peritonitis (e.g. intraperitoneal rupture of the bladder or a hæmoperitoneum) the peritoneum soon becomes infected by transmigration of organisms from the bowel, and it is not long (often a matter of hours) before chemical peritonitis becomes a peritonitis in the usual meaning of the term.

Factors which favour Localisation of Peritonitis. These factors are anatomical and pathological:—

Anatomical.—Excluding the subphrenic spaces (p. 972), the greater sac of the peritoneum is divided into (*a*) the pelvis, and (*b*) the peritoneal cavity proper. The latter is re-divided into a supracolic and an infracolic compartment by the transverse colon and transverse mesocolon, which deter the spread of infection from one to the other. When the supracolic compartment overflows, as is often the case when a gastric ulcer perforates, it does so over the colon into the infracolic compartment, or, by way of the right paracolic gutter to the right iliac fossa, and thence to the pelvis.

Pathological.—The clinical course is largely governed by the manner in which adhesions form around the affected organ. Inflamed peritoneum loses its glistening appearance and becomes reddened and velvety. Flakes of fibrin appear and cause coils of intestine to become adherent to one another and to the parietes. There is an outpouring of serous fluid rich in leucocytes and antibodies, that soon becomes turbid; if localisation occurs, the turbid fluid becomes frank pus. Peristalsis is retarded in affected coils,

Carl Friedländer, 1847–1887. Prosector, Berlin-Friedrichshain Hospital.
William Alexander Gillespie, Contemporary. Formerly Professor of Clinical Bacteriology, University of Bristol.
Gabriele Falloppio (Fallopius), 1523–1563. Professor of Anatomy, Surgery and Botany, Padua, Italy.

and this helps in preventing distribution of the infection to other coils. The greater omentum, by enveloping and becoming adherent to inflamed structures, often forms a substantial barrier to the spread of infection.

Factors which tend to cause diffusion of peritonitis

(*a*) A prime factor in the spread of peritonitis is whether it develops rapidly or slowly. If an inflamed appendix (fig. 1056) or other hollow viscus perforates before localisation has taken place, there is a gush of intestinal contents into the peritoneal cavity which spreads over a large area almost instantaneously.

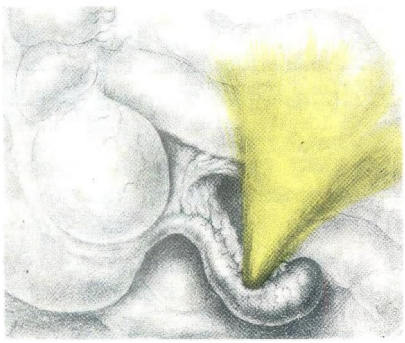


FIG. 1056. — Sudden perforation, especially if engendered by purgation, often results in an immediate widespread bacterial peritonitis.

(*b*) The ingestion of food, or even water, by stimulating peristaltic action, hinders localisation. Violent peristalsis occasioned by the administration of a purgative or an enema, causes a widespread distribution of an infection that would otherwise have remained localised.

(*c*) When the virulence of the infecting organism is so great as to render the localisation of the infection difficult or impossible.

(*d*) In children the omentum is small.

CLINICAL FEATURES

Localised peritonitis is bound up intimately with the causative lesion, and the initial symptoms and signs are those of that lesion. When the peritoneum becomes inflamed the temperature, and especially the pulse-rate, rise. The pain increases and usually there is repeated vomiting. The most important sign is rigidity of the abdominal wall over the area of the abdomen which is involved. If inflammation arises under the diaphragm, shoulder tip (referred) pain may be felt (p. 973). In cases of pelvic peritonitis arising from an inflamed appendix in the pelvic position or from salpingitis, the abdominal signs are often slight, deep tenderness of one or both lower quadrants alone being present, but a rectal or vaginal examination reveals tenderness, often exquisite, of the pelvic peritoneum. With appropriate treatment localised peritonitis usually resolves. In about 20% of cases an abscess follows. Infrequently, localised peritonitis becomes diffuse. Conversely, in favourable circumstances diffuse peritonitis can become localised, most frequently in the pelvis.

Diffuse (*syn.* **Generalised**) **Peritonitis.**—1. *Onset.*—Pain, which is 'sore', 'cutting', or 'burning', is made worse by moving or breathing. It is at first localised to the site of the original lesion, and spreads outwards from this point. Vomiting may occur. The patient usually lies still. Tenderness, guarding, and rigidity on palpation are typically found when the

peritonitis affects the anterior abdominal wall. Abdominal tenderness, guarding, and rigidity are diminished or absent if the anterior wall is unaffected, as in pelvic peritonitis or, rarely, peritonitis in the lesser sac. Patients with pelvic peritonitis may complain of urinary symptoms; they are tender on rectal or vaginal examination. Bowel sounds may still be heard for a few hours but they gradually cease with the onset of paralytic ileus. The pulse rises progressively, but if the peritoneum is deluged with irritant fluid, there is a sudden rise. The temperature changes are variable.

2. *Intermediate.*—Peritonitis may resolve, so that the pulse slows, the pain and tenderness diminish, leaving a silent, soft, abdomen. (These are features that can easily mislead the observer.) The condition may localise, producing one or more abscesses, with overlying swelling and tenderness.

3. *Terminal.*—If resolution or localisation have not occurred, the abdomen remains silent, and increasingly distends. Circulatory failure ensures, with cold, clammy extremities, sunken eyes, dry tongue, drawn and anxious face (Hippocratic facies—fig. 1057). The patient finally lapses into unconsciousness. With adequate treatment, this condition is rarely seen in hospitals.

FIG. 1057.—The facies in terminal diffuse peritonitis.

Diagnostic aids.—Investigations will not save the need for careful confirmation of the history and physical signs, which are tenderness, guarding and rigidity with a distending silent abdomen. It is unwise to rely slavishly on any single symptom or sign; not one is incontrovertible.

A battery of investigations *may* elucidate a doubtful diagnosis. More often, they only confuse it. Peritonitis usually, but not always, produces leucocytosis. *Peritoneal diagnostic aspiration* may be helpful but is usually unnecessary. After infiltrating the skin of the abdomen with local anæsthetic, the peritoneum is entered in each quadrant with a sterile needle attached to a syringe, into which is sucked any free fluid. Bile-stained fluid indicates perforated peptic ulcer, the presence of pus indicates bacterial peritonitis; blood is aspirated in a high proportion of patients with intraperitoneal bleeding. When aspiration fails, the introduction of a small quantity of sterile physiological saline, followed after a few minutes by peritoneal aspiration may produce fluid of diagnostic value.

An X-ray film of the abdomen may reveal free air, or confirm the presence of dilated gas-filled loops of bowel with multiple fluid levels.

Serum amylase estimation may uphold the diagnosis of pancreatitis, provided it is remembered that raised values are frequently found following other abdominal catastrophes, and operations.

Treatment

The essential principles in the treatment of peritonitis are: (1) Correction of circulating volume and any electrolyte imbalance. (2) Decompression. (3) Antibiotics. (4) Appropriate surgical treatment.

1. *Intravenous Fluids.*—These patients are frequently hypovolæmic with electrolyte disturbances. The plasma volume must be restored and the

Hippocrates, by common consent the Father of Medicine, was born on the Greek Island of Cos, off Turkey, about 460 B.C. and died in 375 B.C.

plasma electrolyte concentrations corrected. Plasma protein depletion may need correction—inflamed peritoneum leaks protein calamitously and continuously.

2. *A nasogastric tube* is passed into the stomach, which is aspirated. Intermittent aspiration is maintained until the paralytic ileus resulting from peritonitis has recovered. After operation, measured volumes of water are allowed by mouth, when only small amounts are being aspirated. If the abdomen is soft and not tender, and bowel sounds return, oral feeding may be progressively introduced. It is important not to prolong the ileus by missing this stage.

3. *Antibiotics.*—Administration of antibiotics prevents the multiplication of bacteria, and the release of endotoxins. As the infection is usually a mixed one, initially parenteral ampicillin, gentamicin and metronidazole may be given.

4. *Surgical correction of the underlying cause* must not be delayed in appropriate cases, once the patient is fit for operation. At the same time the results of peritonitis are corrected—necrotic material and pus are removed from the peritoneal cavity (see Peritoneal Toilet, below).

Ancillary Care

5. *A fluid balance chart* must be started at once so that daily output by gastric aspiration and urine are known, losses from the lungs, skin, and in fæces are estimated, so that the intake requirements can be calculated and seen to have been administered. Throughout recovery, the hæmatocrit values, blood electrolytes, and urea must be checked.

6. *Intravenous fluids* are continued after operation until the patient can be fully maintained by oral feeding. If recovery is delayed, high caloric, vitamin-rich supplements must be added to the balanced electrolyte solutions.

7. The patient, nursed in the sitting-up position, must be relieved of pain before and after operation. Morphine may be given, and small doses continued for 48 hours. Freedom from pain allows early mobilisation and adequate physiotherapy in the post-operative period, to prevent basal collapse, deep-vein thrombosis and pulmonary embolism.

Treatment of the Cause.—If the condition is amenable to surgery, such as in perforated appendicitis, gangrenous cholecystitis, diverticulitis, peptic ulcer or rarely in perforation of the small bowel, operation must be carried out as soon as the patient is fit for anæsthesia. This is usually within a few hours. In peritonitis due to pancreatitis, salpingitis, or in cases of primary peritonitis of streptococcal or pneumoccocal origin, conservative treatment is the procedure of choice (if the diagnosis can be made with certainty).

Peritoneal Toilet.—In surgery for general peritonitis it is essential that after the cause has been dealt with, the whole peritoneal cavity should be explored with the sucker and mopped dry, if necessary until all sero-purulent exudate is removed. It is inadvisable to put in any antibiotic or antiseptic; the peritoneal fluid which is secreted is one of nature's best defence mechanisms.

Prognosis.—With modern treatment diffuse peritonitis carries a mortality of about 10 per cent. The lethal factors are (*a*) bacterial toxæmia, (*b*) paralytic ileus, (*c*) bronchopneumonia, (*d*) electrolyte-imbalance.

COMPLICATIONS OF PERITONITIS

All the complications of a severe bacterial infection are possible, but the special complications of peritonitis are as follows:

(1) *Acute intestinal obstruction due to peritoneal adhesions* (p. 1047). This usually gives central colicky abdominal pain with evidence of gas and fluid levels confined to the upper portion of the small intestine on X-ray. It is essential to distinguish this from:

(2) *Paralytic ileus* where there is usually little pain and gas-filled loops with fluid levels are seen distributed throughout the small and large intestines on the X-ray film. In intestinal obstruction, bowel sounds are increased. In paralytic ileus bowel sounds are reduced or absent.

(3) *Residual abscesses.*—Abscess formation following local or diffuse peritonitis usually occupies one of the situations shown in fig. 1058. When palpable, an intraperitoneal abscess should be treated by marking out its limitations on the abdominal wall, and meticulous daily examination. In the majority of cases, with the aid of antibiotic treatment, the mass becomes smaller and smaller, and finally is impalpable. In others, the abscess fails to resolve, or becomes larger, in which event it must be opened. In many situations, by waiting a few days the abscess becomes adherent to the abdominal wall, so that it can be drained without opening the general peritoneal cavity.

In the case of a laterally-placed abscess, the incision is made on the lateral side of the swelling. The layers of the abdominal wall are divided until the peritoneum is reached. With the finger, the extra peritoneal tissues are separated from the peritoneum until the abscess is opened. A drainage tube is inserted; if the path is tortuous, a Penrose drain (a thin tube of latex rubber containing a wick of gauze) is valuable.

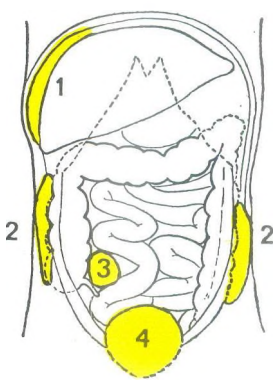


FIG. 1058. — Common situations for residual abscesses. 1. Subphrenic. 2. Paracolic. 3. Appendix. 4. Pelvic.

PELVIC ABSCESS

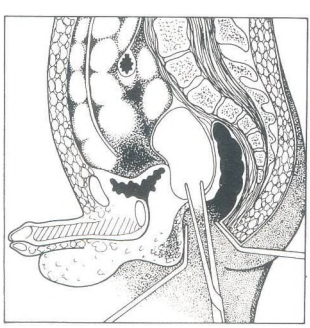


FIG. 1059.—Opening a pelvic abscess into the rectum.

The pelvis is the commonest site of an intraperitoneal abscess, because the vermiform appendix is often pelvic in position and also the Fallopian tubes are frequent foci of infection. A pelvic abscess can also occur as a sequel to any case of diffuse peritonitis. Pus can accumulate in this area without serious constitutional disturbance, and unless the patient is examined carefully from day to day, such abscesses may attain considerable proportions before being recognised. The most characteristic symptoms of a pelvic abscess are diarrhœa and the passage of mucus in the stools. It is no exaggeration to say that the *passage of mucus, occurring for the first time in a patient who has, or is recovering from, peritonitis, is pathognomonic of pelvic abscess.* Rectal examination reveals a bulging of the anterior rectal wall which, when the abscess is ripe, becomes softly cystic. Left to Nature, a proportion of these abscesses burst into the rectum, after which the patient nearly always recovers rapidly. If this

Charles Bingham Penrose, 1862–1925. Professor of Gynæcology, University of Pennsylvania, Philadelphia, U.S.A.

possible happy termination does not readily occur, the abscess should be drained deliberately. In women vaginal drainage through the posterior fornix is often chosen. In other cases, where the abscess is definitely pointing into the rectum, rectal drainage (fig. 1059) is employed. If any uncertainty exists as to the presence of pus, an aspirating needle introduced through the rectal wall into the bulging swelling will settle the question. Occasionally, in the case of a large abscess which can be palpated above the pubes, after the bladder has been emptied by catheterisation, lower laparotomy should be undertaken in order to be quite certain of the diagnosis. Provided the abscess is shut off from the general peritoneal cavity, a point which can be ascertained undeniably when the abdomen has been opened, rectal drainage of a pelvic abscess is preferable to suprapubic drainage, which breaks down Nature's barriers and exposes the general peritoneal cavity to the dangers of spreading infection.

SUBPHRENIC ABSCESS

Anatomy.—The complicated arrangement of the peritoneum results in the formation of four intraperitoneal and three extraperitoneal spaces in which pus may collect. Three of these spaces are on either side of the body, and one approximately in the mid line (fig. 1060).

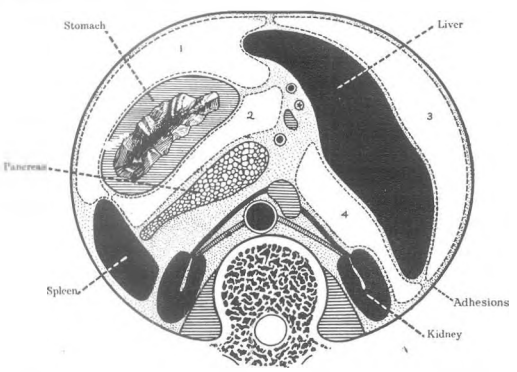


FIG. 1060.—Transverse section through the lesser sac of the peritoneum showing situations which intra-peritoneal subphrenic abscesses may occupy. 1. left anterior space. 2. left posterior space (lesser sac). 3. right anterior space which becomes shut off by adhesions from 4, right posterior space.

Left Anterior intraperitoneal, bounded above by the diaphragm, behind by the left triangular ligament and left lobe of the liver, the gastro-hepatic omentum and anterior surface of the stomach. To the right is the falciform ligament, and to the left the spleen, gastrosplenic omentum, and diaphragm. The common cause of an abscess here is following operations on the stomach, the tail of the pancreas, the spleen or the splenic flexure of the colon or in diverticulitis.

Left Posterior intraperitoneal is another name for the 'lesser' sac. The commonest type of suppuration here is the pancreatic pseudo-cyst (p. 950). In practice perforated gastric ulcer rarely causes a collection here, because the potential space is obliterated by adhesions.

Right Anterior intraperitoneal, which lies between the right lobe of the liver and the diaphragm. It is limited posteriorly by the anterior layer of the coronary and the right triangular ligaments, and to the left by the falciform ligament. Common causes here are perforating cholecystitis, a perforated duodenal ulcer, a duodenal cap 'blow out' (p. 843) and appendicitis.

Right Posterior intraperitoneal (*syn.* Rutherford Morison's kidney pouch) lies transversely beneath the right lobe of the liver. It is bounded on the right by the right lobe of the liver and the diaphragm. To the left is situated the foramen of Winslow, and below this lies the duodenum. In front are the liver and gall-bladder, and behind, the upper part of the right kidney and diaphragm. The space is bounded above by the liver, and below by the transverse colon and hepatic flexure. It is the deepest space of the four and the commonest site of a subphrenic abscess which usually arises from appendicitis, cholecystitis, a perforated duodenal ulcer or following upper abdominal surgery.

Extraperitoneal.—There are three of these.

Right and Left extraperitoneal which are terms given to perinephric abscesses (Chap. 56).

Midline extraperitoneal which is another name for the 'bare' area of the liver which may develop an abscess in amœbic hepatitis (p. 886) (the commonest cause) or it may be a pyogenic liver abscess (p. 885).

James Rutherford Morison, 1853–1939. Professor of Surgery, Durham, England.
Jacob Benignus Winslow, 1669–1760. Professor of Anatomy, Physic and Surgery, Paris.

Clinical Features.—The symptoms and signs of subphrenic infection are frequently obscure, and it is well to remember the aphorism, " Pus somewhere, pus nowhere else, pus under diaphragm ".

Symptoms.—A common history is that when some infective focus in the abdominal cavity has been dealt with, the condition of the patient improves temporarily, but after an interval of a few days or weeks, symptoms of toxæmia reappear. The condition of the patient steadily, and often rapidly, deteriorates. Sweating, wasting, and anorexia are present. There is sometimes epigastric fullness and pain, or pain in the shoulder on the affected side, owing to irritation of sensory fibres in the phrenic nerve, referred along the descending branches of the cervical plexus. Persistent hiccup may be a presenting symptom.

Signs.—If the abscess is anterior, abdominal examination will reveal some tenderness, rigidity, or even a palpable swelling. Sometimes the liver is displaced downwards, but more often it is fixed by adhesions. Examination of the chest is important, and in the majority of cases collapse of the lung or evidence of effusion or empyema is to be found.

FIG. 1061.—Radiograph in erect position showing subphrenic abscess containing gas.

Accessory Investigations.—(i) *Blood Count.*—A relative and absolute leucocytosis is the rule.

(ii) *X-ray.*—A plain radiograph sometimes demonstrates the presence of gas (fig. 1061) or a pleural effusion. On screening, the diaphragm is often seen to be elevated (so-called ' tented ' diaphragm) and its movements impaired. A lateral film is also useful to show the position of the ' tented ' area ascertained. This helps in deciding on the surgical approach to the abscess.

Differential Diagnosis.—Pylephlebitis, tropical abscess, pulmonary collapse, and empyema give rise to most of the diagnostic difficulties.

Treatment.—The clinical course of suspected cases is watched, and blood and radiological examinations are made at suitable intervals. If suppuration seems probable, surgical intervention is indicated.

If a swelling can be detected in the subcostal region or in the loin, an incision is made over the site of maximum tenderness, or over any area where œdema or redness is discovered. The parietes usually form part of the abscess wall, so that contamination of the general peritoneal cavity is unlikely.

If no swelling is apparent, the subphrenic spaces should be explored either by an anterior sub-costal approach or from behind after removal of the *outer part* of the twelfth rib according to the position of abscess if indicated on a lateral X-ray film (see above). With the posterior approach the pleura must *not* be opened, and after the fibres of the diaphragm have been separated a finger is inserted beneath the diaphragm so as to explore the adjacent area.

Thus both approaches must be *extra-serous* to avoid dissemination of pus into the peritoneal or pleural cavities.

When the cavity is reached, all the fibrinous loculi must be broken down with the finger and one or two drains or drainage tubes must be fully inserted. These drains

are withdrawn gradually during the next ten days and the closure of the cavity checked by X-ray sino-grams.

The appropriate antibiotics are given in support.

Post-operative Peritonitis.—The patient is ill, with raised pulse and peripheral circulatory failure. Following post-operative leakage from a suture line, the general condition of a patient is far more grave than if the patient had suffered leakage from a perforated peptic ulcer with no preceding operation. Local symptoms and signs are less definite. Abdominal pain may not be prominent, tenderness may be masked by the presence of a recent wound. The patient's deterioration may be wrongly attributed to pulmonary collapse which is usually concomitant.

Peritonitis follows abdominal operations more frequently than is realised. The principles of treatment do not differ from those of peritonitis of other origin. Antibiotic therapy alone is inadequate; no antibiotic can stay the onslaught of bacterial peritonitis due to leakage from a suture line, which must be dealt with appropriately.

Peritonitis in Patients under Treatment with Steroids.—Pain is frequently slight or absent. Physical signs are similarly vague and misleading.

Peritonitis in Children.—The diagnosis is in some ways more difficult, in other ways more easy, than in adults. If a history can be taken it is plain and unembroidered. Any physical signs elicited by a gentle, patient, and sympathetic examiner are meaningful.

Peritonitis in Senile Patients.—These can be as fractious as children and unable to give a reliable history. Abdominal tenderness is usually well localised, but guarding and rigidity are less marked because the abdominal muscles are thin and weak.

Bile Peritonitis.—Unless there is reason to suspect that a bile duct was damaged at an operation in which drainage was not provided, it is improbable that bile as a cause of peritonitis will be thought of until the abdomen has been opened and bile is seen therein. The common causes of bile peritonitis are: (1) following biliary surgery—damage to the common bile duct, slipping of a ligature on the cystic duct, leakage from a divided accessory bile duct in the gall-bladder bed or dislodgement of a T-tube drain in the early post-operative phase; (2) following perforation or gangrene of the gall-bladder or leakage from a choledochus cyst; and (3) following gastro-duodenal surgery, e.g. duodenal cap ' blow out ' (p. 843) or leakage from a suture line.

Unless the bile has extravasated slowly, and the collection becomes shut off from the general peritoneal cavity there are signs of diffuse peritonitis with a degree of shock. After a few hours a tinge of jaundice is not unusual. Local drainage, and when necessary suprapubic peritoneal drainage, is imperative, and if performed early enough, these measures will often save the patient's life. When bile is seen issuing from a perforation of some part of the biliary tree, a drainage tube should be passed through the opening and secured there. Infected bile is more lethal than sterile bile. The gall-bladder, if present, should be drained. A ruptured duodenum must be drained; it is too œdematous to repair.

Meconium Peritonitis.—Meconium is a sterile mixture of epithelial cells, mucin, salts, fats, and bile, and is formed when the fœtus commences to swallow amniotic fluid. By the third month of intra-uterine life the upper third of the small intestine has become filled with meconium ; by the fourth month the accumulation has reached the ileo-cæcal valve ; during the remainder of intra-uterine life the colon becomes increasingly filled.

Meconium peritonitis is an aseptic peritonitis which develops late in intra-uterine life or during, or just after, delivery. Meconium enters the peritoneal cavity through [redacted] of some form of neonatal intestinal obstruction; in the remainder no cause for the perforation is discernible. When meconium, which is sterile, enters the peritoneal cavity an exudate is secreted that organises rapidly; matting of intestinal loops occurs, and in many cases in a matter of weeks the extruded meconium becomes calcified.

Meconium remains sterile until about three hours after birth; thereafter, unless the perforation has become sealed, sterile meconium peritonitis gives place to acute bacterial peritonitis which, unless treated promptly, is rapidly fatal.

Clinical Features.—Meconium peritonitis should always be considered when a baby is born with a tense abdomen. There is vomiting, and failure to discharge meconium. The differential diagnosis between neonatal intestinal obstruction and peritonitis is, in many cases, virtually impossible; indeed, in half the cases both are present. Free fluid in the peritoneal cavity is often sufficient to give a fluid thrill.

Radiography (fig. 1062).—Free air in the peritoneal cavity, an abundant quantity of abdominal fluid, fluid levels, calcification (often most distinct on the surface of the liver or the spleen, and most readily seen in a lateral view) are characteristic findings, all of which are unlikely to be present in every case. Meconium peritonitis has been diagnosed by radiography of the fœtus *in utero* two days before birth.

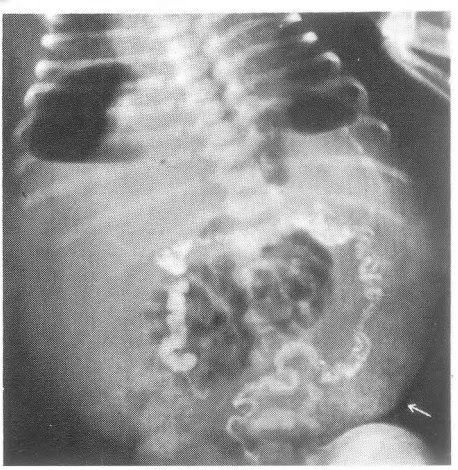


FIG. 1062.—Meconium peritonitis. Note: Free air and fluid in the peritoneal cavity; intra-abdominal calcification [↖] and on the spleen; air in the small intestine; microcolon shown by a barium enema. *(Dr. Jack Lester, Copenhagen.)*

Treatment.—The prognosis is bad, but recovery has followed prompt operation in some cases. The greatest chance of survival is in those patients who have an intestinal perforation but no intestinal obstruction, in which case closing the perforation and draining the peritoneal cavity is all-sufficient, and can be performed expeditiously.

Pneumococcal Peritonitis.—There are two forms of this disease:

1. Primary.
2. Secondary to pneumonia.

Primary pneumococcal peritonitis is much the more common. The patient is often an under-nourished girl between three and six years of age, and it is probable that the infection sometimes occurs via the vagina and Fallopian tubes, for pneumoccocci have been cultured from patients' vaginæ. At other times, and always in males, doubtless the infection is blood-borne from the upper respiratory tract or the middle ear. After the age of ten years pneumococcal peritonitis is most unusual. Children with nephritis are more liable to this condition than others. During the past thirty years the incidence of pneumococcal peritonitis has declined greatly and the condition is now rare. Perhaps this is on account of the greater cleanliness and higher standard of living of what was the poorer classes.

Clinical Features.—The onset is sudden, and the earliest symptom is pain localised to the lower half of the abdomen. The temperature is raised to 39·8° C or more, and there is usually frequent vomiting. After twenty-four to forty-eight hours profuse diarrhœa, occasionally blood-stained, is characteristic. There is usually increased frequency of micturition. The last two symptoms are due to severe

pelvic peritonitis. Herpes on the lip or nostril is often present. In acute forms of the disease, even in cases where there is no involvement of a lung, there is a tinge of cyanosis of the lips and cheeks, and movement of the alæ nasi is often discernible. On examination rigidity is usually bilateral, but is less than in most cases of acute appendicitis with peritonitis.

Differential Diagnosis.—A leucocytosis of 30,000/mm^3 (30 × 10^9/l) or more with approximately 90 per cent. polymorphs speaks more for pneumococcal peritonitis than appendicitis. Even so, it is often impossible, especially in males, to exclude perforated appendicitis. The other condition which is extremely difficult to differentiate from primary peritonitis in its early stage is pneumonia. An unduly high respiratory rate and the absence of abdominal rigidity are the most important signs supporting the diagnosis of pneumonia, which is usually clarified by a radiograph of the thorax.

Treatment.—*Early operation* is always required. After correcting dehydration and electrolyte imbalance, a short paramedian incision is made over the right rectus abdominis. The peritoneum is incised. Should the exudate be odourless and sticky, the diagnosis of pneumococcal peritonitis is practically certain, but it is desirable to perform a routine laparotomy to exclude other lesions. Assuming that no other cause for the peritonitis is discovered, some of the exudate is removed with a syringe, and sent to the laboratory for culture and sensitivity tests. Thorough peritoneal toilet is carried out and the incision closed. The patient is returned to bed and antibiotic and fluid replacement therapy continued. Gastrointestinal suction drainage is essential.

Primary streptococcal peritonitis of infants and children is rather more frequent than the foregoing but still uncommon. When a streptococcus is the infecting organism, the peritoneal exudate is thin, slightly cloudy, and contains flecks of fibrin. From the standpoints of clinical aspects and treatment, streptococcal peritonitis in infants and children does not differ from those detailed in the account of pneumococcal peritonitis (*vide supra*), but the mortality is higher.

Idiopathic streptococcal peritonitis in adults is fortunately rare, for prior to the antibiotic era it was nearly always fatal, and the mortality is still very high. Rightly, in early cases the abdomen is opened, usually on a diagnosis of acute appendicitis. In streptococcal peritonitis the peritoneal exudate is odourless, thin, contains small flecks of fibrin, and may be blood-stained. In these circumstances pus is removed by suction, the abdomen closed with suprapubic drainage, and the measures detailed in the conservative treatment of peritonitis carried out.

Peritonitis following Abortion Parturition.—The abortionist has usually pushed an instrument through the uterine vault and streptococcal peritonitis follows. When peritonitis follows puerperal infection, it is a notifiable disease. It is more common after first deliveries. Rigidity is seldom much in evidence; this, at any rate in part, is due to the stretched condition of the abdominal musculature. The lochia may be offensive but not necessarily so. Diarrhœa is common.

Treatment.—Provided the infection is limited strictly to the pelvis, the correct treatment is rest for the alimentary canal, intravenous fluids, the required antibiotic, and attention to electrolyte balance. Posterior colpotomy may be necessary if a pelvic abscess forms. If the peritonitis is generalised, the patient is usually extremely ill and drainage is advisable. This is best carried out by making a small suprapubic incision under local anæsthesia and inserting a drain. This can be done with the patient in bed, if necessary.

In the pre-antibiotic era the mortality of general peritonitis following parturition or abortion was at least 50 per cent; with antibiotic therapy and timely operation, the mortality has fallen to less than 10 per cent (Brews).

Periodic peritonitis is characterised by abdominal pain and tenderness, mild pyrexia, polymorphonuclear leucocytosis, and occasionally pain in the thorax and joints. The duration of an attack is twenty-four to seventy-two hours, when it is followed by complete remission, but exacerbations recur at regular intervals. Most of the patients have undergone appendicectomy in childhood. The disease, often familial, is limited principally to Arabs, Armenians, and Jews; other peoples occasionally are affected. At laparotomy, which may be necessary to exclude other causes, the peritoneum—particularly in the vicinity of the spleen and the gall-bladder—is inflamed. There is no evidence that the interior of these organs is abnormal.

Richard Alan Brews, 1902–1965. Obstetric and Gynæcological Surgeon, The London Hospital.

Differential Diagnosis.—Patients with abdominal epilepsy do not have positive physical signs or pyrexia, and their attacks are usually controlled by anti-convulsive medication.

The ætiology of periodic peritonitis is unknown, and no form of treatment has been found to be of the slightest avail.

Usually children are affected, but it is not rare for the disease to make its first appearance in early adult life, when females outnumber males by two to one. Exceptionally the disease becomes manifest in patients over forty years of age.

TUBERCULOUS PERITONITIS

Acute Tuberculous Peritonitis.—Tuberculous peritonitis sometimes has an onset that resembles so closely acute peritonitis that the abdomen is opened. Straw-coloured fluid escapes, and tubercles[1] are seen scattered over the peritoneum and greater omentum. Tubercles occasionally simulate fat necroses (p. 941) or the nodules of peritoneal carcinomatosis (p. 980). On opening the abdomen and finding tuberculous peritonitis, the fluid is evacuated, some being retained for bacteriological study, a portion of the diseased omentum is removed for histological confirmation of the diagnosis, and the wound closed without drainage.

At other times, although acute abdominal symptoms arise, the presence of ascites makes the diagnosis of acute tuberculous peritonitis reasonably evident.

Chronic Tuberculous Peritonitis.—Although the incidence of tuberculous peritonitis has declined in Britain, in many parts of the world where measures for eradicating tuberculosis (especially the disease in cows) are enforced less strictly, the condition still occurs.

Presentation.—Abdominal pain is present in 90%, fever in 60%, loss of weight 60%, ascites 60%, night sweats 37%, abdominal mass 26%.

Origin of the infection:

1. From tuberculous mesenteric lymph nodes.
2. From tuberculosis of the ileo-cæcal region.
3. From a tuberculous pyosalpinx.
4. Very occasionally it is due to a blood-borne infection from pulmonary tuberculosis.

There are four varieties of tuberculous peritonitis:

1. **Ascitic Form.**—The peritoneum is studded with tubercles, and the peritoneal cavity becomes filled with pale, straw-coloured fluid. The onset is insidious. There is loss of energy, facial pallor, and some loss of weight. The patient is usually brought for advice because of enlargement of the abdomen. Pain is often completely absent; in other cases there is considerable abdominal discomfort which may be associated with constipation or diarrhœa. On inspection dilated veins can be seen coursing beneath the skin of the abdominal wall. Shifting dullness can be elicited readily. In the male child congenital hydroceles sometimes appear, due to the patent processi vaginales becoming filled with ascitic fluid from the peritoneal cavity. Because of the increased intra-abdominal pressure, an umbilical hernia commonly occurs. On abdominal palpation a transverse solid mass can often be detected. This is rolled-up greater omentum infiltrated with tubercles.

The diagnosis is seldom difficult, except when it occurs in an acute form or when it first appears in an adult, in which case it has to be differentiated from other forms of ascites, especially from malignant secondary deposits (p. 980). A positive Mantoux test in a child with ascites strongly suggests, and a negative test is good evidence against, tuberculosis. In adults this test is of negligible value. The diagnosis of tuberculous peritonitis having been made, it is always important to look for tuberculous disease elsewhere, and in this respect the possibility of tuberculous salpingitis in females should be remembered.

The fluid is pale yellow, usually clear, and rich in lymphocytes. The specific gravity is comparatively high, often 1·020 or over. Even after centrifugation, rarely

[1] Early tubercles are greyish and translucent. They soon undergo caseation, and appear white or yellow, and are then less difficult to distinguish from carcinoma.

Charles Mantoux, 1877–1947. Physician, Le Cannet, Alpes Maritimes, France.

can the *mycobact. tuberculosis* be found, but its presence can be demonstrated by culture or by guinea-pig inoculation.

Treatment.—Sanatorium treatment is helpful.

Chemotherapy.—Streptomycin, P.A.S., isoniazid or other drugs are given as detailed on p. 37. If the general condition is good, the patient can return home and, if an adult, to light work, before the course of chemotherapy has been completed.

2. **Encysted** (*syn.* **Loculated**) **form** is similar to the above, but one part of the abdominal cavity alone is involved. Thus is produced a localised intra-abdominal swelling which gives rise to difficulty in diagnosis. In a female above the age of puberty, when the swelling is in the pelvis, an ovarian cyst will probably be diagnosed. In the case of a child it is sometimes difficult to distinguish the swelling from a mesenteric cyst. For these reasons laparotomy is often performed, and if an encapsulated collection of fluid is found, it is evacuated and the abdomen is closed. The general treatment already detailed is required, but the response to this treatment is more rapid. Late intestinal obstruction is a possible complication.

3. **Fibrous** (*syn.* **plastic**) **form** is characterised by the production of widespread adhesions, which cause coils of intestine, especially the ileum, to become matted together and distended. These distended coils act as a 'blind loop' (p. 996) and give rise to steatorrhœa, wasting and attacks of abdominal pain. On examination the adherent intestine with omentum attached, together with the thickened mesentery, may give rise to a palpable swelling or swellings. The first intimation of the disease may be subacute or acute intestinal obstruction. Sometimes the cause of the obstruction can be remedied easily by the division of bands. Lateral anastomosis between an obviously dilated loop and a collapsed loop of small intestine should not be done or the 'blind loop' syndrome (p. 996) is a certain outcome. If the adhesions are accompanied by fibrous strictures of the ileum as well, it is best to excise the affected bowel, provided not too much of the small intestine needs to be sacrificed. If adhesions only are present a Noble plication operation (p. 1048) should be done. This is a dangerous condition and the surgeon must adhere to strict physiological principles in its treatment. Fortunately, chemotherapy after adequate surgery will rapidly bring it under control.

4. **Purulent form** is rare. When it occurs, usually it is secondary to tuberculous salpingitis. Amidst a mass of adherent intestine and omentum, tuberculous pus is present. Sizeable cold abscesses are wont to form, and point on the surface, commonly near the umbilicus, or burst into the bowel. In addition to prolonged general treatment, operative treatment may be necessary for the evacuation of cold abscesses and possibly for intestinal obstruction. If the patient survives long enough to overcome the infection, it may be possible to close a fæcal fistula, which otherwise usually persists because of obstruction distal to it. Closure must therefore be combined with some form of anastomosis between the segment of intestine above the fistula and an unobstructed area below. The prognosis of this variety of tuberculous peritonitis is relatively poor.

SCLEROSING PERITONITIS

Practolol, a β-adrenergic blocking agent used in the treatment of cardiovascular disorders, has been found to be associated with the late development of a sclerosing condition in the peritoneum which may cause intestinal obstruction. At laparotomy the condition resembles plastic tuberculous peritonitis. It may be confused with mesothelioma or carcinoid syndrome. Lysis of the adhesions is indicated. The condition may cause recurrent obstruction.

PERITONEAL BANDS AND ADHESIONS

Congenital bands and membranes occur in the peritoneum as described in textbooks of anatomy. None of them causes intestinal obstruction except an obliterated vitello-intestinal duct (p. 1128).

Peritoneal adhesions are discussed on p. 1047.

Talc Granuloma.—Talc (silicate of magnesium) should never be used as a lubricant for rubber gloves for it is a cause of peritoneal adhesions and granulomas in the Fallopian tubes. Potassium bitartrate, which is completely soluble, is free from these serious objections. *Starch peritonitis* (see p. 980).

ASCITES

Ascites, an excess of serous fluid within the peritoneal cavity, can be recognised clinically only when the amount of fluid present exceeds 1,500 ml.; in the obese a greater quantity than this is necessary before there is clear evidence of the presence of intraperitoneal fluid.

Mechanism of Ascites.—The balanced effects of plasma and peritoneal colloid osmotic pressures and hydrostatic pressures determine the exchange of fluid between the capillaries and the peritoneal fluid. Normal intraperitoneal pressure and normal peritoneal fluid colloid osmotic pressure cannot be measured. Protein-rich fluid enters the peritoneal cavity when capillary permeability to protein is increased, as in peritonitis and carcinomatosis peritonei. Capillary pressure may be increased because of generalised water retention, cardiac failure, constrictive pericarditis, or vena caval obstruction. Capillary pressure is raised selectively in the portal venous system in the Budd-Chiari syndrome (p. 893), portal cirrhosis, or extrahepatic venous obstruction.

Plasma colloid osmotic pressure may be lowered in patients with reduced intake, diminished absorption, abnormal losses, or defective protein synthesis as occurs in cirrhosis.

Clinical Features.—The abdomen is distended evenly, with fullness of the flanks, which are dull to percussion. Usually shifting dullness is present, but when there is a very large accumulation of fluid, this sign is absent. In such cases, on flicking the abdominal wall, a characteristic fluid thrill is transmitted from one side to the other. In the female, ascites must be differentiated from an enormous ovarian cyst.

Type 1. *Due to Congestive Heart Failure.*—This, the commonest cause of ascites, is due to chronic venous stasis in the thoracic segment of the inferior vena cava, and consequent obstruction to the venous outflow from the liver. There is stasis also in the superior vena cava, as evinced by engorgement of the veins of the neck—a striking sign in this condition. The ascitic fluid is light yellow serum of low specific gravity, about 1·010.

Type 2. *Due to Hepatic or Biliary Cirrhosis.*—In cirrhosis there is obstruction to the venous outflow of the liver due to obliterative fibrosis of the intrahepatic venous bed (p. 894). Lymph flow may be increased.

Type 3. *Due to Tuberculous Peritonitis* (p. 977).

Type 4. *Secondary Carcinoma of the Peritoneum.*—Again the ascites is due to excessive outpouring, due to lymphatic blockage. The fluid is dark yellow and frequently blood-stained. The specific gravity is high—1·020 or over. Microscopical examination often reveals cancer cells.

Type 5. *Chronic Constrictive Pericarditis* (syn. *Pick's Disease*).—In addition to the peritoneal effusion, effusions occur into the pleural cavity. These effusions are due to engorgement of the venæ cavæ consequent upon diminished capacity of the right side of the heart.

Type 6. *Due to depletion of blood protein* consequent upon albuminuria or starvation. The ascites in this instance is due to alterations in the osmotic pressure of the capillary blood.

Type 7. *Meigs' Syndrome.*—This is ascites and pleural effusion associated with solid fibroma of the ovary. The effusions disappear when the tumour is excised.

Friedel Pick, 1876–1926. Professor of Laryngology, Prague, Czechoslovakia. (*See footnote, p. 769.*)
Joe Vincent Meigs, 1892–1964. Gynæcological Surgeon, Massachusetts General Hospital, Boston, Mass., U.S.A.

Treatment.—Ascites may be tapped (paracentesis abdominis) but unless other measures are taken, the fluid soon re-accumulates and repeated tappings remove valuable protein. Dietary sodium restriction to 200 mg per day is frequently successful. Diuretics may be effective in selected patients.

If portal venous pressure is raised, it may be possible to lower it by treatment of the primary condition. (See Chapter 45).

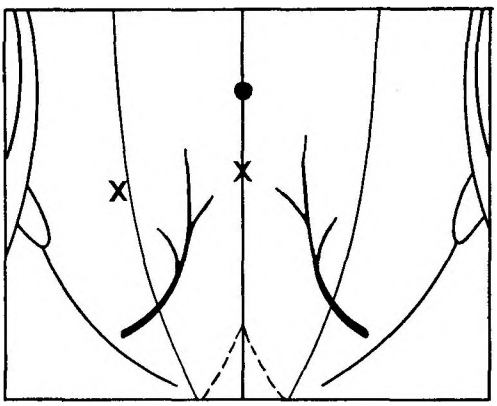


FIG. 1003.—Usual points of puncture for tapping ascites. The bladder must be emptied by a catheter before the puncture is made. Note the relationship of the sites of puncture to the inferior epigastric artery. (*Drs. V. C. and V. V. Shah, Jamnagar, India.*)

Paracentesis Abdominis.—The bladder having been emptied by a catheter, under local anæsthesia puncture of the peritoneum is carried out with a moderate-sized trocar and cannula at one of the points shown in fig. 1063. In cases where the effusion is due to cardiac failure the fluid must be evacuated slowly. In other circumstances this precaution is unnecessary. If the cannula becomes blocked with fibrin, it is cleared with a stylet. After the fluid has been evacuated the puncture is sealed, and a tight binder is applied to the abdomen.

Permanent Drainage of Ascitic Fluid.—In rare cases where ascites accumulates rapidly after paracentesis, and the patient is otherwise fit, permanent drainage of the ascitic fluid renders the patient more comfortable. A number of procedures have been described, the best of which is probably the operation of ileoentectropy (segmental eversion of the ileum). A 40-cm loop of isolated terminal ileum is opened along its anti-mesenteric border, turned inside out and sutured with catgut in the triangular area of posterior abdominal wall between the root of the mesentery and the right colon. The ileal mucosa then absorbs the ascitic fluid (Neumann). To prevent its adhesion to the ileal mucosa, the greater omentum should be removed.

STARCH PERITONITIS

Like talc (p. 978), starch powder has found disfavour as a surgical glove lubricant. In a few starch-sensitive patients it causes a painful ascites, fortunately of limited duration. Should laparotomy be performed, any small granulomas in, say, the omentum, will be found to contain birefringent starch particles.

PERITONEAL LOOSE BODIES (Peritoneal mice)

Peritoneal loose bodies almost never cause symptoms. One or more may be found in a hernial sac or in the pouch of Douglas. The loose body may come from an appendix epiploica that has undergone axial rotation, followed by necrosis of its pedicle, and detachment, but they are also found in those who suffer from subacute attacks of pancreatitis. These hyaline bodies attain the size of a pea or bean, and contain saponified fat surrounded by fibrin.

NEOPLASMS OF THE PERITONEUM

Carcinoma peritonei is a common terminal event in many cases of carcinoma of the stomach, colon, ovary, or other abdominal organs and also of the breast. The peritoneum, both parietal and visceral, is studded with secondary growths, and the peritoneal cavity becomes filled with clear, straw-coloured, or blood-stained ascitic fluid.

Charles G. Neumann, Contemporary. Associate Professor of Clinical Surgery, New York University, U.S.A.
James Douglas, 1675–1742. Anatomist and Obstetrician, London.

The main forms of peritoneal metastases are:

1. Discrete nodules. This is by far the most common variety.
2. Plaques varying in size and colour.
3. Diffuse adhesions. This form occurs at a late stage of the disease, and gives rise, sometimes, to a ' frozen pelvis '.

Gravity probably determines the distribution of free malignant cells within the peritoneal cavity. Cells not caught in peritoneal folds along the attachments of mesenteries gravitate into the pelvic pouches or into a hernial sac, enlargement of which is occasionally the first indication of the condition. Implantation occurs also on the greater omentum, the appendices epiploicæ, and the inferior surface of the diaphragm.

Differential Diagnosis.—Early discrete tubercles common in tuberculous peritonitis are greyish and translucent, and closely resemble the discrete nodules of peritoneal carcinomatosis, but the latter feel hard when rolled between the finger and the thumb, making the differential diagnosis tolerably simple. Fat necroses usually can be distinguished from carcinomatous nodules by their opacity. Peritoneal hydatids can also simulate malignant disease after rupture of a hydatid cyst.

It is remarkable how often patients riddled with intraperitoneal carcinoma preserve their nutrition, and look and feel comparatively well until the terminal stage.

Treatment.—Ascites due to carcinomatosis of the peritoneum can be considerably ameliorated by instillations of radio-active gold.

Radio-active gold (^{198}Au) has a half-life of two and a half days, and is supplied as a purple colloidal solution. One hundred millicuries or more of the solution are introduced into the peritoneal cavity after paracentesis. To improve distribution the foot of the bed is raised; then the patient lies on one side and then on the other, and finally prone, each for fifteen minutes. There follows a period of nausea, but approximately half the patients so treated are benefited for a time. The treatment is of no avail if, after paracentesis, the secondary deposits can be palpated, as the isotope can penetrate only to a depth of 1 millimetre.

Radio-active chromic phosphate (^{32}P) is also supplied as a colloidal solution, and is as effective as gold. Not only is it less expensive, but this solution requires none of the troublesome precautions connected with the protection of personnel. It emanates pure beta radiation, and has a longer half-life, viz. 14·3 days. In its administration rubber gloves are the only protection needed.

Pseudomyxoma Peritonei.—This rare condition occurs more frequently in females. The abdomen is filled with a yellow jelly, large quantities of which are often more or less encysted. The condition arises in one of two ways: more often from rupture of a pseudomucinous cyst of the ovary, less often from rupture of a mucocele of the appendix. It is painless, and there is no impairment of general health for a long time. When the condition arises from the appendix the mass is often more localised, but in cases of ovarian origin the whole peritoneal cavity is involved. Although an abdomen distended with what seems to be fluid that cannot be made to shift should suggest the possibility, it is highly improbable that a correct pre-operative diagnosis will be made. At laparotomy masses of jelly are scooped out, and the primary focus, if it can be found, is removed. Unfortunately, recurrence is usual. When pseudomyxoma peritonei arises from a mucocele of the appendix, repeated recurrence is less common. Pseudomyxoma peritonei is locally malignant

active isotopes, which certainly should be employed in recurrent cases.

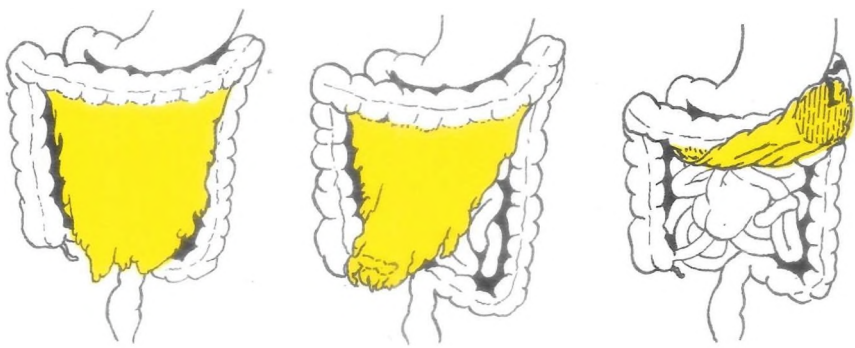
Mesothelioma.—As in the pleural cavity this is a highly malignant tumour. Asbestos is a recognised cause. It has a predilection for the pelvic peritoneum and may mimic pubo-prostatic carcinoma, clinically. It is not radio-sensitive. Alkylating agents have given remissions. Benign forms are reported.

PERITONEOSCOPY (LAPAROSCOPY)

The peritoneoscope resembles a cystoscope. Preferably under general anæsthesia, a midline incision 6 mm. long is made close to the umbilicus. A trocar and cannula are pushed into the peritoneal cavity and the requisite amount of air (1 to 1½ litres) is introduced with a pneumoperitoneum apparatus. The patient is tilted so that the air will rise into the upper abdomen or the pelvis, as required. The telescope is inserted via a special cannula and the organs inspected. Biopsy of a particular area of the liver is possible, but hæmorrhage from it is not infrequent. Air embolism is another possible danger.

THE GREATER OMENTUM

Rutherford Morison called the greater omentum 'the abdominal policeman.'[1] Relatively larger and structurally more substantial in the adult than in the child, the discharge of its life-saving constabulary duties becomes more effective after puberty, and remains unabated throughout life. The greater omentum attempts, often successfully, to limit intraperitoneal infective and other noxious processes (fig. 1064). For instance, an acutely



(*a*) (*b*) (*c*)

FIG. 1064.—The greater omentum. (*a*) Normal. (*b*) In appendicitis. (*c*) In a (comparatively small) laceration of the spleen.

inflamed appendix is often found wrapped in omentum, and this saves many a patient from developing diffuse peritonitis. Some sufferers from herniæ are also greatly indebted to this structure, for it often plugs the neck of a hernial sac and prevents a coil of intestine from becoming strangulated.

Apart from a small portion of it becoming gangrenous while performing the last-mentioned duty (strangulated omentocele), this good Samaritan[2] of the peritoneal cavity seldom itself becomes diseased; when it does become overwhelmed, as in tuberculous peritonitis and carcinomatosis peritonei, it becomes rolled like a scroll.

Torsion of the Omentum.—Torsion of the omentum (fig. 1065) is a rare emergency, and consequently is seldom diagnosed correctly. It is usually mistaken for appendicitis with somewhat abnormal signs. It may be primary or secondary to

[1] But it has not any feet, i.e. it does not move across the abdomen of its own volition, but passively due to peristalsis, and it may be pushed by the movements of the abdominal wall into an area of immobility (rigidity) where there is local peritoneal irritation.

[2] Samaritan, an inhabitant of Samaria, Jordan. The parable of the Good Samaritan is told in the Bible, *Luke x*, 30–37.

an adhesion of the omentum, to an old focus on infection, or to a hernia. Successive herniations of a portion of the omentum into a hernial sac of irregular bore are credited with giving the necessary stimulus to omental torsion.

The patient is most frequently a middle-aged, obese male. A tender lump may be present in the abdomen. The blood supply having been jeopardised, the twisted mass sometimes becomes gangrenous, in which case bacterial peritonitis soon follows.

Treatment.—The abdomen having been opened, the pedicle above the twist is ligated securely and the mass removed.

Omental Cyst (p. 988).

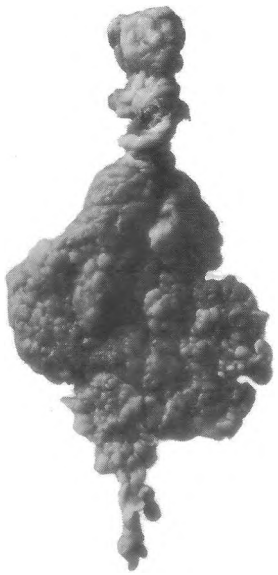


FIG. 1065.—Torsion of the greater omentum. Specimen removed by operation. Actual size. (*Archibald Ronald, F.R.C.S., Barrow-in-Furness.*)

THE MESENTERY

A wound of the mesentery can follow a severe abdominal contusion,[1] and is a cause of hæmoperitoneum. In about 60% of cases the mesenteric laceration is associated with a rupture of the intestine. If the tear is a large one, and especially if it is transverse (fig. 1066), the blood supply to the neighbouring intestine is cut off, and a limited resection of gut is imperative. Small wounds and wounds in the long axis (fig. 1067) should be sutured.

Torsion of the Mesentery (see Volvulus Neonatorum, p. 1040, and Volvulus of the Small Intestine, p. 1045).

Embolism and Thrombosis of the Mesenteric Vessels (p. 1051).

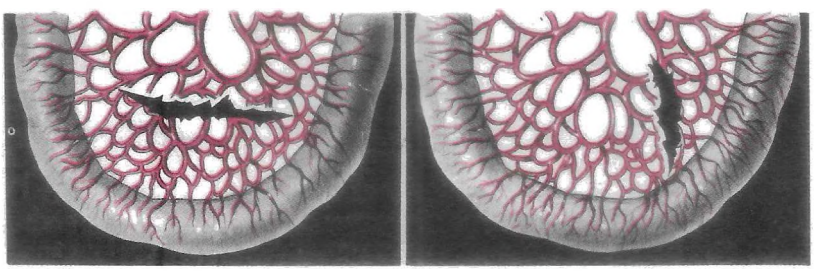


FIG. 1066 FIG. 1067

Laceration of the mesentery, a common injury in car accidents. A transverse tear (fig. 1066) often imperils the blood supply of a segment of intestine, making resection necessary. A longitudinal tear (fig. 1067) can be closed by suture.

ACUTE NON-SPECIFIC ILEO-CÆCAL MESENTERIC ADENITIS

Ætiology.—Non-specific mesenteric adenitis was so named to distinguish it from specific (tuberculous) mesenteric adenitis. It is now very much commoner than the tuberculous variety. Despite much investigation, the

[1] *Seat Belt Syndrome.*—If a car accident occurs when a seat belt is worn, sudden deceleration can result in a torn mesentery. This possibility should be borne in mind particularly as multiple injuries may distract attention from this injury. If there is any bruising of the abdominal wall, or even marks of clothing impressed into the skin, laparotomy is indicated.

ætiology remains unknown. As so often happens in other inflammatory diseases when no causative bacterium can be found, an unidentified virus is blamed. In about 25% of cases a respiratory infection precedes an attack of acute non-specific mesenteric adenitis. In spite of the fact that the vermiform appendix is not diseased in this condition, which is definitely recurrent, appendicectomy does reduce the incidence of further attacks, perhaps by removing what is sometimes known as ' the abdominal tonsil '. This self-limiting disease is never fatal.

Living Pathology.—There is a small increase in the amount of peritoneal fluid. As seen and felt between the leaves of the mesentery, the ileo-cæcal mesenteric lymph nodes are enlarged, being firmly elastic and usually about the size of a haricot bean. In very acute cases they are distinctly red, and many of them are the size of a walnut. The nodes nearest the attachment of the mesentery are the largest. They are not adherent to their peritoneal coats, and if a small incision is made through the overlying peritoneum, a node is extruded easily.

Clinical Features.—During childhood, acute non-specific mesenteric adenitis is a common condition, the ratio of acute appendicitis to acute non-specific adenitis being about 10:1. It is unusual after puberty, but is sometimes seen in teenage girls (nurses syndrome). The typical history is one of short attacks of central abdominal pain lasting from 10–30 minutes, and associated with circum-oral pallor. They tend to come on when the patient is tired. Vomiting is usual, but there is no alteration of bowel habit.

On Examination.—There are spasms of general abdominal colic, usually referred to the umbilicus, with intervals of complete freedom, which never appertains in obstructive appendicitis. The patient seldom looks ill. In more than half the cases the temperature is elevated; in severe examples it exceeds 38·3°C. Abdominal tenderness is greatest along the line of the mesentery. When present, shifting tenderness is a valuable sign for differentiating the condition from appendicitis. After laying the patient on the left side for a few minutes, the maximum tenderness moves to the left of the original site.

The pelvic peritoneum is tender to rectal palpation in 30% of cases. The neck, axillæ, and groins should be palpated for enlarged lymph nodes—if these nodes are enlarged, brucellosis[1] should come to mind (p. 985).

Leucocyte Count.—There is often a leucocytosis of 10–12,000/mm^3 (10 – 12^9/l) or more on the first day of the attack, but this falls on the second day.

Treatment.—When the diagnosis can be made with assurance, bed rest for a few days is the only treatment necessary. If at a second examination, an hour or two after confinement to bed, acute appendicitis cannot be excluded, it is safer to perform appendicectomy.

Tuberculous mesenteric lymphadenitis is considerably less common than acute non-specific lymphadenitis, and it has become increasingly less

[1] Sir David Bruce described Malta Fever in 1887.

Sir David Bruce, 1855–1931. Major-General, Royal Army Medical Service.

frequent in Britain during the past thirty years. Tubercle bacilli, usually but not necessarily bovine, are ingested, and enter the mesenteric lymph nodes by way of Peyer's patches. It is possible for one draught of raw milk to start the infection; it is equally possible that a toddler can become infected with human tubercle bacilli by placing one dust-covered small object in its mouth. Sometimes only one lymph node is infected; usually there are several; occasionally massive involvement occurs (fig. 1068).

Presentation:

1. **Demonstrated Radiologically.**—The shadows cast by one or more calcified tuberculous lymph nodes are seen frequently in a plain radiograph of the abdomen (fig. 1069). Often the shadow cast by such a lymph node or nodes is situated in the

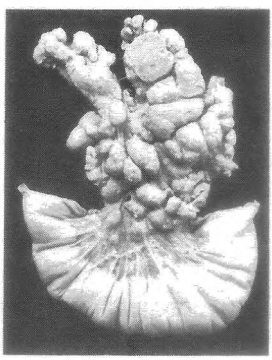


FIG. 1068.—Massive tuberculosis of the lymph nodes of the mesentery.

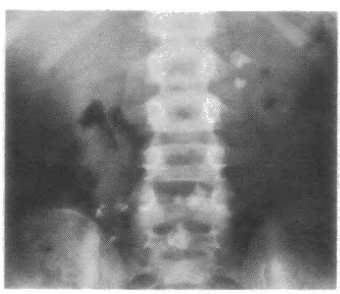


FIG. 1069.—Radiograph showing calcified tuberculous lymph nodes of the mesentery. (*Dr. H. R. E. Wallis, Bath.*)

ileo-cæcal region, but nearly as many are displayed along the line of attachment of the mesentery. Usually the radiological characteristics are unmistakable. Each node is round or oval, not homogenous, but mottled, and its outline is not regular, but bosselated like a blackberry. Calcification of these lymph nodes occurs at the earliest in eighteen months. It is often assumed that because a tuberculous lymph node is calcified, the infection is necessarily defunct. Especially in children, this assumption may not be valid. Firstly, radio-opaque lymph nodes (fig. 1069) do not become white in a single night,[1] and secondly, calcification does not eliminate the presence of uncalcified tuberculous sister lymph nodes.

2. **As a Cause of General Symptoms.**—Less frequently the tuberculous process is more active, and gives rise to general symptoms. The patient, usually a child under ten years of age, loses appetite, looks pale, and there is some loss of weight; sometimes evening pyrexia occurs. In children with these symptoms, especially those who live in the country, if the Mantoux test is negative, brucellosis, the 'disease of mistakes,' should be thought of, and an intradermal test with brucellin performed.

3. **As a Cause of Abdominal Pain.**—Sometimes abdominal pain is the cause of the patient being brought for advice; usually this pain is central, not severe, but rather a discomfort, and is often constant. On examination the abdomen is somewhat protuberant and there is tenderness on deep pressure to the right of the umbilicus. In these circumstances the condition resembles acute non-specific mesenteric lymphadenitis. On deep palpation inflamed mesenteric lymph nodes are sometimes palpable

[1] My hair is grey, but not with years,
Nor grew it white
In a single night,
As men's have grown from sudden fears:
Byron, *The Prisoner of Chillon.*

Johann Conrad Peyer, 1653–1712. Professor of Logic, Rhetoric and Medicine, Schaffhausen, Switzerland.

as firm, discrete, tender bean-like objects most frequently to the right of and near the umbilicus. In both conditions a normal leucocyte count favours tuberculosis, and in a child a positive Mantoux test is confirmatory evidence of tuberculosis.

4. **Symptoms Indistinguishable from those of Appendicitis.**—On occasions the abdominal pain is acute and may be accompanied by vomiting. This, combined with tenderness and some rigidity in the right iliac fossa, makes the diagnosis from subacute appendicitis almost impossible. When, as is sometimes the case, the tuberculous infection of the mesenteric lymph nodes becomes reactivated in adolescent or adult life, the diagnosis difficulties are even greater. A radiograph may show calcified lymph nodes, but as such a condition can co-exist with appendicitis, in some cases laparotomy for appendicectomy and visualisation of the lymph nodes is necessary. If the mesentery is found to be in an inflamed state with caseation of some of the lymph nodes, the diagnosis of active tuberculosis of the nodes is confirmed.

Treatment is similar to that of other surgical tuberculosis (p. 607).

Most cases subside, but from time to time a local abscess forms, usually in the right iliac fossa, when the tuberculous pus should be evacuated and the abdomen closed without drainage (see Pseudo-mesenteric Cyst, below).

Tuberculous Mesenteric Lymph Nodes as a Cause of Intestinal Obstruction.—Remote, rather than recent, tuberculous mesenteric adenitis can be the cause of intestinal obstruction. For instance, a coil of small intestine becomes adherent to a caseating node, and is thereby angulated (fig. 1070), or a free coil may become imprisoned in the tunnel beneath the site of adherence and the mesentery.

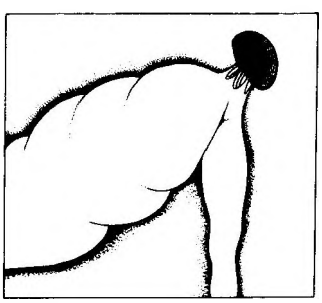


FIG. 1070. — Obstruction by angulation. The gut has become adherent to a tuberculous lymph node.

As a Cause of Pseudo-mesenteric Cyst.—When tuberculous mesenteric lymph nodes break down, the tuberculous pus may remain confined between the leaves of the mesentery, and a cystic swelling having the characteristics of a mesenteric cyst is found. When such a condition is confirmed at operation the tuberculous pus should be aspirated without soiling the peritoneal cavity, the wound closed, the sensitivity of the organism should be sought and specific treatment continued until the infection has been overcome.

Calcifying Mesenteric Lymph Nodes as a Confusing X-ray Shadow.—Sometimes the shadow cast by a calcified mesenteric lymph node or nodes simulates that of a ureteric or renal calculus. A change of posture often causes a lymph-node shadow to alter in position. Pyelography clarifies the diagnosis in doubtful cases.

Carcinoid Tumour of the Ileum (p. 1016) may give rise to yellowish enlarged glands in the ileo-cæcal mesentery. The diagnosis is usually only made at laparotomy.

MESENTERIC CYSTS

Classification:
- Chylolymphatic.
- Enterogenous.
- Urogenital remnant.
- Dermoid (teratomatous cyst).

Chylolymphatic cyst, the commonest variety of mesenteric cyst, probably arises in congenitally misplaced lymphatic tissue that has no efferent communication with the lymphatic system: it arises most frequently in the mesentery of the ileum. The thin wall of the cyst, which is composed of connective tissue lined by flat endothelium, is filled with clear lymph or, less frequently, with chyle varying in consistency from watered milk to cream. Occasionally the cyst attains a great size (fig. 1071). More often unilocular than multilocular, a chylolymphatic cyst is almost invariably solitary, although there is an extremely rare variety in which myriads of cysts are found in the various mesenteries of the abdomen. A chylolymphatic cyst has a blood supply independent of that of the adjacent intestine, thereby enucleation is possible without the necessity of resection of gut.

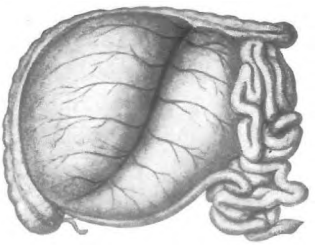


FIG. 1071.—A large chylolymphatic mesenteric cyst. (*Higgins and Lloyd.*)

Enterogenous cyst is believed to be derived either from a diverticulum of the mesenteric border of the intestine which has become sequestrated from the intestinal canal during embryonic life, or from a duplication of the intestine. An enterogenous cyst has a thicker wall than a chylolymphatic cyst, and it is lined by mucous membrane, sometimes ciliated. The content is mucinous, and is either colourless or yellowish-brown from bygone hæmorrhage into the cyst. As can be seen at operation, the muscle in the wall of an enterogenous cyst and the bowel with which it is in contact have a common blood supply; consequently removal of the cyst always entails resection of the related portion of intestine.

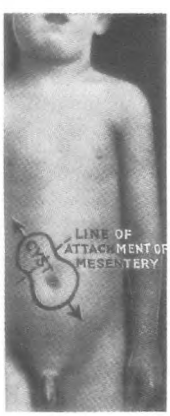


FIG. 1072.—A mesenteric cyst moves freely in the direction of the arrows, i.e. at right angles to the attachment of the mesentery.

Clinical Features of a Mesenteric Cyst.—A mesenteric cyst is encountered most frequently in the second decade of life, less often between the ages of one and ten years, and exceptionally in infants under one year.

The patient presents on account of:

(*a*) *A painless abdominal swelling.*—A cyst of the mesentery presents characteristic physical signs.

1. There is a fluctuant swelling near the umbilicus.

2. The swelling moves freely in a plane at right angles to the attachment of the mesentery (fig. 1072).

3. There is a zone of resonance around and, classically, a belt of resonance across the cyst.

(*b*) *Recurrent attacks of abdominal pain* with or without vomiting. The pain results from recurring temporary impaction of a food bolus in a segment of bowel narrowed by the cyst, or possibly from torsion of the mesentery.

(*c*) *An acute abdominal catastrophe* arises as a result of (1) torsion of that portion of the mesentery containing the cyst; (2) rupture of the cyst, often due to a comparatively trivial accident; (3) hæmorrhage into the cyst; (4) infection.

Radiography.—In most instances the patient should be submitted to X-ray after a barium meal. The hollow viscera will be found to be displaced around the cyst, and not infrequently some portion of the lumen of the small intestine will be narrowed. In order to exclude or confirm the diagnosis of a hydronephrosis an excretory pyelogram should not be omitted. In cases of painless enlargement of the abdomen this examination should be undertaken first.

Treatment.—As has been indicated already, many chylolymphatic cysts can be enucleated *in toto.*

When, after aspiration of about half the contents of the cyst, the major portion of the cyst has been dissected free but one portion abutting on the intestine or a major blood-vessel seems too dangerous to remove, this portion can be left attached and its lining destroyed by careful diathermy.

In the case of an enterogenous cyst, enucleation must not be attempted. If a comparatively short segment of the intestine is involved, resection of the cyst with the

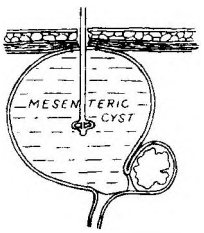


FIG. 1073.—Method of treating a large mesenteric cyst by marsupialisation.

adherent portion of the intestine, followed by intestinal anastomosis, is the correct course. Should a very large segment of small intestine be implicated, an anastomosis should be made between the apex of the coil of small intestine and the cyst wall which, in this instance, holds sutures well.

The older treatment of marsupialisation of a mesenteric cyst has little to recommend it, for a fistula or recurrence sometimes results. Occasionally, however, on account of its simplicity, it is advisable in a poor-risk subject (fig. 1073).

Omental cyst occurs nearly as frequently as a mesenteric cyst. Preoperative differentiation is possible because a lateral radiograph shows the cyst in front of the intestines. Treatment is omentectomy.

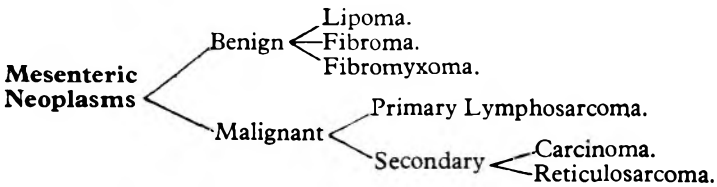
Cyst of the mesocolon is uncommon, and it is differentiated from a mesenteric cyst only at operation. The treatment is similar.

Cysts arising from a urogenital (Wolffian or Müllerian) remnant are essentially retroperitoneal, but they are included in the classification because it is not impossible for such a cyst to project forward into the mesentery.

The following, while not being mesenteric cysts in the meaning of the term, give rise to the same physical signs, and, in practice they *are* mesenteric cysts: *Serosanguineous cyst* is probably traumatic in origin, but a history of an accident is seldom obtained. *Tuberculous abscess of the mesentery. Hydatid cyst of the mesentery.*

NEOPLASMS OF THE MESENTERY

Tumours situated in the mesentery give rise to physical signs similar to those of a mesenteric cyst, the sole exception being that they sometimes feel solid.



A benign tumour of the mesentery is excised in the same way as an enterogenous mesenteric cyst, i.e. with resection of the adjacent intestine. When possible, a malignant tumour of the mesentery is subjected to the same treatment. In inoperable cases radiotherapy can be employed if the biopsy specimen reveals that the growth is probably radio-sensitive.

THE RETROPERITONEAL SPACE

Pus or blood in the retroperitoneal space tends to track to the corresponding iliac fossa. If a retroperitoneal abscess develops, it should be evacuated by an incision through the abdominal wall, meticulously avoiding opening the peritoneum. Should the retroperitoneal collection be found at laparotomy, it must be drained by a counter-incision in the flank. Retroperitoneal hæmatoma may be caused by fractured spine (p. 478), a leaking abdominal aneurysm (p. 150), or acute pancreatitis (p. 941).

Retroperitoneal Cyst.—A cyst developing in the retroperitoneal space often attains very large dimensions, and has at first to be distinguished from a hydronephrosis. Even after the latter condition has been eliminated by pyelography, a retroperitoneal cyst can seldom be diagnosed with certainty from a retroperitoneal tumour, until displayed at operation. The cyst may be unilocular or multilocular. Many of these cysts are believed to be derived from a remnant of the Wolffian duct, in which

Kasper Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad, U.S.S.R.).
Johannes Peter Müller, 1801–1858. Professor of Anatomy and Physiology, Berlin.

case they are filled with clear fluid. Others are teratomatous, and are filled with sebaceous material.

Excision of these and other retroperitoneal swellings is best performed through a transperitoneal incision (see below).

Idiopathic Retroperitoneal Fibrosis.—See Chapter 56.

PRIMARY RETROPERITONEAL NEOPLASMS

Although *neuroblastomas* and *ganglioneuromas* of the adrenal gland are retroperitoneal tumours, they have now been segregated as clinical entities, and have been dealt with on p. 657.

Retroperitoneal lipoma, in the first instance, is usually mistaken for a hydronephrosis, a diagnosis which is ruled out by pyelography. Women are more often affected. These swellings sometimes reach an immense size. We have removed such a tumour weighing 2·5 kg, and much larger specimens have been recorded. A retroperitoneal lipoma sometimes undergoes myxomatous degeneration, a complication which does not occur in a lipoma in any other part of the body. Moreover, a retroperitoneal lipoma sometimes becomes malignant (liposarcoma) (fig. 1074).

Retroperitoneal sarcoma presents signs similar to a retroperitoneal lipoma. The patient may seek advice on account of a swelling or because of indefinite abdominal pain. On other occasions the tumour, by pressure on the colon, causes symptoms of subacute intestinal obstruction. On examination a smooth fixed mass, which is not tender, is palpated. The most probable original diagnosis is that of a neoplasm of the kidney. This is ruled out by pyelography. The ureter, however, is liable to become displaced by the tumour. Exploratory laparotomy should be performed, and when possible the tumour is removed. Often it is found widely disseminated in the retroperitoneal space, rendering complete removal impossible, in which case a portion is excised for microscopy. Even when excised at a comparatively early stage, recurrence always takes place, and these tumours must be looked upon as being necessarily fatal. Deep X-ray therapy sometimes keeps recurrences in abeyance for a time.

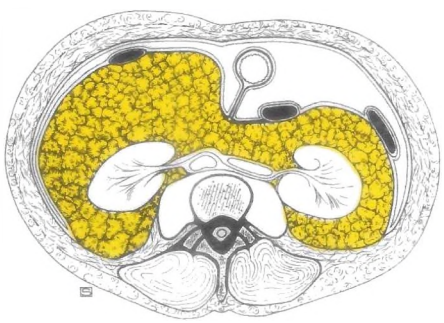


FIG. 1074.—Rapidly growing retroperitoneal liposarcoma.

Removal of a Retroperitoneal Cyst or Neoplasm.—After the anterior abdominal wall has been opened and the diagnosis of a retroperitoneal tumour has been confirmed, the incision is extended as necessary. The small intestine is packed away in the upper abdomen, and the cæcum and the sigmoid are relegated to their respective fossæ. The posterior peritoneum is then incised throughout its length over the area to be exposed, the incision parallel to the left border of the aorta. The peritoneum is dissected from the tumour which is removed as completely as possible, the intestines being kept out of the way with packs or exteriorised temporarily, depending on which manœuvre is the more useful.

CHAPTER 49

THE INTESTINES

Abdominal Pain arising from the Alimentary Canal is of two types:

1. *Visceral Pain.*—The alimentary tract is primarily a midline structure with a bilateral nerve supply. Although rotation about the midline occurs during development, nevertheless true visceral pain is referred to the midline as illustrated in fig. 1075. It is dull and poorly localised. The presence of a bilateral nerve supply may account for contralateral pain from some diseased structures as with left sided pain in cholecystitis.

2. *Peritoneal Pain* however is of the somatic type—and is much more precise, more severe and localised to site of origin. These components account for the changes in character and site of pain which occurs in appendicitis (p. 1060).

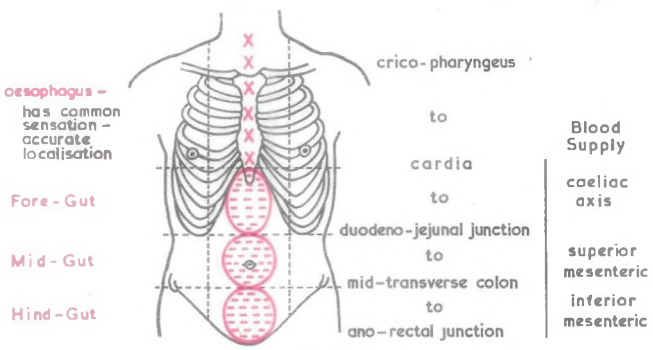


FIG. 1075.—Sites of origin of visceral pain.

Surgical Anatomy.—It is of great practical importance to be able:

1. To distinguish various portions of the intestinal canal at sight.
2. To know in which part of the abdomen the upper coils, as opposed to the lower coils, of the small intestine lie in relationship to the anterior abdominal wall.
3. To be able to decide which is the proximal and which the distal end of any coil under consideration.
4. To distinguish irrefutably large from small intestine.

For practical purposes these problems are settled as follows:

(*a*) The mesentery of the jejunum has only two series of arches of blood-vessels, whereas the lower ileum has several series of arches.

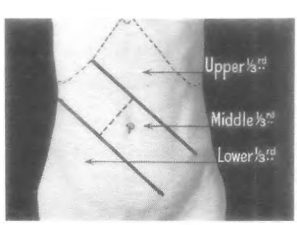


FIG. 1076.—Monks's method of localising the small intestine upon the surface.

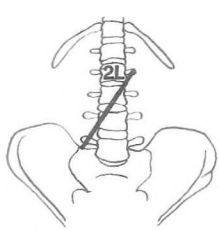


FIG. 1077.—The attachment of the mesentery.

(*b*) Monks's method of intestinal localisation roughly indicates the disposition of the upper, middle, and lower thirds of the small intestine (fig. 1076).

(*c*) The mesentery, after being made taut, is examined. As the mesenteric attachment runs from left to right (fig. 1077), if palpation reveals that the mesentery is not twisted, then the upper end of the bowel in the wound is the proximal end. Such a test is useful, but not as easy to perform in the living as in the dissecting-room subject.

(*d*) As the 'small' intestine is sometimes found enormously distended and the 'large' intestine entirely collapsed, size is no criterion. The large intestine is characterised by its tæniæ coli and appendices epiploicæ.

CONGENITAL MALFORMATIONS OF THE INTESTINES

Congenital Atresia of the Duodenum (p. 1038).
Congenital Atresia of the Small Intestine (p. 1039).
Volvulus Neonatorum (p. 1040).
Vascular Anomalies (Angiodysplasia) (p. 995).

Failure of Descent of the Cæcum.—The cæcum remains under the right lobe of the liver—a normal situation of the structure in the mangabey monkey. This anomaly is associated with malposition of the vermiform appendix.

MEGACOLON

There are two varieties of this relatively uncommon condition (1) *primary* or true megacolon (*syn.* Hirschsprung's disease, congenital aganglionic megacolon) and (2) *secondary* or acquired megacolon (fig. 1078).

Primary Megacolon.—Hirschsprung's disease:

Pathology.—This disease is characterised by enormous dilatation and hypertrophy of the pelvic colon, sometimes extending into the descending colon but rarely involving the more proximal parts of the large intestine. The pelvic mesocolon is elongated and thickened and its blood-vessels are large and prominent. All coats of the dilated intestine show gross pathological changes. The mucosa is chronically inflamed and frequently ulcerated. There is a terminal constricted, non-hypertrophied segment of bowel usually involving the anal canal, the rectum, and a variable part of the large intestine. In nine out of ten cases the upper limit of the contracted segment is the pelvi-rectal junction; occasionally the deficiency extends to a higher level (fig. 1079). It is in this contracted segment that physiological obstruction lies[1], and the dilatation hypertrophy of the colon above is due to absence of peristalsis in the spastic segment.

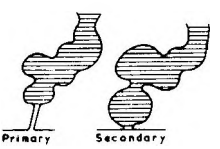


FIG. 1078.—Barium enema findings in primary and secondary megacolon.

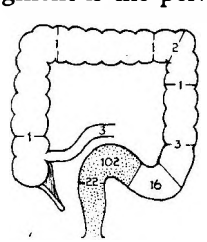


FIG. 1079.—The extent of the aganglionic segment in 152 cases of Hirschsprung's disease treated by operation at the Hospital for Sick Children, Great Ormond Street. (*After G. G. Wyllie.*)

On histological examination the cause of the immotility of the spastic segment is evident; there is a complete absence of parasympathetic ganglion cells, and this ganglion deficiency

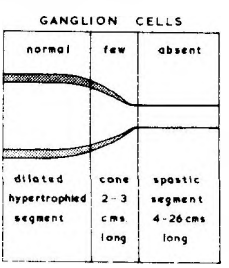


FIG. 1080.—Showing the essential pathological histology in congenital Hirschsprung's disease. (*After M. Bodian, London.*)

[1] In 1898 Treves attributed Hirschsprung's disease to congenital spasm of the distal segment, thus preceding the re-discovery of this concept by fifty years.

George H. Monks, 1853–1933. Surgeon-in-Chief, City Hospital, Boston, U.S.A.
Harald Hirschsprung, 1830–1916. Physician, Queen Louise Hospital for Children, Copenhagen.
Sir Frederick Treves, 1853–1923. Surgeon, The London Hospital.

extends for a distance of 1 to 5 cm into a transitional zone or cone (fig. 1080) between the terminal spastic segment and the hypertrophied portion (Bodian). Above the transitional zone, parasympathetic ganglion cells are present as in normal intestine.

Clinical Features.—Hirschsprung's disease shows a familial tendency. It is much more common in males than females. In 90 per cent. of cases symptoms appear within three days following birth.

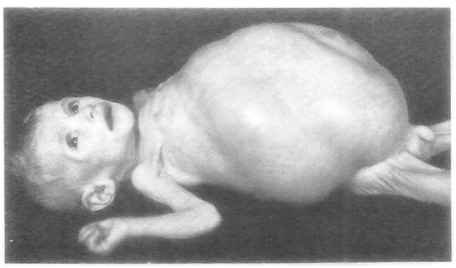


FIG. 1081.—Hirschsprung's disease showing enormous colonic dilatation and a wave of visible peristalsis. (*The late Sir Denis Browne, F.R.C.S., London.*)

Constipation.[1]—The infant fails to pass meconium during the first two or three days of life, and then only after the insertion of a little finger or a tube into the rectum. Subsequently motions are tooth-paste-like, and inadequate in amount; straining is in evidence during their passage.

Abdominal distension is usually unmistakable by the third day. In a proportion of cases the abdominal distension progresses, and sometimes it is evident that the colon is obstructed (fig. 1081); in others it is impossible clinically to differentiate large from small intestinal obstruction. Loud borborygmi and visible peristalsis are much in evidence.

Rectal Examination.—The anus is free from fissures and excoriation and there is no perianal soiling. The rectum is empty and *grips the examining finger*.

Complete intestinal obstruction occurs quite frequently within a few days of birth and may be fatal. As a rule, the attacks are recurring and relief is given by a small enema, by passing a greased examining finger or by the spontaneous passage of a large stool sometimes followed by diarrhœa. Owing to the enormous abdominal distension, chest infection may supervene. In any case, if the child survives, malnutrition and stunted growth are obvious features together with enormous abdominal distension.

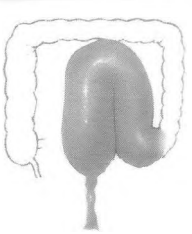


FIG. 1082.—Radiological appearances in Hirschsprung's disease; coning, as well as dilatation, is diagnostic. (*After B. C. H. Ward, London.*)

Radiography (p. 1021). *Barium Enema.*—When the clinical findings are atypical a barium enema is often helpful in confirming the diagnosis. The objective is to demonstrate the contracted aganglionic segment, if such be present, and to this end preliminary wash-outs are withheld. For reasons explained below, the barium should be admixed with normal saline solution, not tap-water. By using only a little dilute barium emulsion—just sufficient to run over the fæcal masses—a good outline of the bowel can be obtained (fig. 1082).

Biopsy of the ano-rectal wall is employed in order to prove or disprove the presence of ganglia. A piece of rectal wall 1 cm² is removed and searched for ganglia in the sub-mucosa (Meissner's plexus) and intermyenteric layer (Auerbachs plexus).

[1] *Diarrhœa.* It cannot be emphasised too strongly that these infants sometimes present with diarrhœa and abdominal distension, and unless a rectal tube is passed, the patient may rapidly succumb to what is mistakenly thought to be simple gastroenteritis.

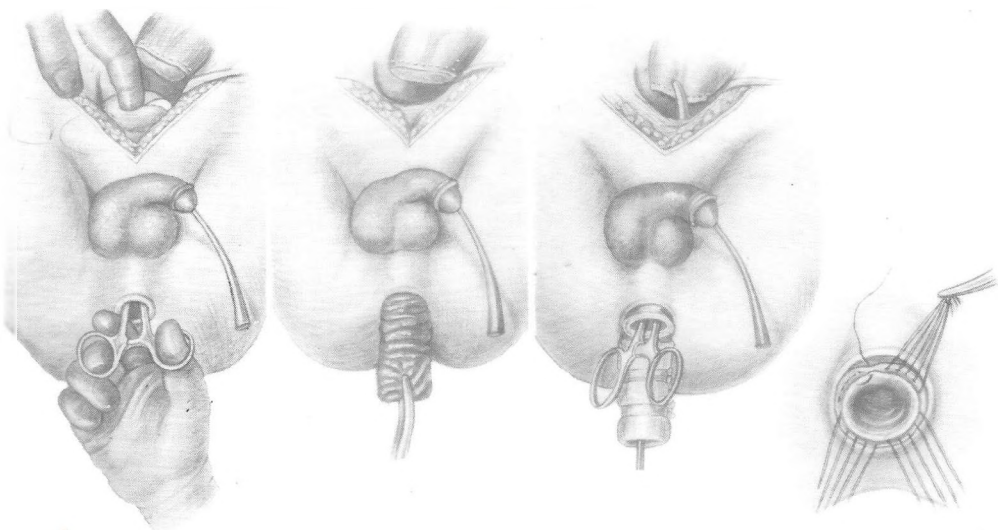
Martin Bodian, 1910–1963. Morbid Anatomist, Hospital for Sick Children, Great Ormond Street, London.
Georg Meissner, 1829–1905. Professor of Physiology, Göttingen, Germany.
Leopold Auerbach, 1828–1897. Professor of Neuropathology, Breslau, Germany (now Wroclaw, Poland).

Laparotomy is required when the patient has acute-on-chronic intestinal obstruction, and other causes cannot be eliminated. If the case proves to be one of Hirschsprung's disease it is advisable to establish a temporary transverse colostomy or, in the rare event of the whole colon being narrowed, an ileostomy.

Pre-operative Treatment.—Colonic lavage in Hirshsprung's disease is dangerous, because the use of tap-water in the enemas in this condition may cause water intoxication. The megacolon absorbs water much more rapidly than does a normal colon, and the disturbance of electrolytic balance, especially of sodium, may prove fatal. Provided the general condition is good, normal saline solution can be used with safety. In patients with impaired cardiac or renal reserve, 7 per cent. gelatine solution should be substituted. Unless measurements show that distension is increasing, four weeks' pre-operative preparation is desirable (Svenson). During the last five pre-operative days, Sulfasuxidine is given for bowel sterilisation.

Treatment.—The only curative treatment of Hirschsprung's disease is excision of the entire aganglionic segment. No reparative operation should be done till the child is 8·2 kg in weight and thriving (Brown).

Operation.—The patient is placed in a semi-lithotomy position, to give good access to the abdomen and the peri-anal region. Through a suitable incision the rectum, the contracted part of the adjoining colon, and the commencement of the hypertrophied portion are freed from their attachments as far downwards as possible towards the anal canal. In contrast to excision for carcinoma of the rectum, the dissection must be kept immediately outside the fascia propria. In this way the seminal vesicles and the autonomic nerve trunks to the bladder are protected. Should doubt exist as to whether the aganglionic segment has been encompassed, frozen section biopsy will provide the answer. The intestine is then transected proximally through normal colon, and the distal end is closed with a purse-string suture. The mobilised aganglionic segment is then everted through the anus (fig. 1083(*a*) and (*b*)). The mucosa thus exposed is painted with antiseptic solution, and dried. The anterior half of the junction of the inverted rectum with the anal canal is opened transversely, and the proximal colon is pulled through the opening (fig. 1083(*c*)). End-to-end anastomosis



(*a*) (*b*) (*c*) (*d*)

FIG. 1083.—(*a*) Long hæmostat introduced through the anus so that the top of the closed bowel can be grasped. (*b*) Mobilised segment is everted through the anus. (*c*) Long hæmostat introduced through the incision in the rectal wall to grasp the proximal end of the divided colon. (*d*) Anastomosis of the colon to the anal canal. (*After O. Svenson.*)

Orvar Svenson, Contemporary. Professor of Surgery, Northwestern University, Chicago, Illinois, U.S.A.
James Johnston Mason Brown, 1908–1964. Surgeon-in-Charge, Royal Hospital for Sick Children, Edinburgh.

between the colon and the anal canal is carried out (fig. 1083(*d*)) as the everted aganglionic segment is excised. The union having been completed, it is reduced into the anal canal. Gloves having been changed, the abdominal incision is repaired. By two teams working simultaneously, one abdominally and one perineally, the operation can be performed expeditiously. Cases of Hirschsprung's disease involving the entire colon have been treated successfully by total colectomy and ileoproctostomy.

Modified Duhamel Operation.—This operation is designed to preserve rectal sensation and storage capacity. Preliminary colostomy and bowel preparation is carried out as above. The aganglionic segment is removed down to the level of the peritoneal reflection over the rectum. At this point the rectum is divided, turned in, and closed over. The sacral hollow is then opened up and the normal colon brought down to the posterior aspect of the rectal stump. The anus is now widely stretched and a transverse incision is made in the posterior wall just above the sphincter. Through this opening the colon lying behind the rectum is seized and brought down to present at the anus. The colon is then fixed to the rectum by a few catgut stitches to make a somewhat loose colo-rectal anastomosis. A special crushing clamp is now introduced to crush the spur between the rectum and the colon. It is left in position till the spur separates, when the clamp becomes loose and can be removed. The colostomy is closed a few weeks later.

ACQUIRED OR SECONDARY MEGACOLON

Dilatation and hypertrophy of an otherwise normal large bowel extends to the anal canal. The obstruction is due to fæcal impaction. Characteristically, there is a fissured anus, a spastic sphincter and much perianal soiling. As a rule, faulty bowel care and training are the sources of the trouble, and usually they can be traced to infancy; the onset, however, is never from birth. Sometimes the condition is encountered in the insane and the old.

Rectal Examination.—This is usually painful and the finger encounters a scybalous mass just inside the anus, which is contrary to the findings in Hirschsprung's disease.

FIG. 1084.—Radiological appearances of acquired megacolon.

Radiography.—In all cases of acquired megacolon the dilatation as shown by a barium enema ends at the anal canal (fig. 1084).

Biopsy of the Rectal Wall.—When acquired megacolon cannot be differentiated from Hirschsprung's disease by clinical and radiological means, biopsy is required. The presence of ganglion cells in the submucosa is the signal for conservative treatment.

Conservative Treatment.—Should an anal stricture or a fissure-in-ano be present, these lesions must be treated appropriately. The essential feature is bowel training so that a regular habit is established. It is wise to start with regular enemas and Senokot until the bowel is clean. The family environment must be thoroughly investigated. Upset of bowel habit in a child is a not uncommon result of domestic upheaval or emotional deprivation.

Bernard Duhamel, Contemporary. Chief-Surgeon, Hôpital Saint-Denis, Saint-Denis, France.

Redundant Colon.—Some constipated patients (usually female) are found on investigation by barium enema X-ray to have an elongated sigmoid colon (dolichocolon). Volvulus formation is a theoretical risk, but the wise surgeon avoids removing the redundant bowel for these patients may be neurotic, and will plague him after the operation with the same or other symptoms.

Cathartic Colon.—In some patients the dilatation and immobility of the colon is due to chronic poisoning (' intoxication ') of the intestinal wall by large doses of purgatives taken over many years. The cascara and senna derivatives are the worst agents. In a few cases the generalised picture of dilated bowel is complicated by strictures (especially in the right colon). Melanosis coli is often observed on sigmoidoscopy. Such cases are grouped under the title of ' cathartic colon '. Conservative management is often successful in these patients.

Vascular Anomalies (**Angiodysplasia**), being capillary or cavernous hæmangiomas are a cause of hæmorrhage from the colon at any age, presenting with hæmorrhagic shock. In the middle aged or elderly patient the lesion needs to be distinguished from the other causes of sudden massive hæmorrhage viz., diverticulitis, ulcerative colitis, ischæmic colitis. In 50% of cases the bleeding arises in the ileocæcal region or right colon. Whereas a sigmoidoscopy will help to distinguish between an anorectal hæmangioma (bluish subepithelial venous swellings) and colitis (acute mucosal inflammatory changes), a barium enema will only detect diverticular disease and ischæmic colitis, and a colonoscopy is disappointing. Selective superior and inferior mesenteric angiography alone suffices to show the site and extent of the lesion by a ' blush '.

The treatment may well involve an emergency total colectomy with ileorectal anastomosis if the site cannot be identified. This drastic step is preferable to a situation of continued massive bleeding after a subtotal left or right sided colectomy.

TRAUMATIC RUPTURE OF THE INTESTINE

The intestine can be ruptured with or without an external wound. The most frequent cause of the latter is a blow on the abdomen which crushes the bowel against the promontory of the sacrum (see also seat belt syndrome in traffic accidents p. 983). Rupture is particularly liable to occur in the presence of an irreducible inguinal hernia (John). Rupture typically occurs where a fixed part of the alimentary tract joins a mobile one, such as the duodenojejunal flexure, in which case the damage may be retroperitoneal and easily overlooked; this type of lesion is usually due to runover accidents.

In small perforations the mucosa prolapses and tends partially to seal the rent (fig. 1085); consequently the early signs are misleading. In general the signs simulate closely those of a perforated peptic ulcer.

· *Laceration of the mesentery* (p. 983) is a frequent operative finding in the type of injury under consideration.

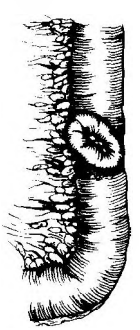


FIG. 1085.—Traumatic rupture of the ileum. Note the prolapse of the mucous membrane.

Traumatic rupture of the large intestine is much less frequent. Compressed-air rupture of the colon is sometimes the result of a damnable form of practical joke, whereby a hose, carrying air under considerable pressure, is turned on near the victim's anus.

Blast injuries of the abdomen sustained during air-raids resulted in a number of cases of traumatic rupture of the intestine. The pelvic colon was found to be injured more frequently than other segments of the intestine. Rupture of the upper reaches of the rectum is not unknown during sigmoidoscopy. In ulcerative lesions the air insufflation has been sufficient to perforate the intestinal wall.

Howell Thomas John, 1921–1979. Surgeon, Royal United Hospital, Bath, England.

Treatment.—In all cases of suspected rupture of the intestine immediate laparotomy must be performed. In many instances, simple closure of the perforation is all that is required. In others, e.g. where the mesentery is lacerated, resection may be necessary (fig. 1086). In the case of the large intestine, exteriorisation is the procedure of choice; if this is not feasible, the perforation is closed and proximal colostomy is performed.

FIG. 1086.—Laceration of the mesentery resulting in gangrene of the associated portion of gut.

THE BLIND LOOP SYNDROME

It has been shown in dogs that if a blind loop of the small intestine is made (fig. 1087*A*) defects of absorption will appear. If this occurs in the upper intestine the defect is chiefly of fat absorption; if in the lower intestine there is vitamin B_{12} deficiency. This has been found to occur in humans and is referred to as the blind loop syndrome, or the ' stenosis-anastomosis loop ' syndrome (Witts).

Essentially, the stasis produces an abnormal bacterial flora, which prevent proper breakdown of the food (especially fat) and mop up the vitamins that are present. Sometimes the only manifestation is anæmia, due to Vitamin B_{12} deficiency, but if steatorrhœa appears, other serious malabsorption features follow. In general, high loops produce steatorrhœa, etc., whereas low loops tend to produce anæmia.

Temporary improvement will follow the use of antibiotics to destroy the bacteria causing the trouble, but the main treatment is surgical extirpation of the cause of the stasis.

ALIMENTARY DIVERTICULA[1]

Diverticula occur from the stomach to the recto-sigmoid junction. There are two varieties:—

(*a*) *Congenital.*—All three coats of the bowel are present in the wall of the diverticulum, e.g. Meckel's.

(*b*) *Acquired.*—The wall of the diverticulum lacks a proper muscular coat. Most alimentary diverticula are thought to be acquired (Edwards).

DIVERTICULA OF THE SMALL INTESTINE

The belief that a diverticulum of the small intestine originates as a mucosal herniation through a point of entrance of blood-vessels is based on the fact that most of these diverticula arise from the mesenteric side of the bowel (fig. 1087).

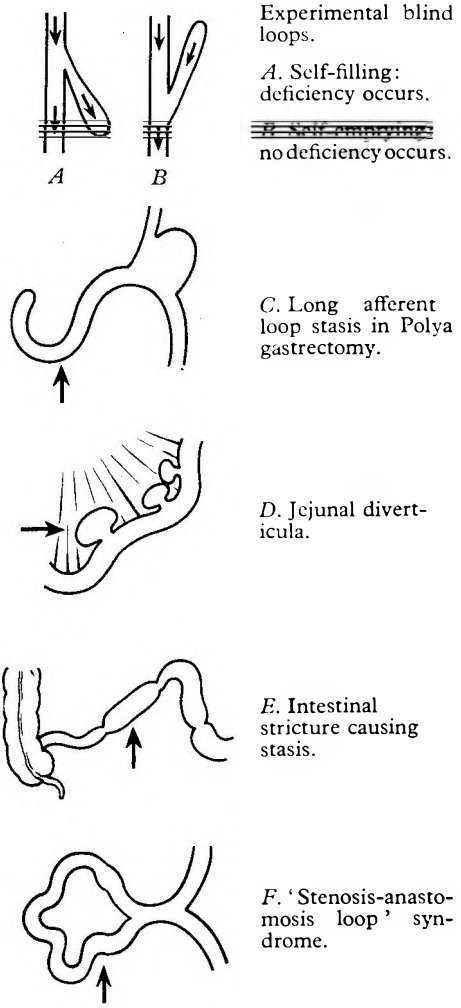
Duodenal Diverticulum.—There are two types.

1. *Primary.*—Mostly in older patients on the inner wall of the second and third parts, they are found incidentally on barium meal and usually do not cause symptoms.
2. *Secondary*, of the duodenal cap resulting from long standing duodenal ulceration.

[1] Literally, a diverticulum means a wayside house of ill-fame, and these wayside houses certainly live up to their evil reputation.

Leslie John Witts, Contemporary. Emeritus Nuffield Professor of Clinical Medicine, Oxford, England.
Johann Friedrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle, Germany.
Harold Clifford Edwards, Contemporary. Consulting Surgeon, King's College Hospital, London.

Jejunal diverticula vary in size, are sometimes single (fig. 1087*A*). More often several are present (figs. 1087*D* and 1090). Clinically they may (*a*) be symptomless (*b*) give rise to abdominal pain, flatulence and borborygmi or (*c*) produce a malabsorption syndrome. This latter consists of anæmia steatorrhœa, hypoproteinæmia and avitaminosis. They are, in fact, examples of the blind loop syndrome (p. 996). If the length of affected bowel is not too extensive, resection and end-to-end anastomosis give excellent results.



Experimental blind loops.

A. Self-filling: deficiency occurs.

B. Self-emptying: no deficiency occurs.

C. Long afferent loop stasis in Polya gastrectomy.

D. Jejunal diverticula.

E. Intestinal stricture causing stasis.

F. 'Stenosis-anastomosis loop' syndrome.

FIG. 1087.—Common types of blind loops.

Meckel's diverticulum is present in 2 per cent. of the human race; it is situated upon the antimesenteric border of the small intestine, commonly 60 cm from the ileo-cæcal valve, and it is usually 5 cm long. Many variations occur. 2 per cent—2 feet—2 inches.

In nearly 90 per cent. of cases the diverticulum arises from the antimesenteric border of the ileum, and, being congenital, it possesses all three coats of the intestinal wall. It will be noted in figs. 1091 and 1092 that it has its own blood supply (like the appendix) and this makes it vulnerable to infection and obstruction. In 20 per cent. of cases the mucosa contains heterotopic epithelium, viz. gastric, colonic, or sometimes pancreatic tissue. When present, heterotopic tissue lines the greater part of the proximal end of the pouch, and not infrequently extends for a short distance into the nearby ileum. Although Meckel's diverticulum occurs with equal frequency in both sexes, symptomatic cases, due almost entirely to the epithelium contained in the diverticulum, are predominant in males. In order of frequency, these symptoms are as follows:

FIG. 1088.—Primary diverticula of second and third parts of duodenum.

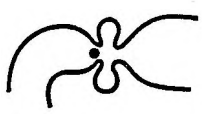


FIG. 1089.—Secondary diverticula of duodenal cap.

1. **Severe hæmorrhage per rectum,** due to peptic ulceration. The blood passed is maroon in colour. Although the patient frequently vomits, the vomitus contains no blood. Seldom is the hæmorrhage preceded by pain; sometimes bleeding precedes perforation. When operation is required for serious progressive hæmorrhage per rectum and no lesion in the stomach or duodenum is found, the next step should be the examination of the terminal 150 cm of ileum.

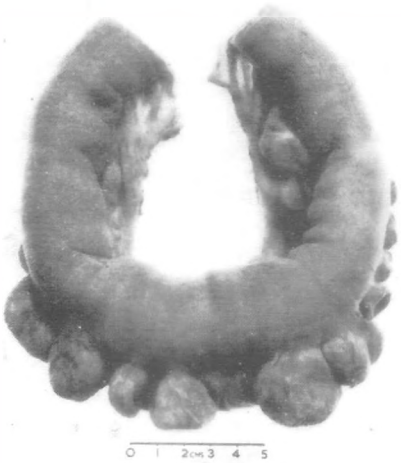


FIG. 1090.—Jejunal diverticula.

2. **Intussusception.**—In the majority of cases the apex of the intussusception is swollen, inflamed, heterotopic epithelium at the mouth of the diverticulum—not inversion of the diverticulum, as is commonly stated. Intussusception due to Meckel's diverticulum is discussed on p. 1041.

FIG. 1091.—Meckel's diverticulum.

3. **Meckelian diverticulitis** with or without perforation is usually due to lodgement of coarse food residue or a sharp foreign body. The symptoms of Meckelian diverticulitis without perforation are those of acute appendicitis, and unless the appendix has been removed the diagnosis is impossible before operation. When a diverticulum perforates, so rapid is the onset of peritonitis that the symptoms simulate those of a perforated duodenal ulcer. Whether or not the diverticulum has perforated, urgent operation is required. In non-perforated cases an inflamed diverticulum should be sought as soon as it has been ascertained that the vermiform appendix (and, in the case of a female, the Fallopian tubes) is not at fault.

4. **Chronic Peptic Ulceration.**—Because the diverticulum is part of the midgut, the pain, though related to meals, is felt around the umbilicus.

5. **Intestinal Obstruction.**—The presence of a band between the apex of the diverticulum and the umbilicus may cause obstruction either by the band itself or to a volvulus around it.

Radiography.—In cases of Meckel's diverticulum giving rise to symptoms, failure to visualise the diverticulum by radiography after a barium meal, which is very common, is of no significance, because so often the entrance of the diverticulum is blocked by œdema.

'Silent' Meckel's Diverticulum.—A Meckel's diverticulum usually remains symptomless throughout life, and is found only at necropsy. When a silent Meckel's diverticulum is encountered in the course of an abdominal operation, and can be excised without appreciable additional risk, this should be done in order to exclude the possibility of subsequent complications.

Exceptionally a Meckel's diverticulum is found in an inguinal or femoral hernial sac, when it is known as a Littre's hernia (p. 1134).

Meckelian Diverticulectomy.—A Meckel's diverticulum should not be amputated at its base and invaginated in the same way as a vermiform appendix, because of the risk of a stricture. Moreover, it does not remove heterotopic epithelium completely, if such be present. The steps of diverticulectomy are displayed in

Gabriele Falloppio, 1523–1562. (*See footnote, p. 156.*)
Alexis Littre, 1658–1726. Surgeon and Anatomist, Paris.

fig. 1092. Should there be considerable induration of the base of the diverticulum, and particularly when such induration extends into the neighbouring ileum, it is advisable to resect a short segment of the ileum containing the diverticulum, and to restore the continuity of the bowel by anastomosis.

FIG. 1092.—Steps in the performance of Meckelian diverticulectomy

DIVERTICULAR DISEASE OF THE COLON

Diverticula of the colon are acquired herniations of colonic mucosa, protruding through the circular muscle at the points where the blood vessels penetrate the colonic wall (fig. 1093). Thus they tend to occur in rows in between the strips of longitudinal muscle (tænia coli) (fig. 1094). The condition may be localised to one part of the colon, usually the sigmoid, but sometimes the entire large bowel is involved from the cæcum to the rectosigmoid junction. The rectum with its complete muscle layers is not affected. In 90% of cases the sigmoid colon is involved and becomes almost always the site of inflammation, i.e. diverticulitis. Some 5% of patients have associated gallstones and hiatus hernia (Saint's triad, p. 918).

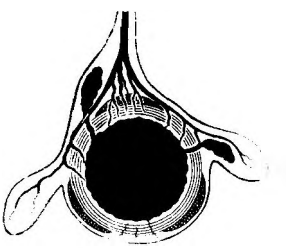


FIG. 1093.—The usual sites of diverticulation of the colon. (After Hamilton Drummond.)

Diverticular disease is rare in Africans and Asiatics who eat a diet which contains natural fibre. In Western countries, where the roughage has been removed from flour and sugar, diverticula are found in 5% of barium enemas over the age of 40, and the incidence increases with age.

Diverticulosis, the primary stage of the disease, is caused by muscular inco-ordination and hypertrophy resulting in increased segmentation and intraluminal pressure (fig. 1095 *a*, *b*, and *c*). This may lead to diverticulum formation (fig. 1095*d*). Segmentation plays an important part in colonic physiology. Emotion and drugs like morphine and prostigmine[1] increase segmentation and intra-colonic pressure (see treatment).

Diverticulitis is the secondary state and is due to inflammation of one or more diverticula, usually with some pericolitis. Episodes of diverticulitis

[1] Muscle relaxants of the curare type usually require the use of prostigmine at the end of an operation. This use of prostigmine may prejudice the safety of a colonic anastomosis.

may be followed by years free of symptoms, but the condition is essentially progressive—the longer the duration, the worse are the symptoms and

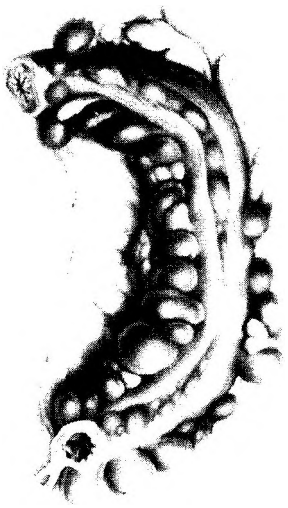


FIG. 1094.—Pelvic colon with multiple diverticula.

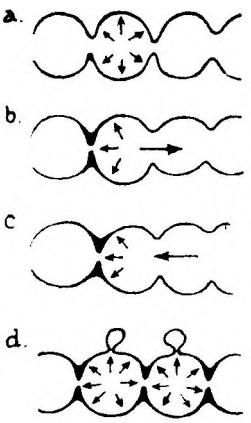


FIG. 1095.—Segmentation of the colon.

the greater the risk of complications. Diverticulitis is not a precancerous condition, but cancer may coexist.

The Complications are:

1. Recurrent periodic inflammation and pain.
2. Perforation leading to general peritonitis or local (pericolic) abscess formation.
3. Intestinal obstruction, due to progressive fibrosis leading to stenosis.
4. Hæmorrhage. diverticulitis may present with *profuse* colonic hæmorrhage in about 1% of cases, often requiring blood transfusion.
5. Fistula formation (vesico-colic, vagino-colic, entero-colic, colo-cutaneous) occurs in about 5% of cases.

Clinical Features.—Diverticulosis may be asymptomatic, but the disordered colonic function may cause symptoms of distension, flatulence and a sensation of heaviness in the lower abdomen. Excessive colonic segmentation can cause severe colic pain in the left iliac fossa. This pain waxes and wanes too fast to be compatible with the onset and resolution of inflammation. Indeed, the sigmoid colon may only have thickened muscle (Morson). This condition is called *Painful Diverticular Disease* and is, in fact, due to spastic obstruction of the colon (Painter).

Diverticulitis.—More persistent lower abdominal pain, usually in the left iliac fossa, or evidence of peritonitis, should always bring the diagnosis to mind in patients of either sex over the age of 40. Fever, malaise, and a leucocytosis help to differentiate diverticulitis from painful diverticular disease. The patient may pass loose stools or may be constipated. Abdom-

Basil Clifford Morson, Contemporary. Pathologist, St Mark's Hospital, London.
Neil Stamford Painter, Contemporary. Surgeon, Manor House Hospital, London.

inal distension may be relieved by flatus. The lower abdomen is tender especially on the left, occasionally in the right, iliac fossa. The sigmoid colon may be palpable, tender and thickened. Rectal examination may reveal a tender mass. The condition has been likened to left-sided appendicitis. Any urinary symptoms may herald the formation of a vesico-colic fistula, which leads to pneumaturia (flatus in the urine), and even fæces in the urine.

Diagnosis

Radiology.—Diverticulosis must never be blamed for symptoms unless all other diseases of the gastrointestinal tract have been excluded by barium meal, barium enema and by sigmoidoscopy. The diagnosis of acute diverticulitis is made on clinical grounds and is confirmed by barium enema and sigmoidoscopy *only when any acute inflammation has subsided* as any enema given in an acute attack may cause perforation and peritonitis. Diverticula may be revealed by a barium meal followed through the gut, but a barium enema must also be done to exclude a carcinoma and to assess the extent of the disease. Barium enema may show diverticula and the typical narrowed sigmoid but sometimes no diverticula are seen but only the 'saw tooth' appearance of the 'pre-diverticular state' (fig. 1096). Such spastic strictures are sometimes relaxed by Probanthine and this may help to differentiate them from a neoplasm.

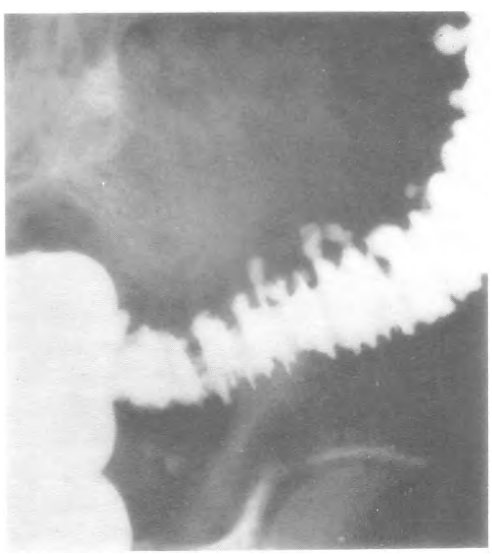


FIG. 1096.—Barium enema showing diverticular disease—'saw' teeth' and diverticula.

Sigmoidoscopy.—The mucosa may be normal. In acute attacks, sigmoidoscopy may be painful and the mucosa inflamed. The bowel may be rigid, narrow and acutely angled at about 15 cm. so that the instrument cannot be passed further. The necks of diverticula are seldom seen. Sigmoidoscopy and colonoscopy excludes a neoplasm but diverticula are best diagnosed by barium enema.

Management

Diverticulosis should be treated with a high residue diet containing roughage in the form of wholemeal bread and flour, fruit and vegetables. Bulk formers such as bran, Celevac, Isogel, may be given until the stools are soft. Painful diverticular disease may require bed rest and antispasmodic drugs. Habitual liquid paraffin and purgative-taking should be avoided.

Acute diverticulitis is treated by bed rest, warmth to the abdomen, antispasmodic drugs and a 7–10 day course of broad-spectrum antibiotics (e.g. tetracycline) and/or non-absorbable sulphonamides (e.g. phthalylsulphathiazole). Morphine and enemas are contraindicated. Probanthine rests the bowel but in large doses may cause ileus and retention of urine. After the acute attack has subsided the diagnosis must be confirmed by radiology and a carcinoma must be excluded.

Operative Procedures for Diverticular Disease.—About 10% of the patients require operation, either for recurrent attacks which make life a misery or for the complications (p. 1000):

1. *The ideal operation*, done as an interval procedure after careful preparation of the gut (p. 1022), is a *One Stage Resection.* This involves removal of the affected segment—10 to 20 cm long, and restoration of continuity by end-to-end anastomosis.

At operation the sigmoid loop is often found adherent in the pouch of Douglas. Patient yet persistent dissection, taking care to preserve the rectum in the retroperitoneal area, is rewarded by exposure of normal rectum and greater mobility.

2. *BUT, if there is obstruction, inflammatory œdema and adhesions or the bowel is loaded with fæces*, a preliminary transverse colostomy must be done as a first stage; the second stage of resection is performed after 3 weeks or when the inflammation has subsided. The colostomy is closed after a further 2 weeks.

3. *In acute perforation*, peritonitis which soon becomes general is usually purulent with a mortality of about 15%. Fæcal peritonitis carries a 50% mortality. Pneumoperitoneum is present, but in this acute emergency often a diagnosis of peritonitis calling for urgent laparotomy is the only diagnosis which can be made. There is a choice of procedures:

(*a*) *Proximal colostomy* with suture of perforation.

(*b*) *Exteriorisation* of the affected bowel which is then opened as a colostomy.

(*c*) *Primary resection* of the inflamed bowel with immediate anastomosis in experienced hands. Primary resection removes the toxic tissues and may lower the mortality of this desperate emergency. Permanent colostomy with closure of bowel below the disease is safer.

All the above procedures must be accompanied by drainage of the abdomen. Simple drainage alone should be reserved for a localised pericolic abscess.

4. *Fistulas* can only be cured by resection of the diseased bowel and closure of the fistula. Ideally these procedures are done at the same operation but a staged procedure with a preliminary colostomy is often necessary. Diverticular disease is a benign condition so the surgeon plays for safety even if it means staged procedures.

5. *Sigmoid Myotomy (Reilly).*—Resection of the thickened sigmoid colon may be avoided by sigmoid myotomy. The operation consists of a longitudinal incision through one tænia extending over the length of the affected colon. The incision is deepened until the mucosa bulges as in a Heller (p. 803) or Ramstedt's (p. 819) operation. This restores the colonic lumen and lowers the intracolonic pressures. The operation must never be used in acute diverticulitis. There is a risk of leakage post-operatively. The operation has not gained general acceptance yet, and other

Michael Charles Tempest Reilly, Contemporary. Previously Surgeon, Plymouth General Hospital, Plymouth, England.

types of myotomy are being tried out, e.g. multiple transverse myotomies of the tænia coli.

DIFFERENTIATION OF DIVERTICULITIS FROM CARCINOMA OF THE COLON

These conditions co-exist in 12 per cent of cases:

	Diverticulitis	*Carcinoma*
History	Long	Short
Pain	More common	
Mass	25 per cent. have tenderness	25 per cent. painless
Bleeding	17 per cent. often profuse, periodic	65 per cent.—usually small amounts persistently
X-ray	Diffuse change	Localised: no relaxation with Probanthine
Sigmoidoscopy	Inflammatory change over an area	No inflammation until ulcer reached

Exploration may be necessary, but even then, differentiation may be difficult until histology is available. Weight loss, falling hæmoglobin, and a persistently positive occult blood test are sinister features.

Solitary diverticulum of the cæcum and ascending colon, is rare, and it is congenital for it has a complete muscular coat. It is usually situated just above the ileocæcal valve, viz.: ——→ Inflammation of such a diverticulum is indistinguishable from acute appendicitis. Recurrent diverticulitis may cause the formation of a mass whose nature can only be ascertained after resection of the cæcum. An uninflamed cæcal diverticulum found casually at laparotomy should be invaginated with a purse-string suture.

ULCERATIVE COLITIS

Ætiology.—The cause is unknown. In spite of intensive bacteriological studies, no organisms or group of organisms can be incriminated. Possibly the disease is linked with emotional stress. It may be related to the auto-immune diseases but this is by no means certain. Some cases are allergic to milk protein. In cases of extensive ulceration secondary infection plays a large part. In some families there is a strong tendency for the disease to occur in successive generations: either a genetic predisposition is inherent in the members of these families, or an unidentified environmental factor is present. The disease has been rare in Eastern populations, but is now being reported more commonly, suggesting an environmental cause that has developed as a result of increasing 'Westernisation' of diet and/or social habits and better access to medical care.

Pathology.—In 95 per cent. of cases the disease starts in the rectum and spreads proximally. When the ileocæcal valve is incompetent, retrograde ('backwash') ileitis involving the last foot (30 cm) of the ileum is liable to occur.

The disease is characterised by the appearance of multiple minute ulcers—sometimes the ulcers are discrete, in others there is a sea of ulceration. Microscopical evidence nearly always proves that the ulceration is more severe and extensive than the gross appearance indicates (Cuthbert Dukes). As time goes on the small ulcers are apt to coalesce to form larger ones, mainly due to the crypts of Lieberkühn becoming distended with pus and bursting into the bowel. When the ulceration ex-

Cuthbert Esquire Dukes, 1890–1977. Pathologist, St. Mark's Hospital, London.
Johann Nathaniel Lieberkühn, 1711–1756. Anatomist, Berlin. He demonstrated his anatomical preparations in London, and was awarded the F.R.S.

tends into the submucosa it causes reflex muscle spasm[1], and the appearance of a stricture. In long-standing cases there is always considerable intramural fibrosis, causing the affected part of the colon to become permanently contracted. In addition, attempts at healing may produce epithelial thickening between the ulcers—the so-called pseudopolyposis.

Clinical Features.—The onset of the disease, more common in women than men is the 3rd, 4th, and 2nd decade, in that order; exceptionally it is encountered in childhood. The first symptom is watery diarrhœa occurring day and night, in a person of previously normal bowel habit. A rectal discharge of mucus, sometimes blood-stained and sometimes purulent is very common. Pain as an early symptom is unusual. In the majority of cases, the disease is chronic, characterised by relapses and remissions. In general, a bad prognosis is indicated by (1) a severe initial attack; (2) disease involving the whole colon; (3) increasing age, especially after sixty years. If the disease remains confined to the left colon, the outlook is better.

FIG. 1097.—Emaciation and dehydration in ulcerative colitis. (*Dr. Peter Hansell, London.*)

Two types are encountered:

Fulminating type (5 per cent.) is ushered in with a temperature of 38·9° to 39·4° C and incessant diarrhœa containing blood, mucus, and pus; the patient looks and feels very ill. There may be abdominal distension due to toxic dilatation of the colon. Here immediate surgery is indicated. The condition must be differentiated from dysentery and typhoid (p. 1010).

Chronic Type (95 per cent.).—As a rule the initial attack is of moderate severity, but exacerbations occur at intervals of weeks, months or, in mild cases, years. As the disease progresses the patient becomes wasted from diarrhœa, and severely anæmic from loss of blood (fig. 1097). Often during the attacks there are ten to twenty stools a day, accompanied by tenesmus. The frequency of the motions and the degree of invalidism go hand in hand, and are proportional to the extent of the involvement of the colon. Lesions of limited length are commonly found in milder cases. Sometimes the disease is confined entirely to the right hemi-colon. When the whole colon is involved the patient may be so weak as to be partially or wholly bed-ridden.

Radiology after a barium enema shows one of the following:

(1) The earliest sign is loss of haustration, especially in the distal colon. Established disease will show:

(2) A narrow contracted (pipe stem) colon (fig. 1098).

(3) Alteration in mucosal outline.

(4) Pseudopolyposis (fig. 1099) in 15 per cent. of cases.

Sigmoidoscopy is indispensable in the diagnosis of early cases and in

[1] Dilatation may occur in acute cases.

FIG. 1098.—Advanced ulcerative colitis. Showing tubular contraction and shortening of the colon. *(W. B. Gabriel, F.R.C.S., London.)*

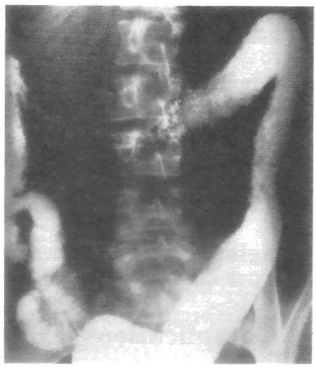


FIG. 1099.—Ulcerative colitis showing pseudopolyposis ('stippled' area in descending colon). *(Dr. Clifford Hawkins, Birmingham.)*

mild cases when the pathological effects of the disease are insufficient to alter the barium shadow. The initial findings are those of proctitis (p. 1102)—the mucosa is hyperæmic, bleeds on touch and there is considerable exudate. Later, tiny ulcers may be seen and appear to coalesce (fig. 1100). This is very different from the picture seen in amœbic dysentery, where there are large deep ulcerations with the intervening mucosa comparatively healthy. As the disease progresses, the ulceration may become so severe that practically no normal mucous membrane remains.

A barium enema and sigmoidoscopy should be avoided during the fulminating phase. Rectal (proctoscope) biopsy is valuable not only in establishing the diagnosis, but also as an index of the effect of therapy.

FIG. 1100.—Ulcerative colitis. The granular inflamed appearance of the mucosa is characteristic. *(After the late J. P. Lockhart-Mummery, F.R.C.S., London.)*

Local Complications

Pseudopolyposis 15 per cent. (see above).

Carcinoma.—Although this is an important complication, it must be seen in its true perspective. The overall risk is about 3·5 per cent.; this risk is much less in early cases but increases with the duration of the disease. Thus, after twenty years of the disease, the risk may be as much as 12 per cent. Carcinoma is more liable to occur where the whole colon is involved and where the disease started in early life. Carcinomatous change may occur at many sites at once and these, often, are atypical. The colon is involved rather than the rectum and maximal incidence is during the 4th decade (Truelove). The golden rule is that after the disease has been present for more than ten years, regular radiological and colonoscopic checks must be done, even though the disease seems to be quiescent. It has been shown that the presence in a rectal biopsy of severe epithelial dysplasia denotes a pre-malignant potential, and may be the only sign that a carcinoma has already developed. At least an annual biopsy of all long-term cases of total colitis is indicated to enable this mucosal change to be detected (Morson and Pang). This precaution is also necessary after operations that have conserved the rectal stump.

Sidney Charles Truelove, Contemporary. Physician, Radcliffe Infirmary, Oxford, England.
Basil Clifford Morson, Contemporary. Pathologist and Director of Research, St. Mark's Hospital for Diseases of the Rectum and Colon, London.
Lillian Shu Chao Pang, Contemporary. Histopathologist in Charge, Tumour Reference Collection, Imperial Cancer Research Fund, London.

Fibrous Stricture occurs in 6 per cent. of cases. The common sites are the recto-sigmoid junction and the anal canal. Although they usually respond to simple dilatation where the disease is confined to the distal colon, surgery may be advisable if the whole colon is involved.

Toxic Dilatation—(1·5 per cent) occurs in the fulminating type of the disease. It may result in perforation. Both require immediate surgery. Excessive steroid therapy may be a factor. Frequently the only sign that perforation has occurred is abdominal distension. This is a cardinal sign of impending doom.

Massive Hæmorrhage (3 per cent.) usually arises from the rectum rather than the colon.

Recto-vaginal Fistulæ[1] (3 per cent.), **Fistula in Ano** (4 per cent.), **Ischio-rectal Abscess** (4 per cent.), and **Hæmorrhoids** (20 per cent) also occur (Truelove).

General Complications

Liver Changes (7 per cent.) sometimes resulting in cirrhosis, may occur as the result of heavy protein loss and portal toxic absorption.

Skin Lesions (18·5 per cent.) including Pyodermia Gangrenosum (0·5 per cent.) and Erythema Nodosum (2 per cent.).

Arthritis (5 per cent.) usually of small joints, may occur at any stage of the disease.

Iritis (7 per cent.), **Ankylosing Spondylitis** (2 per cent.), **Stomatitis** (10 per cent.), **Renal Disease** (5 per cent.) and **Anæmia** (20 per cent.) are also important complications.

Treatment

Several agents may induce a remission but it is difficult to predict the outcome in individual cases. The main *general principles* are:

(1) Maintenance of fluid and electrolyte balance.

(2) Anæmia must be speedily corrected.

(3) Adequate nutrition—at least 3,000 C (12,560 kJ) per day—with high protein, carbohydrate and vitamin content and low fat.

(4) Sedatives and tranquillizers are useful. Antibiotics may precipitate moniliasis.

Specific Treatment

Quiescent Cases.—Are best maintained on Salazopyrin (2 g daily). This has been shown to reduce the incidence of relapse.

Mild to Moderate Relapses.—The dose of Salazopyrin is increased (4–6 g daily). Topical steroids are of real value particularly in the distal type of the disease. Retention enemata of prednisolone phosphate 20 mg in 100 ml daily are commenced. If the response is inadequate then as in—

Acute Fulminating Cases.—Systemic prednisolone 20 mg or more daily may be required for up to three weeks.

Dangers of Prolonged Steroid Therapy.—1. Not only is there an increased risk of massive hæmorrhage and of free perforation into the peritoneal cavity, but in patients who have not responded to a course of steroids over a period of four to six weeks, the colonic wall becomes excessively friable and in some areas disintegrates, its place being taken by the parietes or adjacent viscera, usually the small bowel. In such cases the surgeon may then be unable either to remove the colon or even to perform ileostomy (Brooke).

2. Steroid therapy renders the patient more susceptible to pyogenic infection.

[1] Recto-vaginal and Anal Fistulæ usually mean that the underlying condition is Crohn's disease (p. 1010) and not ulcerative colitis.

Bryan Nicholas Brooke, Contemporary. Formerly Professor of Surgery, St. George's Hospital, London.

Indications for Surgery

(1) To save life—fulminating cases, toxic dilatation, severe hæmorrhage, perforation.

(2) Local complications (p. 1005).

(3) General complications (p. 1006).

(4) Risk of neoplastic change—especially in pseudo-polyposis and colitis of long standing.

(5) Onset in children or adolescents.

(6) Chronic invalidism.

Between 15 and 20 per cent. of sufferers are recommended for surgical treatment by their physicians.

When surgical treatment is advised, about six days' intensive pre-operative preparation by the methods already enumerated is carried out.

Operations

(i) The ideal procedure is *a one-stage total procto-colectomy with ileostomy*. If the disease is acute, it is wise to leave the rectal stump and either close it or bring it out as a midline colostomy or mucous fistula. The rectum can be removed about eight weeks later.

(ii) Some surgeons advocate *total colectomy* with immediate or subsequent *ileo-rectal anastomosis*—this can only be done satisfactorily in about 5 per cent. of cases where the rectum is not involved. Some surgeons have reported a much greater degree of success, and use total colectomy with ileo-rectal anastomosis in the majority of their cases (Aylett). If the degree of rectal inflammation makes the ileo-rectal anastomosis unsound, a temporary loop ileostomy proximal to the anastomosis is employed. After the operation a physiological bowel frequency of three to five actions a day is to be expected. It should not be performed if peri-anal complications are present. Should ileo-rectal anastomosis fail and the disease recur in the rectal stump, an ileostomy can be performed.

(iii) *Ileostomy alone* may sometimes be indicated for a gravely ill patient with fulminating disease and localised abscess around a perforation.

Colectomy is not a difficult operation when it is performed on an emaciated individual whose peritoneal cavity is free from adhesions. After a successful ileostomy both these desiderata are absent; firstly, the patient has gained weight and, secondly, adhesions are bound to be present, especially in the right iliac fossa.

Ileostomy

As the ileostomy is nearly always permanent, the success of the operation lies in scrupulous attention to the details of technique in siting and fashioning the ideal 'spout'. It is best to arrange the position of the stoma beforehand with the patient (fig. 1101).

A disc of skin not more than 3 cm. in diameter is removed at least 5 cm. lateral to the umbilicus (fig. 1101).

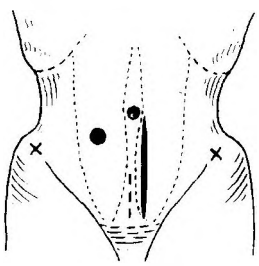


FIG. 1101.—Site of the ileostomy. The 'trephine' wound should be at least 5 cm. from all scars and bony points.

Stanley Aylett, Contemporary. Surgeon, Westminster Group Hospitals, and the Gordon Hospital, London.

FIG. 1102.—Suture of the mesentery to the parietal peritoneum, to prevent prolapse. *(After J. C. Goligher, F.R.C.S., Leeds.)*

The abdomen is opened by a left paramedian incision and the ileum is divided near its termination, together with its mesentery. It may be necessary to divide the ileum more proximally if the terminal ileum is diseased also.

Important steps in the performance of ileostomy are (*a*) to close the peritoneal space on the external side of the ileostomy (otherwise a tunnel exists which invites intestinal obstruction), and (*b*) to anchor the anterior edge of the mesentery to the parietal peritoneum (fig. 1102). The latter prevents prolapse of the mucous membrane. The ileostomy opening must be prepared meticulously. A disc of tissue equal to the size of the skin disc should be removed from the anterior and posterior rectus sheath so that no stricture will occur in the parieties. The peritoneum is opened by a cruciform incision. About 7·5 cm. of the divided ileum (closed temporarily with a noncrushing clamp) is brought through the opening and its periphery is stitched to the skin edges by interrupted catgut in such a way that it is everted to form a 'spout' projecting 4 cm. from the skin surface. The greatest care must be taken in the construction of this projection (fig. 1103). A proctocolectomy is carried out and the abdomen is closed. A disposable plastic ileostomy bag (fig. 1104) is cut so that it exactly fits the skin at the muco-cutaneous junction and is fixed in position by latex rubber adhesive.

FIG. 1104.—Disposable ileostomy bag. *(Bryan N. Brooke, F.R.C.S., London.)*

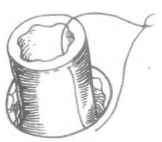


FIG. 1103.—Suturing the free extremity of the proximal ileum to the skin edges.

Care of Ileostomy. — During the first few post-operative days the fluid electrolyte balance must be adjusted with great care. For permanent use it is wise to fit a Chiron-type bag which has a firm rubber flange to be attached around the ileostomy spout. It is supported both by a waist strap and also made adherent to the skin by special double-sided adhesive plaster. The bag is attached to and detached from the flange which remains adherent to the skin for as long as is convenient. Great care must be taken that the lower rim of the bag does not press on and penetrate the lower margin of the ileostomy 'spout'. If soreness and excoriation of the skin occur as a result, a paste of aluminium 10 parts and zinc oxide 90 parts is helpful. Also a paste of Karaya gum (Goligher) acts as an adhesive and protective. The stools thicken in a few weeks and are semi-solid in a few months. A thin discharge from the stoma may indicate bolus obstruction and stenosis ; digital dilatation will help to relieve this.

Continent Ileostomy (Koch).—Professor Nils Koch has recently developed an ileostomy with a 'reservoir' in the terminal ileum that opens to the surface by a one-way valve that prevents any escape of ileal effluent until a catheter is passed through the valve-spout to empty the ileal pouch. This type of ileostomy requires no bags or appliances and can be emptied at will by the patient. The ileostomy is called 'continent' for this reason. The continent ileostomy is not easy to construct, and has a considerable morbidity: even the occasional death has been reported. It should not be used routinely at present.

Procto-colectomy requires no special description. The cæcum and colon are detached from their mesenteries and when the rectum is reached the procedure is exactly as for a combined abdomino-perineal resection (p. 1119). If the patient is too ill to stand the perineal dissection, the rectum is freed as far as the pelvic diaphragm and is transected at this point. The large intestine is removed, the upper end of the

John Cedric Goligher, Contemporary. Emeritus Professor of Surgery, Leeds, England.
Professor Nils Koch, Contemporary. Professor of Surgery, Malmo, Sweden.

rectum closed and the pelvic peritoneum united over it. Alternatively, the upper end of the preserved distal segment can be brought out as a colostomy through the paramedian incision (' mucous fistula ').

INTESTINAL AMŒBIASIS

Amœbiasis denotes an infestation with *Entamœba histolytica*. Contrary to general belief, *E. histolytica* is not strictly a tropical parasite: *it has a world-wide distribution*, and is found in Great Britain particularly in the Southern counties. It is surprisingly common in overcrowded institutions. The reason why in some cases it becomes pathogenic is unknown.

Life History of the Parasite.—The active form of the parasite or trophozoite lives in the intestinal mucous membrane, where it ingests red blood corpuscles and other cells, and multiplies by mitosis. Should the parasite become pathogenic, it makes its way into the follicles of Lieberkühn, and by dissolving interglandular tissue by cytolysins, submucous loculi are produced. Some of these burst through the mucous membrane to become amœbic ulcers. While the trophozoites continue their activities in the base of the ulcer, others cease to feed, migrate towards the surface, and become transformed into cysts (fig. 930), which pass into the outer world with the fæces. Amœbiasis is transmitted mainly in drinking water.

Pathology.—The ulcers, which have been described as ' bottle-necked ' because of their considerably undermined edges, have a yellow necrotic floor, from which blood and pus exude. While on rare occasions the ulcers are scattered throughout the large intestine, in 75 per cent. they are confined to the lower sigmoid and the upper rectum.

Biopsy material is obtained by scraping the suspected area of mucosa with a long-handled Volkmann's spoon; this removes a thin slice of the diseased mucosa, which must be examined immediately. It should be noted particularly that the finding of *Entamœba histolytica* is not conclusive evidence that the symptoms are due to amœbiasis. On numerous occasions a positive slide examination has caused the clinician to assume that the diagnosis has been established when there has been a proximal carcinoma of the colon and the *Entamœbae histolyticae* have been non-pathogenic and incidental.

Clinical Features.—Dysentery is only one manifestation of the disease. In various guises amœbiasis obtrudes itself into the surgeon's diagnostic arena:

Appendicitis or Amœbic Typhlitis[1] *?*—In tropical countries where amœbiasis is endemic, this is a constantly recurring problem. To operate upon a patient with amœbic dysentery without the precautions subsequently described is to invite an exacerbation of amœbiasis that may prove fatal. Especially in cases where a palpable mass is present, the bowel is friable and satisfactory closure of the appendix stump becomes difficult or impossible. The death-rate from peritonitis and wound infection in the notorious Chicago epidemic of amœbiasis in 1933 was appalling, which emphasises that surgeons in temperate climates should be familiar with the condition. In the case of amœbic typhlitis there is rarely rigidity, and pain commences in the right iliac fossa. In amœbic typhlitis there are two characteristics and localised zones of tenderness on deep palpation—one over the cæcum and one over the sigmoid. The latter is sharply defined, and being comparable to McBurney's point on the right side, it has been aptly named ' *the amœbic point* ' by Sir Philip Manson-Bahr. Routine sigmoidoscopy is of great value. If doubt still exists, 60 mg of emetine hydrochloride in 20 ml. of normal saline solution given intravenously very slowly is likely to ameliorate the symptoms within two hours (Andreasen).

Perforation.—The most common sites are the cæcum and recto-sigmoid. Usually perforation occurs into a confined space where adhesions have previously formed, and a pericolic abscess results, which eventually needs draining. When there is sudden fæcal flooding of the general peritoneal cavity, drainage of the region of the perforation, gastro-intestinal aspiration, intravenous fluid replacement, antibiotic therapy, and a full course of emetine are sometimes successful.

[1] Typhlitis = inflammation of the cæcum.

Richard von Volkmann, 1830–1889. Professor of Surgery, Halle, Germany.
Charles McBurney, 1845–1913. Surgeon, Roosevelt Hospital, New York, U.S.A.
Sir Philip Manson-Bahr, 1881–1966. Physician to the Hospital for Tropical Diseases, London.
Anthony Turner Andreasen, Contemporary. *(See footnote p. 864.)*

Severe rectal hæmorrhage due to the separation of sloughs is liable to occur.

Granuloma.—Progressive amœbic invasion of the wall of the rectum or colon, with secondary inflammation, may produce a granulomatous mass indistinguishable from a carcinoma. The exhibition of emetine as a therapeutic test will prevent mistakes in diagnosis. Amœbiasis and carcinoma occasionally co-exist (Naunton Morgan).

Fibrous stricture may follow the healing of extensive amœbic ulcers.

Intestinal obstruction is a common complication of amœbiasis, and the obstruction is due to the adhesions associated with pericolitis and a large granuloma.

Paracolic abscess, ischio-rectal abscess, and fistula occur from perforation by amœbæ of the intestinal wall followed by secondary infection.

Ulcerative colitis.—A search for amœbæ should always be made in the stools of patients believed to be suffering from ulcerative colitis.

Treatment.—Metronidazole (Flagyl) is the first line drug, 800 mg t.d.s. for 7–10 days. The patient must avoid taking alcohol. Emetine hydrochloride, 60 mg daily s.c. for 4 days, is the alternative if metronidazole cannot be taken by mouth. The patient must be kept in bed during and for one day after the course of emetine. Diloxanide furoate 500 mg t.d.s. for 10 days is another intra-lumenal drug. Intestinal antibiotics, e.g. tetracycline, improve results in the chronic stages, probably by coping with superadded infection.

THE SURGICAL COMPLICATIONS OF TYPHOID AND PARATYPHOID

Chloromycetin exerts a rapidly curative effect on typhoid and paratyphoid infections; consequently complications are uncommon. When complications of typhoid arise, chloromycetin should be given in addition to other necessary treatment, not forgetting that this antibiotic destroys the organisms responsible for the production of vitamin B complex which must be replaced.

1. *Paralytic ileus* is the commonest complication of typhoid (p. 1052).

2. *Intestinal hæmorrhage* may be the leading symptom. The condition must be distinguished from purpura with intestinal symptoms, and intussusception. A Widal reaction should be employed and, if negative, repeated in suspected cases. Urgent blood transfusion will be required.

3. *Perforation.*—Perforation of a typhoid ulcer usually occurs during the third week; occasionally it is the first intimation of the disease (ambulatory typhoid). The ulcer is parallel to the long axis of the gut (fig. 1105), and is situated in the lower

FIG. 1105A.—A typhoid ulcer is longitudinal (Peyer's patch necrosis).

FIG. 1105B.—A tuberculous ulcer is transverse (because it follows the lymphatics).

ileum. In paratyphoid B, perforation of the large intestine sometimes occurs. The former treatment was to perform laparotomy under local anæsthesia, and to close the perforation. The results were so poor that the conservative treatment of peritonitis plus chloromycetin yields better results.

4. *Cholecystitis.*—Acute typhoid cholecystitis is not uncommon and perforation can occur (p. 922). Gallstones occasionally contain typhoid bacilli. Chronic typhoid cholecystitis can result in the patient becoming a typhoid carrier.

5. *Phlebitis.*—Venous thrombosis, particularly of the left common iliac vein, is an occasional complication of typhoid fever.

6. *Genito-urinary Complications.*—Typhoid cystitis, pyelitis, bacilluria, and epididymo-orchitis all occur.

7. *Joints.*—All degrees of arthritis, from a mild effusion to suppuration, occur as a complication of this disease.

8. *Bone.*—Typhoid osteomyelitis and typhoid spine.

9. *Larynx.*—Typhoid perichondritis is met with occasionally, and typhoid laryngitis has been known to obstruct the airway.

REGIONAL ILEITIS (*syn.* CROHN'S DISEASE)

Ætiology.—No causative organism has been found in the lesion or in the stools although abnormal forms of B. coli have been discovered in most patients.

Sir Clifford Naunton Morgan, Contemporary. Consulting Surgeon, St. Bartholomew's Hospital, London.
Burrill Bernard Crohn, Contemporary. Gastroenterologist, Mount Sinai Hospital, New York. First described this disease in 1932.

Dogs receiving finely powdered silicates admixed with their food for long periods develop an identical condition, and it has been suggested that the ingestion of fine, insoluble particulate matter such as toothpaste is a possible cause of regional ileitis. A disease similar to regional ileitis occurs in swine. Electron microscope studies have shown that in Crohn's disease fæcal bacteria enter the colonic wall. Whether it is these or something ingested or a metabolic product which acts as the hapten is not known, but granulomas develop (Aluwihare). There is evidence that heredity is a factor. Possibly Crohn's disease develops in patients with a relatively complete genotype. Ulcerative colitis is common in relatives of patients with Crohn's disease, but the converse is not common, suggesting that people with ulcerative colitis may have fewer of these genes. As with ulcerative colitis it is now believed that Crohn's disease is a pre-cancerous condition.

Pathology.—Regional ileitis is essentially a cicatrising inflammation with ulceration of the mucosa. It usually commences at or near the ileo-cæcal valve, and extends upwards along the ileum for about 30 cm, but as little as 5 cm, and, more often, as much as 1·2 metres may be implicated. In acute cases the affected intestine is seen to be swollen, bright pink in colour, and with a fibrinous exudate on its peritoneal surface; in chronic cases hyperæmia is less in evidence. On palpation the intestinal wall feels like a hose-pipe. The mesentery of the involved intestine is exceedingly thickened, œdematous, and contains enlarged and fleshy lymph nodes. Unlike tuberculosis, the affected lymph nodes neither break down nor calcify. Tracing the diseased ileum upwards, it terminates abruptly in normal intestine. Above this there is commonly another (short) area of diseased intestine; this is a so-called ' skip ' lesion. Doubtless a comparatively inconspicuous additional lesion is sometimes overlooked, and is one cause of recurrence after resection. In 6 per cent. of cases there is an extension into the cæcum and at times the ascending colon is implicated in the interrupted manner referred to. Examples of primary Crohn's disease of the colon, jejunum, duodenum, and even of the stomach and anus have been reported, which justifies the newer term, regional enteritis.

Pathological Histology.—A characteristic finding is granulomatous infiltration of lymphatics of the submucosa with the presence of non-caseating giant-celled systems. In the late stages of the disease fibrosis extends into and obliterates the submucosa, but usually giant-celled systems can be found in the related mesenteric lymph nodes. An experienced histologist can easily distinguish the condition from tuberculosis.

Clinical Features.—The disease, which is independent of age, sex, social and economic conditions, or geographical location, is increasing in frequency. To some extent it is familial.

Acute regional ileitis occurs only in 5 per cent. of cases. The symptoms and signs resemble those of acute appendicitis, with one exception, viz. diarrhœa almost invariably precedes the acute attack. Exceptionally, perforation of the intestine, resulting in local or diffuse peritonitis, occurs.

Chronic regional ileitis is the usual form of the disease. It can be divided into three stages, but sometimes the second stage is lacking.

First Stage.—There is a history of mild diarrhœa extending over months or years, occurring continuously or in bouts accompanied by intestinal colic, relieved by defæcation. Intermittent pyrexia, seldom more than (37·2° C) is usual, but some patients are afebrile throughout. As a rule a tender mass can be felt in the right iliac fossa, and frequently by a pelvic examination also. There is often a moderate secondary anæmia. Occult blood and some mucus is present in the stools; two-thirds of the patients have some degree of steatorrhœa. *A perianal abscess* is a frequent accompaniment of early Crohn's disease. The cause is probably an infected anal crypt associated with the concomitant diarrhœa. The high incidence and diagnostic signifi-

A. P. R. Aluwihare, Contemporary. Surgeon, Sri Lanka.

cance of perianal and perirectal abscesses and fistulæ in patients with regional ileitis is generally accepted.

Second stage is characterised by symptoms of acute or chronic intestinal obstruction. Cicatrisation of the granulomatous area has progressed to such

FIG. 1106.—Crohn's disease. Stage of cicatricial contracture.
(From the R.C.S. Museum, Professor G. Cunningham.)

an extent that the lumen of the affected portion of the intestine is narrowed (fig. 1106).

Third stage is that of adhesions sometimes accompanied by slow perforation of the intestinal wall. Adhesions are dense, abscess formation is common, and fistulous tracts are wont to develop:—

(*a*) *Internally* into neighbouring hollow viscera, e.g. a redundant pelvic colon, or occasionally into the right side of the bladder. In all cases of enterocolic and vesico-intestinal fistulæ the possibility of regional ileitis should be considered.

(*b*) *Externally*, nearly always through the scar of a previous operation for the condition, e.g. appendicectomy.

Radiological Diagnosis.—X-ray examination after a barium meal often shows lack of segmentation and feeble or absent peristalsis in the affected portion of the intestine, the lumen of which remains constant in diameter. Radiologically, cases can be divided into stenosing and non-stenosing. In the non-stenosing phase straightening of the valvulæ conniventes is characteristic. When ulceration has occurred multiple defects (cobblestone reticulation) can be seen after the barium has been evacuated from the segment in question. When cicatrisation has occurred the radiograph is particularly characteristic; sometimes the terminal ileum is so constricted that the 'string' sign of Kantor (fig. 1107) is seen. Characteristic linear fissures

John Leonard Kantor, 1890–1947. Gastroenterologist, Presbyterian Hospital, New York, U.S.A.

extending outwards through the bowel wall ('raspberry thorn' and 'rose thorn' types are very typical) are frequently seen outlined by the barium.

Treatment:

FIG. 1107.—The 'string' sign of Kantor.

Medical Treatment.—In the early stages medical treatment is given an extended trial. It consists of rest in bed, a high protein diet with vitamin supplementation. Iron or blood transfusion may be required. Antibiotics—sulphaphthalidine is best—salazopyrine or steroids (e.g. prednisone or A.C.T.H. depot) are used with caution. In the acute form of the disease, particularly acute colitis, azathioprine has proved effective and may make colectomy unnecessary. It may also reduce fistula formation if operation is required. The drug may also have a place in the treatment of recurrences but not where obstruction is present. The response is less where the disease is chronic. Antibiotics are sometimes successful in abating an acute attack; Gentamycin plus Metronidazole are a good combination to try first of all.

Indications for Surgery.—These are: (1) failure to arrest the course of the disease by adequate medical treatment; (2) intestinal obstruction; (3) the presence of fistulæ.

Operation.—Should the abdomen be opened on the mistaken diagnosis of acute appendicitis and acute regional ileitis is found, the one thing *not* to do is to remove the appendix. Appendicectomy frequently determines the development of an external fistula. The correct procedure is to close the abdomen forthwith. Occasionally the condition resolves completely; more often chronic ileitis supervenes.

Surgeons differ as to whether the affected bowel should be resected or by-passed. Probably a by-pass operation is best for young people, especially if the disease is in a florid state, and resection for older patients with firm strictures and where the main trouble is in the cæcum and colon.

On the whole, unless circumstances, such as an enterocolic fistula, leave no other alternative, many surgeons still follow the advice of Crohn and his surgical colleagues, who for many years have recommended the conservative by-pass type of operation. The mortality is almost zero, and the results are at least as good as those following resection. In the cases that do not improve as a result of a short circuit, resection can still be undertaken. In experienced hands, an immediate one-stage resection is as safe as a by-pass procedure and has become the operation of choice.

TUBERCULOSIS OF THE INTESTINE

In countries where the Public Health control of tuberculosis is not strict, the disease is still common.

1. **Ulcerative tuberculosis** is secondary to pulmonary tuberculosis, and arises

as the result of swallowing tubercle bacilli. It is characterised by the presence of multiple ulcers in the terminal ileum, the long axis of each ulcer lying transversely (fig. 1105). The serous coat overlying the ulcerated segment is thickened, injected, and sparsely bespattered with tubercles. Perforation is rare, but in patients who overcome the infection subsequent stricture or strictures of the ileum are rather frequent.

Clinical Features.—Diarrhœa is the predominant symptom; there is also loss of weight. The stools have a fœtid odour, and contain pus and occult blood. Often the patient has received, or is receiving, treatment for pulmonary tuberculosis; more rarely pulmonary tuberculosis is detected for the first time in the course of the investigations.

Radiology.—A barium meal often discloses complete absence of filling of the lower ileum, the cæcum, and most of the ascending colon, due to hypermotility of the ulcerated segment.

Treatment.—A prolonged course of chemotherapy (p. 37) is given. Provided the intestinal ulceration is not a terminal event of advanced pulmonary tuberculosis, healing often occurs. Operation is required in the rare event of perforation. Occasionally cicatrisation causes intestinal obstruction, and calls for surgical intervention.

2. **Hyperplastic tuberculosis** occurs most commonly in the ileo-cæcal region, although solitary or multiple lesions of the lower ileum are met with occasionally. This form of intestinal tuberculosis is consequent upon the ingestion of *Mycobacterium tuberculosis* by a patient with a high resistance to the organism. In Western countries *mycobacterium tuberculosis bovis* is often the causative organism, while in the East the human variety is the culprit (Toufeeq). The infection establishes itself in lymphoid follicles, and spreads to the submucous and subserous planes. The resulting chronic inflammation causes much thickening of the intestinal wall, and consequent narrowing of its lumen. There is early involvement of regional lymph nodes, which may caseate. Unlike regional ileitis (which in many respects this disease simulates), abscess and fistula formation are rare.

Untreated, sooner or later subacute or acute intestinal obstruction supervenes, and in the East, not infrequently, impaction of an enterolith within the narrowed lumen is the precipitating cause.

Clinical Features.—Attacks of abdominal pain with intermittent diarrhœa are the premonitory symptoms. Frequently, the presenting picture is that of the 'blind loop' syndrome (p. 996). The ileum above the partial obstruction is distended and the stasis and consequent infection lead to steatorrhœa, anæmia and loss of weight. Sometimes the presenting picture is that of a mass in the right iliac fossa in a patient

FIG. 1108.—The difficulties in diagnosis of a mass connected with the cæcum. In this case the lump outlined on the skin was thought at first to be an *appendix abscess*. After the cæcum and ascending colon had been excised the specimen had many of the appearances of *tuberculosis*. Histologically the mass proved to be a *carcinoma*.

with vague ill health. This finding raises the possibility of an appendix mass, a carcinoma of the cæcum, Crohn's disease or a tuberculous or actinomycotic granuloma of the cæcum. The problem can be quite perplexing (fig. 1108).

Radiography.—In an established case a barium meal reveals a long, narrow filling defect consisting of the terminal ileum and ascending colon, lying vertically. The cæcum may become subhepatic (fig. 1109).

Treatment.—When the diagnosis is certain, and the patient has not yet developed obstructive symptoms, sanatorium treatment with chemotherapy is advised and may completely cure the condition. If obstruction is present, operative treatment is required. Right hemicolectomy with removal of the diseased segment of ileum is the treatment of choice. In patients with intestinal obstruction, and those in poor general condition, a defunctioning ileocolostomy similar to that recommended for regional ileitis is advisable. If necessary resection can be undertaken later, for

Ahmed Khan Toufeeq, Contemporary. Professor of Surgery, Sir Ganga Ram Hospital, Lahore, Pakistan.

frequently striking improvement occurs after ileocolostomy and general treatment. However, without formal resection, there is always a considerable risk of further recrudescence of the disease, which may involve the recent by-pass anastomosis as well.

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA

Actinomycosis develops when a breach of the mucous membrane, caused by disease or trauma of a foreign body, permits the entry of *Actinomyces israeli* (p. 38) into the deeper layers. Abdominal actinomycosis is rare. Unlike intestinal tuberculosis, cicatrisation and consequent narrowing of the lumen of the intestine do not occur, neither do the mesenteric lymph nodes become involved. However, suppuration supervenes, and the disease spreads into the retroperitoneal tissues, the abdominal wall becomes the seat of multiple indurated discharging sinuses, and the liver becomes involved by way of the portal vein.

FIG. 1109.—Ileocæcal tuberculosis. The filling defect also occurs in carcinoma of the cæcum. *(Professor A. K. Toufeeq, Lahore, Pakistan.)*

Clinical Features.—The usual history is that appendicectomy has been performed for appendicitis. Possibly, had the appendix been subjected to histological scrutiny, actinomyces might have been found. More usually this examination is omitted or the organism is not discovered, and about three weeks after the operation a mass forms in the right iliac fossa, and soon afterwards the wound commences to discharge. At first the purulent discharge is thin and watery; later, because of secondary infection, it becomes thicker and odorous. Other sinuses form, and fæcal fistulæ are liable to develop. At any stage of the disease, if pus is collected and allowed to trickle down the side of a test-tube, sulphur granules may be discovered. A swab is insufficient. The pus should be sent for immediate bacteriological examination.

Another clinical type is that of a patient, most usually a young adult male, who presents with vague abdominal pain. On examination a firm, slightly tender mass is found in the right iliac fossa. Extension of the disease to the psoas muscle sometimes causes flexion of the hip. Little or no help is derived from radiology in distinguishing actinomycosis of the right iliac fossa from hypertrophic tuberculosis or carcinoma of the cæcum; a deformity of the cæcum is found in all these conditions. Actinomycosis rarely gives rise to obstructive symptoms. When, as is sometimes the case, there is a history of a more or less sudden onset of pain some weeks previously, a subsiding appendix abscess is probably diagnosed, but the mass does not resolve. There is loss of weight, anæmia, and occasional pyrexia. In most instances laparotomy is performed with one of the following findings: (*a*) an abscess is encountered and drained; (*b*) the mass is found to be densely adherent to the posterior abdominal wall, and irremovable—needing a by-pass ileo-transverse colostomy as for Crohn's disease (p. 1010); (*c*) the mass is sufficiently mobile for right hemi-colectomy to be carried out.

Treatment (p. 39).

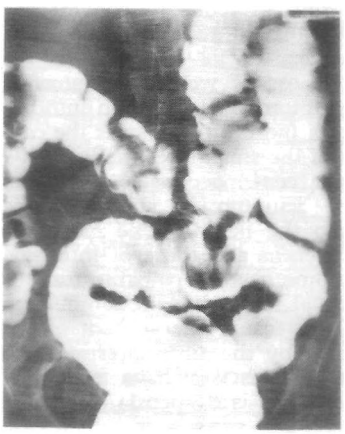


FIG. 1110.—Barium enema showing 'Scalloping' of the edges of the sigmoid, typical of the condition.

PNEUMATOSIS CYSTOIDES INTESTINALES

Multiple gas cysts of the intestinal wall are pathological curiosities. Translucent, thin-walled cysts ranging from 1 to 2 or more centimetres in diameter, containing gas, mainly nitrogen, and having a lining of flattened cells of doubtful origin, occur in clusters under the serosa or in the mesenteries on the intestines. The condition nearly always affects the small intestine, but occasionally the colon, and even the rectum, are implicated. It is believed that air enters a breach in the mucosa, as

would occur in the case, say, of a duodenal ulcer, and the air is driven onwards by peristalsis. The cysts, which are obvious on a plain radiograph, are usually symptomless, but occasionally they so occlude the lumen of the intestine that resection is necessary. A new treatment using an oxygen-enriched atmosphere for the patient to breathe over several weeks has been successful in causing the cysts to disappear, but they usually come back within a year of the treatment. The success of the treatment depends upon replacing the nitrogen in the cysts by oxygen, which is then re-absorbed with ensuing collapse of the cysts.

TUMOURS OF THE SMALL INTESTINE

Compared with the large intestine, the small intestine is rarely the seat of a neoplasm.

Benign.—Adenoma, submucous lipoma and leiomyoma occur from time to time, and sometimes reveal themselves by causing an intussusception. The second most common complication is intestinal bleeding from an adenoma, in which event the diagnosis is frequently long delayed because the tumour is overlooked at a radiological examination by barium meal, and often at operation as well, to be discovered perhaps only after a second or third operation.

Peutz-Jeghers syndrome (small intestinal polyposis) consists of (*a*) familial intestinal hamartomatous polyposis affecting mainly jejunum, where it is a cause of hæmorrhage, and often of intussusception, and (*b*) melanosis of the oral mucous membrane and the lips. The melanosis takes the form of melanin spots which are sometimes present on the digits and the peri-anal skin, but the pigmentation of the lips (fig. 1111) is the *sine qua non*. *Histology*.—The polyps can be likened to trees. The trunk and branches are smooth muscle fibres and the foliage is virtually normal mucosa.——————→

Treatment.—As a malignant change rarely occurs, resection is necessary only for serious bleeding or intussusception. Large single polyps can be removed by enterotomy, or short lengths of heavily involved intestine can be resected. It is now possible to snare these lesions by means of a fibrecolonoscope.

FIG. 1111.—Melanin spots on the lips of a patient afflicted with Peutz-Jeghers syndrome. (*Major P. C. M. Mehta, Indian Medical Service.*)

Malignant :

Sarcoma (40 per cent.).—Lymphosarcoma and spindle-cell sarcoma—more usually the former—occur in the first five decades, the average age of the patient being thirty-five years. There are usually multiple lesions and the disease may follow idiopathic steatorrhœa. The neoplasm tends to convert the affected intestine into a rigid tube without much interference with the size of its lumen until the disease is advanced. Loss of weight and anæmia are the chief early symptoms. Perforation into the peritoneal cavity occurs more often than is the case with other neoplasms of the small intestine.

Carcinoma (35 per cent.) occurs at the usual carcinoma age. The jejunum is affected three times more often than the ileum. The most frequent symptoms are melæna and those of intestinal obstruction, and in some cases a palpable tumour is present. It is unusual for malignant tumours to cause intussusception. Because the content of the small intestine is fluid, by the time intestinal obstruction has ensued metastases have occurred. Another train of symptoms is dyspepsia associated with melæna and increasing anæmia, in which case a tumour, often of the papilliferous variety, may be revealed radiologically at a barium meal.

Carcinoid (Argentaffin) Tumour (25%).—These tumours occur throughout the gastrointestinal tract and also in the bronchus, testis and ovary.

John Law Augustine Peutz, 1886–1957. Chief Specialist for Internal Medicine, St. John's Hospital, The Hague, Holland.
Harold Jos Jeghers, Contemporary. Professor of Internal Medicine, New Jersey College of Medicine and Dentistry, Jersey City, U.S.A.

The appendix is the commonest site (65%) p. 1074. The next most common site is the ileum (25%). They arise in the cells of Kulchitsky at the base of the crypts of Lieberkühn. The primary is usually less than 10 mm in diameter. When they metastasise, the liver is usually involved with numerous secondaries, larger and more yellow than the primary growth. When this has occurred the 'carcinoid syndrome' may become evident.

The tumour produces 5-H.T. (hydroxytryptamine; serotonin) which may be present as 5 hydroxy indole-acetic acid in the urine during attacks. It also produces kinins, indoles, prostaglandins and perhaps histamine.

The clinical syndrome itself consists of reddish-blue cyanosis, flushing attacks diarrhœa, borborygmi, asthmatic attacks, and eventually sometimes pulmonary and tricuspid stenosis. Classically the flushing attacks are induced by alcohol (fig. 1112).

The cyanotic flushing and asthmatic attacks have been attributed to 5-H.T. and histamine—the brick-red to kinins.

Five-H.T. is responsible for the gastrointestinal symptoms. Prostaglandins may have similar effects to the kinins.

FIG. 1112.—Patient with secondary carcinoid tumour of the liver (primary in the terminal ileum) exhibiting flushing after a small dose of alcohol. (*Dr. P. J. D. Snow, Manchester.*)

Treatment.—This tumour must be taken seriously. A radical operation is advisable where metastases are not found. For example, right hemicolectomy for lesions in the ileum or abdomino-perineal for lesions low in the rectum.

Where metastases have occurred, excision of the local lesion and if feasible partial hepatectomy should be considered. It should be borne in mind, however, that such patients may survive for up to 10 years having frequent carcinoid attacks. Medical measures include giving serotonin antagonists, long acting anti-histaminics and alpha-methyl dopa which may help to reduce the severity of attacks. For hepatic metastases 5-fluorouracil given into the hepatic artery is under trial.

TUMOURS OF THE LARGE INTESTINE

Benign

Adenomatous Polyps are dark red pedunculated neoplasms (fig. 1113). A *villous adenoma* is simply a different morphological expression of the same pathological process, and is usually sessile, and may cause diarrhœa and hypokalæmia.

A solitary adenoma of the colon (fig. 1114) is acquired (see below for the multiple familial variety). It occurs in patients over forty years of age, and unless it is pedunculated, it cannot be distinguished macroscopically from a papilliferous carcinoma. In specimens of papilliferous carcinoma of the colon, often tiny adenomas are found adjacent to the parent tumour.

Treatment.—Because the lesions have a malignant potential they should be removed. Those beyond the reach of the sigmoidoscope require laparotomy, but this should only be undertaken if the lesion is larger than 1 cm in diameter and has been demonstrated on two barium enemas. Fibre endoscopic removal is available in an increasing number of centres.

Familial Adenomatous Polyposis of the Colon, which must be distinguished from Peutz-Jegher's syndrome (p. 1016), juvenile polyposis coli (p. 1114), and pseudopolyposis (p. 1004), is transmitted from both sexes to both sexes, though males are affected more frequently than females. Lockhart-Mummery and Cuthbert Dukes

Nicholas Kulchitsky, 1856–1925. Professor of Histology, Kharkov, Ukraine, U.S.S.R.
Hugh Evelyn Lockhart-Mummery, Contemporary. Surgeon, St. Thomas's Hospital, London.

studied 1,069 members of 58 families; of these members 218 had polyposis, and 154 of them developed colonic carcinoma. The adenomatous polypi are most frequently situated in the sigmoid and the rectum. Often hundreds of tumours are present.

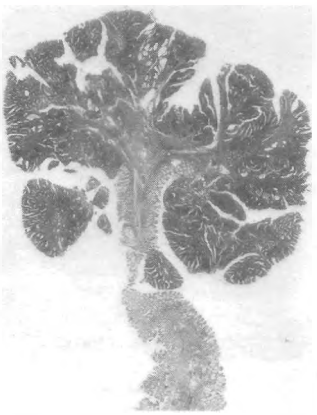


FIG. 1113.—Pedunculated adenomatous polyp of the large intestine. Longitudinal section. (*J. H. Saint, F.R.C.S., Santa Barbara, California.*)

FIG. 1114.—Adenomatous polyp of the colon. (*Max Pemberton, F.R.C.S., Enfield, Middlesex.*)

The patient complains of attacks of lower abdominal pain associated with loss of weight, diarrhœa, and tenesmus, and the passage of blood and mucus, and sometimes pus—all symptoms very like those of ulcerative colitis. A rectal examination may reveal one or more adenomatous polypi. Sigmoidoscopy shows a variety of neoplasms ranging from small sessile pink elevations to pedunculated tumours. A barium enema, especially a contrast barium enema (p. 1021), outlines the larger polypi.

Prevention.—(1) All members of the family should be examined at ten years. Repeat every two years. (2) Most of those who are going to get polypi will have them at twenty and these require operation. (3) If there are no polypi at twenty, continue with five-yearly examination till fifty. If there are still no polypi there is no inherited gene. Carcinomatous change before the age of twenty is very rare.

Treatment.—If operation is necessary, complete colectomy is advisable. A stump of rectum can remain and the terminal ileum anastomosed to it, the reasons being (*a*) the risk of carcinoma in the rectal stump is small if the patient is carefully followed-up and the polyps burnt off before they can develop malignant change; (*b*) an artificial anus is avoided; (*c*) other members of the family can be persuaded more easily to undergo the operation before late symptoms develop.

Hamartomatous Polyps.—Peutz-Jeghers polyps may occur in the colon (p. 1016) either as solitary or multiple lesions. The other type of hamartomatous polyp is the juvenile or mucous polyp (p. 1114) which may occur as multiple lesions of the colon, often associated with a congenital defect such as malrotation of the intestine, Meckel's diverticulum or congenital heart lesions. They have no malignant potential and are only removed if they produce troublesome pain, bleeding or hypoproteinæmia.

Hæmangioma.—A localised submucous telangiectasis is the cause of bleeding, which is often profuse. When the lesion is beyond the sigmoidoscopy field often the only method of detecting it is to operate while the bleeding is in progress. The distribution of blood within the intestine is noted; scrutiny of the blood-containing portion of the intestine usually reveals a dilated leash of veins in one portion of the mesocolon. At this site the intestine is opened and the tumour is resected. Arteriography is being used in many centres with great success (p. 995).

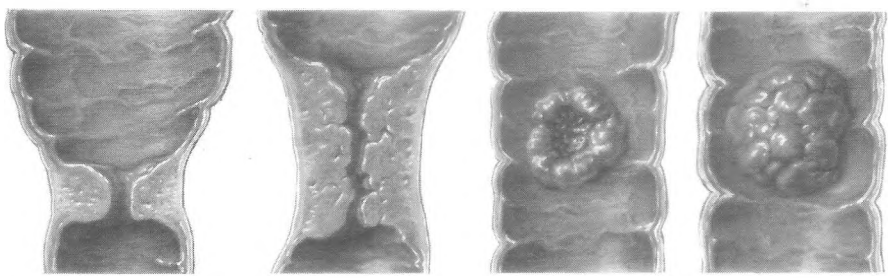
Lipoma is less frequently encountered in the large than in the small intestine, and is almost confined to the cæcum. The tumour is submucous and in more than half the cases it is the cause of an intussusception. Other symptoms to which it gives rise

are almost impossible to distinguish from those of a carcinoma; even macroscopical or microscopical blood is found in the stools. A barium study may suggest a lipoma because of its smooth contour.

Malignant

CARCINOMA OF THE COLON

Pathology.—Microscopically, the neoplasm is a columnar-celled carcinoma originating in the epithelial cells that line the colon, or in the crypts of Lieberkühn. Macroscopically the growth takes one of four forms (fig. 1115):



1. Annular. 2. Tubular. 3. Ulcer. 4. Cauliflower.

FIG. 1115.—The four macroscopical varieties of carcinoma of the colon.

Type 4 is the least malignant form, and this papilliferous carcinoma commences as a benign adenoma. The annular variety carries a relatively good prognosis, not because the growth is of low-grade malignancy, but because it gives rise to early obstructive symptoms, and therefore is often extirpated before metastases have occurred (Grey Turner).

Site.—The most frequent site is toward the termination of the colon, viz. the pelvic colon and the rectosigmoid junction (fig. 1116).

The Spread of Carcinoma of the Colon.—Generally speaking, it is a comparatively slowly growing neoplasm, and if extirpated thoroughly at a reasonably early period a cure can be hopefully anticipated.

Local Spread.—The growth is limited to the bowel for a considerable time. It spreads round the intestinal wall, and to a certain extent longitudinally, but usually it causes intestinal obstruction before it has penetrated adjacent structures. Particularly in the ulcerative variety, penetration of the serous coat is apt to occur and, according to the segment involved, adjacent structures then become invaded by the growth. When a hollow viscus is thus implicated an internal fistula results; also the perforation may lead to the formation of a local abscess and an external fæcal fistula.

Lymphatic Spread.—The lymph nodes draining the colon are grouped as follows:

1. *The epicolic lymph nodes*, situated in the immediate vicinity of the bowel wall.
2. *The paracolic lymph nodes*, lying in relationship to the leash of blood-vessels proceeding to the colonic walls.

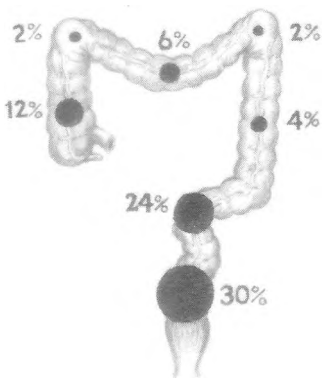


FIG. 1116.—Relative frequency of carcinoma in various portions of the large intestine omitting the rectum proper. (*Mayo Clinic statistics.*)

George Grey Turner, 1877–1951. Professor of Surgery, Post-Graduate Medical School, London.

3. *The intermediate lymph nodes*, arranged along the ileo-colic, right colic, mid colic, left colic, and the sigmoid arteries. In the last instance the paracolic lymph nodes are often absent.

4. *The main lymph nodes*, aggregated around the superior and inferior mesenteric vessels, where they take origin from the abdominal aorta.

Spread by the blood-stream occurs late. Metastases are carried to the liver.

Clinical Features.—Carcinoma of the colon usually occurs in patients over fifty years of age, but it is not rare earlier in adult life. Exceptionally it appears in childhood, when, owing to delayed diagnosis, prognosis is poor. Twenty-five per cent. of cases present as emergencies with intestinal obstruction or peritonitis. It must be remembered that in any case of colonic bleeding in patients over forty years of age, complete investigation of the colon is required.

Men are attacked more frequently than women (3: 2), although carcinoma of the ascending colon is encountered more often in women.

While certain outstanding symptoms frequently prevail in all types (fig. 1115) and at all sites (fig. 1116), it is instructive to contrast and to compare the symptoms produced by a carcinoma of the left side of the colon with that of the right, and to refer to the symptomatology of carcinoma of the sigmoid and that of the transverse colon.

Carcinoma of the Left Side of the Colon.—As will be seen on reference to fig. 1116, in about 75 per cent. of cases the neoplasm is situated on the left side of the colon. Neoplasms in this situation usually are of the stenosing variety (fig. 1115, 1 and 2) and as here the fæcal content is relatively solid, and the lumen of the bowel comparatively narrow, the main symptoms are those of increasing intestinal obstruction in about 25 per cent. of cases.

Symptoms	*Right Colon*	*Left Colon*	*Sigmoid*
Abdominal pain	78	68	51
Alteration of bowel habit	30	58	70
Loss of weight	50	15	20
Vomiting (frequently with colic)	32	15	0
Anorexia	18	9	0
Faintness, breathlessness	20	9	6
Bleeding per rectum	8	9	29
Lump present	67	46	39
Indigestion	8	0	0
Acute-on-chronic obstruction	8	21	29

The duration of symptoms is extremely variable; 5·5 months is the average.

(*E. G. Muir's statistics—figures given are percentages.*)

Pain.—This is usually intestinal colic. If it becomes a constant ache, it suggests inoperability or pericolitis.

Alteration of Bowel Habit.—An adult who has had regular bowel movement all his life, in a short space of time develops irregularity. The patient often states that he has *increasing* difficulty in getting the bowels to move, and that he has to take *increasing* doses of purgatives. Because of the drastic

Sir Edward Grainger Muir, 1906 1973. Surgeon, King's College Hospital, London. President, Royal College of Surgeons of England.

purgation, or on account of irritation by the scybala causing excessive secretion of mucus above the constricting neoplasm, attacks of constipation are followed by diarrhœa.

Palpable Lump.—Very often the lump that is felt on abdominal, rectal, or abdomino-rectal examination, is not the growth itself, but impacted fæces above it (Oldham). When the carcinoma is situated in a pendulous pelvic colon a hard movable swelling is often felt in the rectovesical pouch.

Distension.—Lower abdominal distension is not uncommon, and, like the pain, is relieved by passing flatus.

Carcinoma of the sigmoid follows the general pattern of the above, but there are differences.

Pain, when it occurs, is usually colicky from the commencement.

Tenesmus.—Growths of papilliferous type situated low in the colon are inclined to give rise to a feeling of the need for evacuation, which may result in tenesmus accompanied by the passage of mucus and blood, especially in the early morning.

Bladder symptoms are not unusual, and in some instances they herald colo-vesical fistula.

Carcinoma of the transverse colon may be mistaken for a carcinoma of the stomach because of the position of the tumour together with the anæmia and lassitude that it sometimes engenders.

Carcinoma of the cæcum and ascending colon presents in several guises:

(*a*) Anæmia, severe and unyielding to treatment, is a frequent predominating feature. Should a palpable tumour be present, the diagnosis is, to some extent, simplified.

(*b*) The presence of a mass in the right iliac fossa often proves a diagnostic conundrum (fig. 1108).

(*c*) Cæcal carcinoma sometimes is discovered unexpectedly at operation for acute appendicitis or for an appendix abscess that 'fails to resolve'. On rare occasions the appendix is inflamed, or even gangrenous, from obstruction to its lumen by the carcinoma.

(*d*) Less commonly a papilliferous growth is the apex of an intussusception. A lump, present at one time and smaller or absent at another (owing to partial reduction), associated with attacks of acute abdominal pain, is characteristic of this complication.

METHODS OF INVESTIGATION

Sigmoidoscopy should be performed in all cases where blood and mucus have been passed and also in suspected cases when a barium enema is negative, because early growths in the lower part of the pelvic colon are not always visualised by radiography.

Radiography after a barium enema often shows a carcinoma of the colon as a constant, irregular filling defect (fig. 1117). On the other hand, negative radiography in comparatively early cases of carcinoma of the colon is not by any means conclusive evidence of the absence of a growth. In 75 cases

James Bagot Oldham, 1899–1977. Surgeon, The United Liverpool Hospitals, Liverpool.

of carcinoma of the colon examined radiologically, no abnormality was detected in 8 per cent. This entailed considerable delay in diagnosis (Ramsay).

A tumour of the cæcum is more likely to be discovered by a barium meal than a barium enema. As a rule, in suspected cases of carcinoma of the colon, a barium meal is inadvisable because inspissated barium can precipitate intestinal obstruction if the lumen of the bowel is narrowed.

FIG. 1117.—Barium enema showing a carcinoma of the sigmoid colon. It may have an 'apple core' appearance.

Contrast Enema.—In cases of a neoplasm involving only the mucous membrane, a contrast enema is very valuable. The barium emulsion is partly evacuated and air is injected into the colon. By this means the walls of the colon become delineated and a neoplasm that fails to alter the contour of the barium-filled colon may be demonstrated.

Exfoliative cytology.—In experienced hands colonic exfoliative cytology is a valuable adjunct in the diagnosis of obscure cases of carcinoma of the colon. To obtain satisfactory results, the patient must be prepared carefully by a somewhat laborious procedure. After an interval of 5 to 10 minutes, the returned fluid is collected and centrifuged. Films from the sediment are prepared and stained. Successful diagnoses have been made by this method in growth situated in all parts of the colon.

Colonoscopy.—It has been shown already that this technique can supplement radiography in the diagnosis of both polyps and carcinoma of the colon. It can also help to prevent carcinomas arising by diagnosing and treating colonic polyps at an early stage (Williams). The technique is time consuming, and the apparatus is expensive.

TREATMENT

Pre-operative Treatment.—When there is no intestinal obstruction, blood transfusion to correct anæmia, if present, enemas to cleanse the bowel, a high caloric and low residue diet, together with succinylsulphathiazole, 2 g four-hourly for five days, are required. This sulphonamide gives watery stools so that the bowel mucosa is clean for purposes of anastomosis and it effectively reduces the number of virulent organisms ordinarily present in the colon. Metronidazole (Flagyl) also suppresses anærobic bacteria, and should be used in conjunction with the sulphonamide (200 mg orally twice daily for several days).

When intestinal obstruction is present, preliminary drainage of the intestine proximal to the obstruction must be performed (p. 1035).

Operation.—*The Test of Operability.*—The abdomen having been opened in the first place through a short paramedian incision, which is extended if the growth is removable: (1) the liver is palpated for secondary deposits, the presence of which is not necessarily a contraindication to resection, as the best palliative treatment for carcinoma of the colon is removal of the tumour; (2) the peritoneum, particularly the pelvic peritoneum, is inspected, if possible, and palpated for neoplastic implantations; (3) the various groups of lymph nodes that drain the involved segment are

Gordon Stuart Ramsay, Contemporary. Surgeon, Royal London Homœopathic Hospital, London.
Christopher Beverley Williams, Contemporary. Physician, St. Mark's Hospital for Diseases of Colon and Rectum, London.

palpated. Their enlargement does not necessarily mean that they are invaded by metastases, for the enlargement may be inflammatory; (4) the neoplasm is examined with a view to ascertaining if it is fixed or free, and if it is operable. " The whole colon above the last 7·5 cm of the pelvic portion is either mobile or can be mobilised " (Ogilvie).

The operations to be described are designed to remove the lymph nodes which may be involved by metastases from the particular primary growth concerned. Lesser resections are indicated, however, should hepatic metastases render the condition incurable.

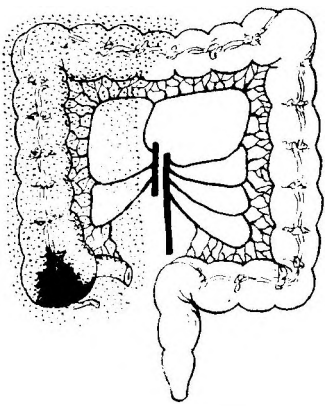


FIG. 1118.—Area to be resected when the growth is situated in the cæcum.

Carcinoma of the cæcum or the ascending colon is treated, when resectable, by right hemicolectomy (fig. 1118).

The abdomen is opened through a right paramedian incision. The technique of right colectomy should include at the outset ligatures placed around the bowel to prevent intraluminar spread. The peritoneum 2·5 cm or more lateral to the ascending colon is incised and the incision is carried around the hepatic flexure. The right colon is elevated, with the leaf of peritoneum containing its vessels and lymph nodes, from the posterior abdominal wall, care being taken not to injure the ureter, spermatic vessels, or the duodenum. The peritoneum is separated medially to near the origin of the ileo-colic artery, which is divided between ligatures, as also is the right colic artery when that vessel has a separate origin from the superior mesenteric. The mesentery of the last 30 cm. of ileum, and the leaf of raised peritoneum attached to the cæcum, ascending colon and hepatic flexure, after ligation of the blood-vessels contained therein, is divided as far as the proximal third of the transverse colon (fig. 1118). The surgeon, having verified that the blood supply to the proposed intestinal stumps is adequate, forthwith clamps and divides the ileum and the transverse colon at the levels of their respective severed mesenteries, and excises the free intestine. An end-to-end anastomosis between the ileum and transverse colon is then carried out and their respective mesenteries approximated with catgut. The abdomen is then closed.

Carcinoma of the Hepatic Flexure.—When the hepatic flexure is the seat of the neoplasm the resection must be extended correspondingly (fig. 1119).

Carcinoma of the Transverse Colon.—When there is no obstruction, excision of the transverse colon and the two flexures (fig. 1120), together with the transverse mesocolon and the greater omentum, followed by end-to-end anastomosis, is a satisfactory procedure.

Carcinoma of the Splenic Flexure or descending colon. The extent of the resection is shown in fig. 1121.

The phrenico-colic ligament is divided, and after incising the parietal peritoneum from the splenic flexure to the pelvic colon, the splenic flexure, descending colon, and pelvic colon are raised from the posterior abdominal wall in the same way as the ascending colon is raised on the right side. When this has been achieved, the left branch of the middle colic and the left colic arteries are ligated near their origins, and the peritoneal leaf with its contained lymph nodes is divided. Excision with end-to-end anastomosis is then carried out.

Sir Heneage Ogilvie, 1887–1971. Surgeon, Guy's Hospital, London.

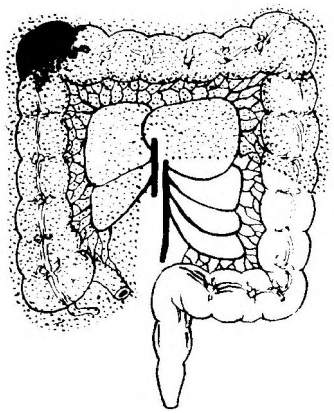


FIG 1119.—Area to be resected when the growth is situated at the hepatic flexure.

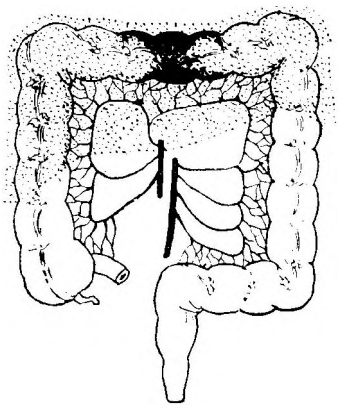


FIG. 1120.—Area to be resected when the neoplasm is situated in the transverse colon.

Carcinoma of the Pelvic Colon.—A left paramedian incision gives a good approach. The left half of the colon is mobilised completely (fig. 1122).

In order that the operation be rendered radical, the inferior mesenteric artery below its left colic branch, together with the related paracolic lymph nodes, must be included in the resection. This entails carrying the dissection as far as the upper

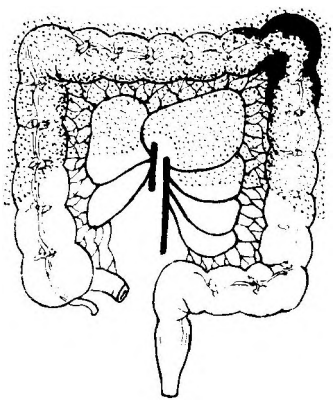


FIG. 1121.—Area to be resected when the neoplasm is situated at the splenic flexure, or the proximal part of the descending colon.

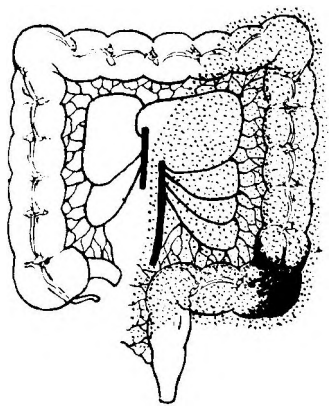


FIG. 1122.—Area to be resected in the case of a carcinoma of the pelvic colon.

third of the rectum, and accordingly the incision in the lateral leaf of peritoneum is extended downward to an appropriate level.

In every case of resection of the colon with anastomosis, drainage down to the site of anastomosis should be provided.

Modern methods of bowel preparation render one-stage resection and anastomosis reasonably safe in *non-obstructed* cases of colonic carcinoma but if any doubt exists a protective colostomy must be added. The methods of dealing with large bowel obstruction due to colonic carcinoma are described on p. 1037.

Post-operative Care following Colonic Resection.—Post-operative treatment includes the administration of antibiotics to guard against possible infection of the anastomotic area by *Cl. welchii*. Fluids are not given by mouth after anastomotic operations until flatus is passed.

When a Growth is found to be Inoperable.—In the upper part of the left colon, transverse colostomy is performed. In the pelvic colon, left iliac colostomy is preferable. With an inoperable growth in the ascending colon, ileo-colostomy is the best procedure. However, about 90 per cent. of colonic carcinomas can be resected, if necessary at a ' second look ' operation, when infection will have subsided following the by-passing of fæces. As these are slow-growing neoplasms, after subsequent colostomy-closure, these patients often enjoy several years of trouble-free life.

FÆCAL FISTULÆ

An external fistula communicating with the cæcum sometimes follows an operation for gangrenous appendicitis or the opening of an appendix abscess. A fæcal fistula can occur from necrosis of a gangrenous patch of intestine after the relief of a strangulated hernia, or from a leak after an intestinal anastomosis. The opening of an abscess connected with chronic diverticulitis or carcinoma of the colon frequently results in a fæcal fistula. Tuberculous peritonitis, ileo-cæcal actinomycosis, amœbiasis, and regional ileitis (in this instance nearly always following operation) are also causes of fæcal fistulæ, which may be multiple. About 4 per cent. of chronic fæcal fistulæ in hospital are kept open by patients pushing in knitting needles or other articles for reasons best known to themselves.

External fæcal fistulæ can be divided into three varieties: 1. A track lined by mucous membrane which protrudes above skin level. 2. A direct track lined by granulation tissue communicating with the exterior. 3. A long, tortuous track lined by fibrous tissue and partly epithelialised. The discharge from a fistula connected with the duodenum or jejunum is bile-stained and causes severe excoriation of the skin. When the ileum or cæcum are concerned, the discharge is fluid fæcal matter; when the distal colon is involved, it is solid or semi-solid fæcal matter. In some cases, when the leak from the small intestine or cæcum is small, it may be difficult to distinguish a fæcal discharge from fæculent pus. If methylene blue is administered by mouth and a fæcal leak is present, the blue colour will be distinguished easily in the discharge a few hours later. Often the site of the leak and the length of the fistula can be determined by radiography after a barium meal or barium enema. Should this fail to demonstrate the internal orifice, injection of lipiodol into the external opening will usually give the desired information.

Treatment.—Fæcal fistulæ, especially those in connection with the small intestine, tend to heal spontaneously, provided there is no obstruction beyond the fistulous opening. The abdominal wall must be protected from erosion by the use of a disposable ileostomy bag (p. 1007).

The higher the fistula in the intestinal canal the more skin excoriation must

William Henry Welch, 1850–1934. Professor of Pathology, Johns Hopkins University, Baltimore, Maryland, U.S.A.

be expected. This reaches its zenith in the case of a duodenal fistula (p. 858). A sump drain (p. 951) to remove the enzyme-laden discharge is a fundamental requirement. Such fistulæ cause dehydration and hypoproteinæmia and intravenous parenteral feeding will be required. If there is no prospect of rapid healing, a feeding jejunostomy is often a life-saving measure (p. 859).

Isogel or Pulv. cretæ aromat. 4 g q.i.d. will make the fæces more solid and avoidance of eggs and fish will help to reduce the malodour.

A fæcal fistula with mucosa visibly continuous with the skin edge will never close spontaneously. In some of these cases, where the opening is a large one, the intestine tends to prolapse upon the surface.

The operative treatment for closure of a fæcal fistula consists in making an incision encircling the fistula and dissecting up the tract through the abdominal wall and the peritoneum. The base of the fistula, now free, is crushed, ligated and oversewn. The abdominal wall is then closed in layers. In the case of a colo-cutaneous fistula connected with colonic diverticulitis, if the fistula fails to heal after a few weeks, resection of the affected segment is usually advisable (p. 1002). Should the mass from which the fistula arises prove to be an inoperable carcinoma, a defunctioning colostomy should be performed at a higher level. In complicated fistulæ, an operation to defunction the loop of intestine involved is probably the best procedure. When there is no obvious cause for the fistula, the discharge must be examined on several occasions for *actinomyces* or *mycobacterium tuberculosis*.

COLOSTOMY

A *colostomy* is an artificial opening made into the large bowel in order to divert fæces (and flatus) to the exterior, where they may be collected in an adhesive bag.

Depending on the purpose for which the diversion has been necessary, a colostomy may be *temporary* or *permanent*.

Temporary Colostomy

This is most commonly established to relieve a distal obstruction of the sigmoid colon either by a carcinoma or diverticulitis. Other indications include vesico-colic fistula, protection of a low colo-rectal anastomosis after anterior resection, to prevent fæcal peritonitis developing after traumatic injury to the rectum or colon, and to facilitate the operative treatment of a high fistula-in-ano.

A temporary colostomy is made by bringing a loop of bowel to the surface ('loop colostomy') where it is held in place by a glass rod passed through the mesentery. When firm adhesion of the colostomy to the abdominal wall has taken place after 7 days, the rod can be removed. As the colostomy has been established for obstruction (or other emergency situations) it is opened immediately at the end of the operation, and the edges of the colonic incision stitched to the surrounding skin margins (fig. 1123).

A loop of colon can be most easily brought to the surface by using large bowel that has a mesentery: most loop colostomies are therefore made in the transverse colon, and if the disease involves the left side of the colon, the right half of the transverse colon is preferred so that the entire left side is

left available for any subsequent formal operation, e.g. left hemicolectomy for a carcinoma of the sigmoid colon. The sigmoid colon is also suitable for a loop colostomy.

Following surgical cure of the distal lesion for which the temporary stoma was constructed, the colostomy is closed. Colostomy closure is most easily and safely accomplished when the stoma is ' mature ', i.e. after the colostomy has been established for two months. Closure is usually performed nowadays by an *intraperitoneal* technique, as this has been shown to be safer than the *extraperitoneal* methods, and to be accompanied by fewer closure breakdowns with fæcal fistulæ.

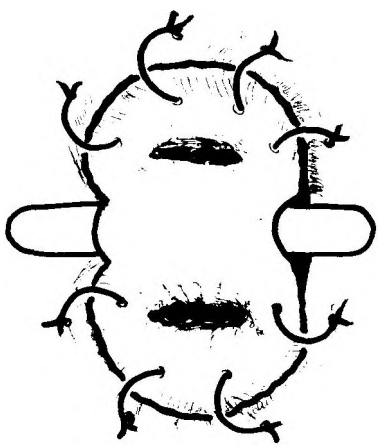


FIG. 1123.—Usual temporary loop colostomy opened over a rod, and immediate suture of colon wall to surrounding skin.

Double-barrelled Colostomy.—This colostomy was designed so that it could be closed by crushing the intervening ' spur ' by an enterotome. It is rarely used now, but occasionally the colon is divided so that both ends can be brought separately to the surface. This ensures that *absolute* rest is given to the distal segment of the colon and rectum (Devine).

Permanent Colostomy

This is usually formed after excision of the rectum for a carcinoma by the abdomino-perineal technique.

It is formed by bringing the distal end (' end colostomy ') of the divided colon to the surface in the left iliac fossa, where it is stitched in place immediately by sutures placed between the colonic margin and the surrounding skin.

The point at which the colon is brought to the surface must be carefully selected to allow a colostomy bag to be applied without impinging on the bony prominence of the anterior superior iliac spine. The best site is usually through the lateral edge of the rectus sheath 6 cm above and medial to the bony prominence (fig. 1124).

An important point after the colostomy has been made is to close the ' lateral space ' between the intraperitoneal segment of the sigmoid colon and the peritoneum of the pelvic wall to prevent internal herniation or strangulation of loops of small bowel through the deficiency.

Colostomy bags and appliances.—Fæces from a permanent colostomy are collected in disposable adhesive bags. A wide range of such bags is currently available. An increasingly popular type employs a Karaya-gum seal (washer) that does not irritate the skin and needs to be changed less often than the regular ' stick-on ' type (fig. 1125).

In large district hospitals, stoma therapists and stoma clinics are being set up to offer advice to patients on stoma care, and to acquaint patients with the latest services.

An abdominal belt is commonly worn, but usually a light elastic ' roll-on ' type rather than the heavy belts (St. Mark's pattern) previously advised.

Sir Hugh Berchmans Devine, 1878–1959. Surgeon, St. Vincent's Hospital, Melbourne, Australia.

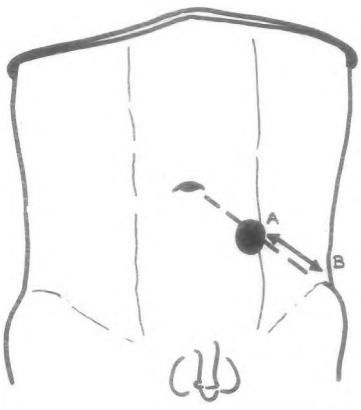


FIG. 1124.—Usual site of a permanent (end) colostomy in the left ilial fossa. Note distance A–B = 2·5 cm at least.

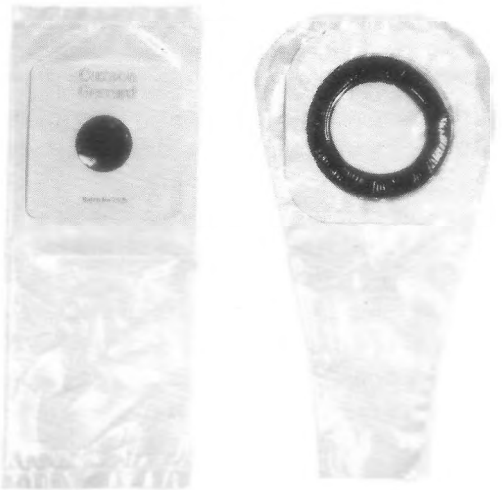


FIG. 1125.—The common *stick-on* bag and the alternative bag with a Karaya-gum seal (Hollister type).

Complications of Colostomies

The following complications can occur to *any* colostomy, but are more common after a badly performed technique:

1. *Prolapse.*
2. *Retraction.*
3. *Necrosis* of distal end.
4. *Stenosis* of the orifice.
5. *Colostomy hernia.*
6. *Bleeding* (usually from granulomas around the margin of the colostomy).
7. *Colostomy ' diarrhœa '* (this is usually an infective enteritis and responds rapidly to treatment by Neomycin 1·0 g every 8 hours).

Many of these complications necessitate that the colostomy should be reformed. Often this can only be done satisfactorily by re-opening the abdomen, freeing up the colostomy which is then re-established in a fresh spot.

CÆCOSTOMY

In desperately ill patients with advanced obstruction, if the lesion is situated in the right half of the colon, a cæcostomy may have to be performed. In late cases of obstruction, the cæcum may become so distended that rupture of the cæcal wall may be anticipated. This may occur spontaneously, giving rise to fæcal peritonitis or at operation when an incision in the abdominal wall reduces its supportive role and allows the viscus to expand. In such cases the over-distended viscus should be decompressed by suction as soon as the peritoneum is opened. The cæcum may then require partial mobilisation. In thin patients it may then be possible to carry out direct suture of the incised cæcal wall to the abdominal skin of the right iliac fossa. More commonly, the decompression is performed by introducing a wide bore-tube (No. 22 Foley or Winsbury-White catheter) which is stitched in place by invaginating purse-string stitches—so placed as to ' inkwell ' the site of entry.

A cæcostomy is only a short-term measure to allow a few days for the condition of the patient to improve. Re-operation should normally be carried out within 7 days of establishing the cæcostomy.

CHAPTER 50

INTESTINAL OBSTRUCTION

INTESTINAL obstruction is a common surgical emergency and because of its serious nature it demands early diagnosis and speedy relief. It may be classified into two types:

1. **Dynamic.**—Here there is increasing peristalsis working against an obstructing agent, which may be in the *lumen*, such as a bolus of incompletely digested material, inspissated fæces, or a gall stone; *in the wall*, such as an inflammatory or malignant stricture; or *outside the wall*, as in herniæ or adhesions.

2. **Adynamic** (p. 1052).—In this condition, peristalsis ceases and no true propulsive waves occur—as in paralytic ileus or mesenteric vascular occlusion.

DYNAMIC OBSTRUCTION

Is classified clinically into three types:

1. *Acute obstruction* confined to the small gut with central abdominal pain, early vomiting, central abdominal distension, and constipation.

2. *Chronic obstruction* confined to the large bowel with lower abdominal colic at first and absolute constipation and distension later.

3. *Acute-on-chronic obstruction* which spreads from the large bowel to involve the small intestine and gives early pain and constipation, followed by general distension and vomiting.

Pathology

At the outset, the intestine *above* the point of obstruction endeavours to overcome the obstruction by vigorous peristalsis. Increased peristalsis continues for a period of from forty-eight hours to several days; the more distal the point of obstruction, the longer does it remain vigorous. If the obstruction is not relieved, a time is reached when increasing distension causes peristalsis to become less and less; finally, peristalsis ceases, and the obstructed intestine becomes flaccid and paralysed.

For two or three hours following the obstruction, the intestine *below* the point of obstruction exhibits normal peristalsis, and absorption from it continues, until the residue of its contents has been passed onwards. Then the empty intestine becomes immobile, contracted, and pale, and so it remains, until the obstruction has been overcome, or death ensues.

Distension.—This occurs proximal to the obstruction, and begins immediately after the obstruction occurs. The cause of distension is two-fold:

(*a*) **Gas.**—This consists of swallowed atmospheric air (68 per cent.), diffusion from blood into the bowel lumen (22 per cent.), and the products of digestion and bacterial activity (10 per cent.). When the oxygen and carbon dioxide has been absorbed into

the blood-stream, the resultant mixture is made up of nitrogen (90 per cent.), and hydrogen sulphide.

(*b*) **Fluid.**—This is made up by the various digestive juices—about 8,000 ml. per twenty-four hours.

Above pylorus	4,000 ml.	Saliva 1,500 ml. Gastric 2,500 ml.
Below pylorus	4,000 ml.	Bile and pancreatic 1,000 ml. Succus entericus 3,000 ml.

In obstruction, absorption from the gut is retarded but excretion of water and electrolytes into the lumen persists and may even be increased. Deprivation of water and electrolytes, therefore, is due to:

(*a*) Vomiting.
(*b*) Defective absorption.
(*c*) Sequestration in lumen of bowel.

The severity of depletion and speed with which it becomes manifest is dependent upon the level of obstruction. It is most severe and occurs early in high small intestinal obstruction, later in ileal obstruction, and is slow to appear in colon obstruction. (For effects of fluid and electrolyte loss, see Chapter 8.)

Intestinal Toxins.—It is well known that release of intestinal obstruction may be followed by death, particularly in cases of strangulation. In unrelieved strangulation, toxic substances appear in the peritoneal cavity only when the viability of the bowel wall is affected. However, when obstruction is relieved, these toxins may pass on to the bowel where absorption can occur. It is probable that the substances involved are endotoxins of gram-negative bacilli. This factor stresses the need for intestinal decompression before and during operation.

Strangulation of the bowel occurs when it is trapped by a hernia or a band, or involved in a volvulus or intussusception in such a way that its *blood supply is progressively interfered with.* It is a very dangerous condition and demands early treatment. The first effect of strangulation is to compress the veins so as to cause the strangulated bowel, and its involved mesentery, to become blue and congested. Mesenteric vascular occlusion alone gives rise to gangrene without mechanical obstruction.

The Onset of Gangrene.—Much depends on the tightness of the constricting agent. When the venous return is completely occluded, the colour of the intestine turns from purple to black. About this time, in many instances, owing to increased œdema at the point of obstruction, the arterial supply is jeopardised. Then the peritoneal coat loses its glistening appearance, the mucous membrane becomes ulcerated, and moist gangrene is imminent.

Loss of blood volume into the congested segment is proportional to the length of that segment. When, as is often the case in strangulated external hernia, only a few inches are involved, the amount of blood thus imprisoned is inconsequential; on the other hand, when a large coil of intestine becomes strangulated the loss of blood is sufficient to render the patient oligæmic; when several feet of small intestine are involved the volume of circulating blood is so reduced as to imperil the patient's life.

Distension.—For a considerable time the strangulated segment (fig. 1126 (B)) alone distends, the greatest distension occurring when the venous return is completely obstructed while the arterial supply remains unimpaired. Unlike non-strangulating obstruction, early distension of the proximal intestine (fig. 1126 (A)) is absent; indeed, for a time varying from

a few minutes to several hours the proximal intestine contracts. After this varying interval, vigorous peristalsis occurs in the proximal segment, but is still unaccompanied by distension. By the time gangrene of the strangulated segment is imminent, retrograde thrombosis is proceeding along the related tributaries of the mesenteric vein. Distension then appears both on the proximal and distal sides of the strangulation (fig. 1126 (A) and (C)) (Chesterman).

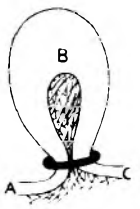


FIG. 1126.

Transmigration of Bacteria and Toxins.—When the wall of the intestine becomes partly devitalised, both bacterial toxins and the products of tissue autolysis pass into the peritoneal cavity, there to be absorbed into the circulation. This is followed by the migration of bacteria, and peritonitis follows. So it comes about that strangulation in an *external* hernia is far less dangerous than intraperitoneal strangulation, for in the former the transudate containing lethal toxins and bacteria is confined to the comparatively small absorptive area of the hernial sac.

FIG. 1127.—Carcinomatous stricture of the hepatic flexure: closed-loop obstruction.

Closed-loop obstruction is present in many cases of intestinal strangulation. In its typical form it is seen in carcinomatous stricture of the colon (fig. 1127). Distally the colon is occluded by the neoplasm, while in one-third of cases the ileo-cæcal valve prevents regurgitation of the contents of the large intestine into the ileum, and consequently that part of the colon proximal to the neoplasm is closed at both ends (fig. 1127). As a result of anti-peristalsis the pressure within the cæcum becomes so high as to compress the blood-vessels within its walls. If the obstruction is unrelieved, stercoral ulceration, gangrene, and perforation of the cæcum will eventually occur.

CLINICAL FEATURES OF ACUTE INTESTINAL OBSTRUCTION

There are four important symptoms and signs: pain, vomiting, distension, and constipation. These must be carefully looked for in each case.

Acute Obstruction

1. *Abdominal pain* is the first symptom : it commences suddenly, and often without warning. It becomes increasingly severe, then passes off gradually, only to return at intervals of a few minutes. These attacks of intestinal colic, which last from three to five minutes, spread all over the abdomen, but are localised mainly at the umbilicus. In between attacks the patient is often quite free from pain. Recurring attacks of severe abdominal pain are a leading feature of all varieties of acute intestinal obstruction, with the sole exception of paralytic ileus (p. 1052).

2. *Vomiting.*—When the jejunum is mechanically obstructed, vomiting occurs with the first and each succeeding attack of pain. In the much more common obstruction of the ileum the patient may vomit once, following which there is an interval of several hours during which time the attacks of pain

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occur without vomiting. Ultimately copious, forcible, oft-repeated vomiting sets in. As acute intestinal obstruction progresses, the character of the vomitus alters. Initially it contains partly digested food; next it consists entirely of mucoid fluid; thereafter the vomitus becomes yellow or green from regurgitation of bile; finally, it is fæculent.

3. *Distension.*—In early cases of obstruction of the small intestine abdominal distension is often slight, or even absent. Centrally placed distension is present in fully established cases of obstruction to the ileum. *Visible peristalsis* may be present (fig. 1128). In order to observe it the abdomen must be watched for several minutes. *Borborygmi* are sometimes loud enough to be heard by the unaided ear. More often auscultation is necessary. The sound of turbulent peristalsis coinciding with an attack of colic is valuable evidence of intestinal obstruction. In all cases of suspected intestinal obstruction it is essential to *examine the common hernial sites*. An irreducible external hernia may be present although the patient is entirely unaware of it.

FIG. 1128.—Visible peristalsis. Intestinal obstruction due to the strangulated right femoral hernia, to which the arrow points.

4. *Constipation.*—In complete intestinal obstruction, after the contents of the bowel below the obstruction have been evacuated, there is constipation, and usually neither fæces nor flatus is passed, i.e. *absolute* constipation. It should be noted that there may be a natural action of the bowels after the onset of the attack, and that it is commonplace for an enema to yield a small fæcal result.

The rule that constipation is present in intestinal obstruction does not apply in cases of Richter's hernia (p. 1136), gall stone obturation (p. 1050), mesenteric vascular occlusion (p. 1051), and intestinal obstruction associated with a pelvic abscess (p. 971).

Dehydration.—Repeated vomiting and also loss of absorptive power by the distended intestine leads to dehydration, and when the patient is first examined obvious signs of dehydration—a dry skin, a dry tongue, and sunken eyes—may be present. The output of urine is small; it is concentrated, and contains little chloride.

Chronic Obstruction.—This is commonly due to carcinoma or diverticulitis of the colon. Here *constipation* appears first, may last for days or weeks and finally becomes absolute when the passage of flatus ceases. Abdominal *distension* then occurs, especially in the flanks. No matter where the obstruction lies, whether in the ascending, transverse, or descending portions of the colon or even in the rectum, the brunt of the obstruction is borne by the cæcum (fig. 1129). This has been likened to a gun back-

firing into its breech, and the cæcum becomes ballooned. *Pain* accompanies the distension and there are regular bouts of colic, usually in the hypogastrium. Soon after the onset of intestinal colic, if the abdomen is watched, the cæcum sometimes can be seen to distend with each attack of colic, and subside as the colic passes off. It is not uncommon for *vomiting* to be delayed for two or three days, and on this account signs of dehydration are exceptional. On abdominal palpation, a neoplasm occasionally can be felt in the line of the colon; but more usually the relatively small lump occasioned by a cicatrising neoplasm is obscured by the distension. A rectal examination will enable a carcinoma of the rectum, or a mass of impacted fæces, to be felt, and it may be possible to feel a neoplasm in the pelvic colon occupying the recto-vesical pouch. An enema is usually given but, in the presence of absolute obstruction, is returned without force, fæces, or flatus.

FIG. 1129.—Wherever the large intestine is obstructed the cæcum bears the brunt.

Acute-on-Chronic Obstruction.—This starts with all the features of chronic obstruction but after a few days the pain, vomiting, and central distension betoken the fact that the ileo-cæcal valve is open and fæculent fluid has passed up into the ileum from which it is rapidly vomited.

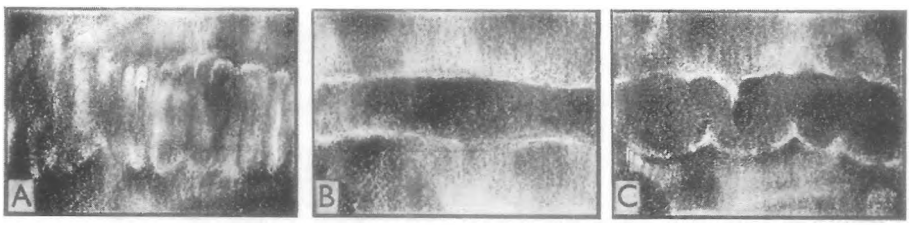
Strangulation.—It is of the highest importance to distinguish strangulating from non-strangulating intestinal obstruction, because if the former is not relieved by an urgent operation, gangrene follows quickly. The diagnosis is made entirely by clinical methods; the picture is usually that of an obstruction together with a degree of shock which is sometimes severe. In some instances the differentiation is not difficult, for when the strangulation is a tight one, unlike non-strangulating obstruction, pain is never completely absent. In strangulation, the symptoms usually commence very suddenly and spasms of intestinal colic recur three or four times a minute. Generalised tenderness and sometimes rigidity are indicative that an early laparotomy should be performed. Much more difficulty is encountered when the strangulation commences as a mild venous occlusion. In such circumstances the presence and character of local tenderness is of great significance, and frequently two or more clinical examinations at half-hourly intervals are required to collect sufficient data to make a confident diagnosis of strangulation. In non-strangulating obstruction there may be an area of localised tenderness at the site of obstruction; in strangulation there is always tenderness over an intra-abdominal strangulated coil and the pain is made worse if the palpating hand is suddenly removed. *This rebound tenderness is a distinctive sign of strangulation.* Cases of intestinal obstruction in which pain persists in spite of one, or at the most two, hours of treatment by gastro-duodenal aspiration, even in the absence of any of the above signs, should be diagnosed as intestinal strangulation. Where the strangulation occurs in an external hernia the lump is *tense*, *tender*, *irreducible*, there is *no expansile cough impulse* and it has *recently increased in size*. These points are most valuable in diagnosis.

X-RAY DIAGNOSIS

In all cases, ' scout ' *films* of the abdomen, with the patient standing and lying down, must be taken in the X-ray department as soon as possible. Ideally, this should be preceded by an enema.

Gas Shadows.—When the jejunum, the ileum, or the colon is distended with gas, each has a characteristic appearance that allows it to be distinguished radiologically. The diameter of the viscus is no criterion as to whether it is small or large intestine. Obstructed small intestine is revealed by relatively straight segments that generally lie more or less transversely; obstructed large intestine is disclosed by its haustration markings; a distended cæcum is shown by a rounded gas shadow.

Jejunum is characterised by its valvulæ conniventes that pass from the antimesenteric to the mesenteric border, spaced regularly, giving rise to a concertina effect (fig. 1130 (*a*)).



(*a*) Jejunum (*b*) Ileum (*c*) Colon

FIG. 1130.—Gas shadows characteristic of various parts of the intestine.

Ileum.—The distal ileum is piquantly described by Wangensteen as being ' characterless ' (fig. 1130 (*b*)).

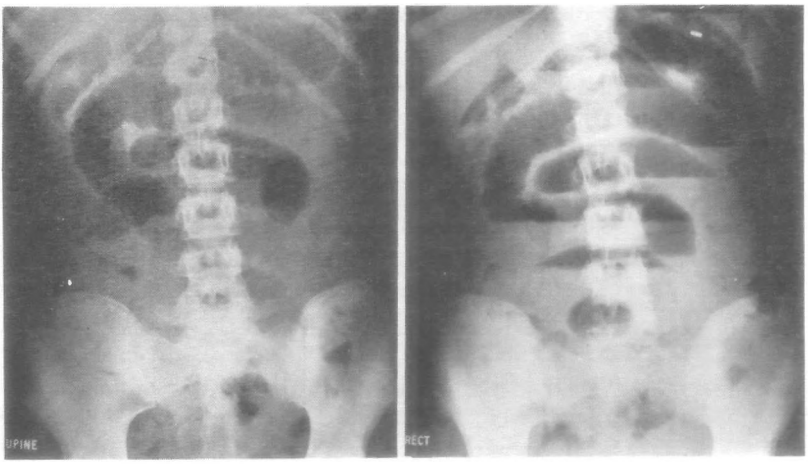
Large intestine (the cæcum excepted) shows haustral folds. Haustral folds, unlike the valvulæ conniventes, are spaced irregularly and the indentations are not placed opposite one another (fig. 1130 (*c*)).

Fluid Levels.—In infants under the age of two years a few fluid levels in the small intestine are a normal occurrence. In adults, two inconstant fluid levels must be regarded as physiological. One is at the duodenal cap; the other, which is more infrequent, is within the terminal ileum. In intestinal obstruction it takes a little time for the gas to separate from the fluid; consequently fluid levels appear later than gas shadows. When paralysis of the intestine has occurred, fluid levels become more conspicuous and more numerous. By the time fluid levels are pronounced, obstruction is advanced. The number of fluid levels is proportionate to the degree of obstruction and to its site in the small intestine; the nearer the obstruction is to the ileo-cæcal valve, the larger the number of fluid levels (fig. 1131). Obstruction *low* in the colon does not commonly give rise to fluid levels in the small intestine, but in the case of obstruction *high* in the large intestine, this phenomenon is not unusual, because in many individuals the ileo-cæcal valve is incompetent.

In obstruction of the large intestine, a plain radiograph always shows a

Owen Harding Wangensteen, Contemporary. Emeritus Professor of Surgery, University of Minnesota, Minneapolis, U.S.A.

large amount of gas in the cæcum. In most cases a barium enema is contra-indicated during an attack of intestinal obstruction.



(*a*) (*b*)

FIG. 1131.—(*a*) Gas filled small bowel loops; patient supine. (*b*) Fluid levels with gas above; 'step-ladder' pattern. Small bowel obstruction by adhesions; patient erect.

Multiple gas-filled loops with fluid levels above in both small and large bowel are seen in established paralytic ileus. Here, however, bowel sounds are absent or much reduced.

TREATMENT OF ACUTE INTESTINAL OBSTRUCTION

There are three measures for combating and overcoming the effects of intestinal obstruction. They are: (1) gastro-duodenal or, when possible, gastro-intestinal suction drainage; (2) replacement and maintenance of fluid and electrolytic balance; (3) relief of the obstruction, usually by operation.

The first two are always necessary preliminaries to the relief of obstruction by operation, and they are also the mainstays of post-operative treatment. In some cases, as will be shown, they are used exclusively.

In every case of acute intestinal obstruction the first step is to empty the stomach by a transnasal aspirating tube and to keep the stomach empty by withdrawing the contents with a syringe or by continuous suction. The second step is to correct the fluid and electrolytic balance (Chapter 8). When, on clinical examination, the cause of the obstruction is not obvious, radiographs of the abdomen are taken and the clinical and radiographic data are correlated. The main indications for early operation (as soon as the fluid and electrolytic depletion has been corrected) are:

1. Obstructed or strangulated external hernia (Chapter 54).
2. Internal intestinal strangulation.
3. Acute or acute-on-chronic obstruction.

The most urgent of these is intestinal strangulation. Gastro-duodenal aspiration should be continued throughout the operation, and also in most instances the intravenous infusion, which, in cases of strangulation, should be supplemented by blood transfusion.

Relief of Obstruction by Operation.—When the cause of the obstruction lies within the abdomen but its site is doubtful, a right lower paramedian incision is employed.

When the Obstruction lies in the Small Intestine.—The hand is passed to the cæcum. In obstruction of the small intestine the cæcum is collapsed. The site of obstruction may be obscured by dilated coils of intestine, in which event an unobstructed contracted coil of ileum is sought and followed upwards. This will guide the fingers to the site of obstruction which, if deeply placed, is exposed by displacing distended coils away from the site with warm, moist abdominal packs. Occasionally it is necessary to withdraw several coils of distended intestine before the site of obstruction can be displayed satisfactorily. Eviscerated coils must be kept covered by abdominal packs. The obstruction is relieved by one of the various methods described under special forms of intestinal obstruction.

Emptying the Small Intestine at Operation.—If there is much distension, it is wise to deflate the bowel using Savage's decompressor. The tube is introduced into the lumen through a stab-wound in the bowel at its most distended part. The bowel is progressively emptied by threading it over the instrument for its full length (fig. 1132). The stab-wound is closed in two layers transversely. If this, or a similar instrument, is not available, an ordinary sucker or a rubber catheter attached to a sucker is an adequate substitute. By these means closure of the abdominal wall is facilitated and intestinal toxins mostly eliminated.

FIG. 1132.—Savage's decompressor introduced into the small intestine which is plicated over it.

Measures to be taken when the Small Intestine is Strangulated.—If, as is frequently the case in intra-abdominal strangulation, blood-stained fluid is present in the peritoneal cavity, the fluid should be removed by suction or mopped up as completely as possible, for it is toxic and infected. After the relief of strangulation a decision must be reached as to whether the segment that was strangulated is viable. When it is black and the peritoneal coat has lost its sheen, when the mesentery shows a lack of arterial pulsation, or thrombosis of its veins, it is non-viable, if not already gangrenous, and, if

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practicable, resection followed by anastomosis is carried out. In doubtful cases when the intestine is blue, purple, or dark red, the effect of wrapping it in a warm moist abdominal pack is noted. At the same time the anæsthetist administers pure oxygen for three minutes. By these means viable is differentiated from non-viable intestine thus:

Intestine	*Viable*	*Non-viable*
Circulation	Dark colour becomes lighter; mesentery bleeds, if pricked.	Dark colour remains; no bleeding if mesentery is pricked.
Peritoneum	Shiny.	Dull and lustreless.
Intestinal musculature	Firm. Pressure rings may or may not disappear. Peristalsis may be observed.	Flabby, thin, and friable. Pressure rings persist. No peristalsis.

Special attention should always be paid to the sites of constriction (' pressure rings ') at each end of the segment, which if of doubtful viability, should be enfolded by passing sutures through the sero-muscular coats and covering the area with omentum.

Pressure rings having received attention, viable intestine is returned to the abdominal cavity and the laparotomy incision is closed. When the strangulated intestine is deemed non-viable, it is resected and the continuity of the alimentary canal restored by end-to-end anastomosis.

When the obstruction occurs in the large intestine it is usually due to a carcinoma, or occasionally, in the case of the pelvic colon, to its imitator, diverticulitis, and the obstruction is of the acute-on-chronic variety. Acute-on-chronic obstruction of the large intestine should always be treated by early operation. If the patient's condition is good, laparotomy is performed through a right or left paramedian incision, according to the site of the obstruction. If the site is unknown, a right lower paramedian incision is employed. Distension of the cæcum at once confirms that the obstruction lies in the large intestine. Palpation of the pelvic colon and, if that be collapsed, the transverse colon, will readily lead to the obstruction. When removable obstruction is present in the cæcum, ascending colon, at the hepatic flexure, or in the proximal part of the transverse colon, emergency *right hemicolectomy* (p. 1023) is the correct procedure. If the growth is irremovable or the patient extremely ill, an ileo-transverse enterostomy will relieve the obstruction. If the obstruction lies at the splenic

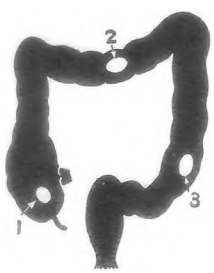

FIG. 1133.—The sites for an artificial anus in acute-on-chronic intestinal obstruction. 1. Cæcostomy. 2. Transverse colostomy. 3. Left iliac colostomy.

flexure, in the descending colon, the pelvic colon, or in the rectum, *transverse colostomy* is performed (fig. 1133). The obstructing growth is removed at a second operation and the colostomy closed.

In very old or enfeebled patients when a carcinoma of the rectum is fixed and probably irremovable, left iliac colostomy is the best site for a permanent artificial anus.

By placing the colostomy some inches proximal to the growth and excising it together with the growth at a second-stage operation, the number of operations and the length of hospitalisation can be reduced.

Blind cæcostomy should be avoided, except in desperate cases. Modern methods of fluid replacement and anæsthesia render an expeditious laparotomy as safe as the blind procedure.

GASTRO-INTESTINAL SUCTION DRAINAGE

When intestinal strangulation can be ruled out and the obstruction lies in the small intestine, a period of a few hours' gastro-intestinal suction drainage is the best form of preliminary treatment.[1] Suction drainage should also be employed almost exclusively in paralytic ileus. In mechanical obstruction the combined effects of relieving distension by suction and the administration of fluid intravenously greatly improve the general condition of the patient for operation. Locally, diminution in size of the distended coils of intestine facilitates the operation and closure of the wound. Another most important consideration is that if highly toxic intestinal contents are aspirated before operation, it spares the patient the danger of absorbing this material after the obstruction has been relieved.

Maintenance of Fluid and Electrolytic Balance.—The fluid and the salt loss must be restored. Commonly as much as 3·5 litres of normal saline solution are required if the patient shows signs of severe dehydration, but considerably less if such signs are absent. A balance chart must be compiled, and the daily needs of the patient adjusted (p. 98).

ACUTE INTESTINAL OBSTRUCTION OF THE NEWBORN

Congenital atresia[2] **and stenosis**[3] are the most common causes of intestinal obstruction in the newborn. The site of the obstruction is as follows:

Duodenum	33 per cent.
Jejunum	15 „
Ileum	25 „
Ascending colon	10 „
Multiple sites	17 „

The high incidence of multiple sites makes it imperative to examine the whole of the small intestine and the ascending colon at operation. Congenital abnormalities of the heart and great vessels are frequently associated with these lesions; only atresia below the duodenum escapes this association. Ileal atresia is commonly due to a vascular accident *in utero*.

Atresia and Stenosis of the Duodenum.—Atresia and stenosis occur with about equal frequency. Unless the obstruction is partial, persistent vomiting occurs from birth. The vomitus may or may not be bile-stained, depending on whether the septum lies below or above the duodenal papilla. Distension is often absent, but visible peristalsis is sometimes seen passing from left to right.

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[1] Even when strangulation can be ruled out, not more than six or eight hours should be expended in this form of treatment. If operation is delayed over twenty-four hours, in spite of suction drainage, the mortality is nearly doubled.

[2] Atresia = imperforation. [3] Stenosis = narrowing.

FIG. 1134.—Duodenal atresia. 'Double-stomach' appearance on erect film. Gas and fluid levels in both stomach and duodenum.

Radiography.—A plain film often confirms the diagnosis. The stomach and upper part of the duodenum are grossly distended with air, the so-called 'double stomach' (fig. 1134).

In cases of partial obstruction a small amount of gastrografin injected through a gastric aspiration tube is useful for demonstrating the location of the obstruction; as soon as the films have been exposed, the medium must be aspirated.

Differential Diagnosis.—Suprapapillary duodenal atresia is distinguished from œsophageal atresia (p. 791) by the fact that there is no dribbling of saliva. The absence of a palpable lump serves to differentiate duodenal obstruction from infantile pyloric stenosis (p. 817). Duodenal obstruction in infancy can also be caused by volvulus of the midgut (p. 1040), obstruction due to a band (p. 1047), or to an annular pancreas (p. 938).

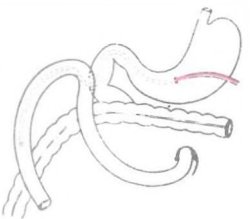

FIG. 1135.—Duodeno-jejunostomy with gastrostomy for feeding below the anastomosis. *(After the late G. H. McNab, F.R.C.S., London.)*

Treatment.—Operation should be carried out as soon as dehydration has been combated. Duodeno-jejunostomy (fig. 1135) is the operation of choice. After completing the anastomosis, a stab incision is made through the wall of the pyloric antrum. Through this incision a rubber catheter is passed, and its tip guided through the anastomosis. A Witzel's valvular opening is constructed in the wall of the stomach and the tube is brought out through the laparotomy incision.

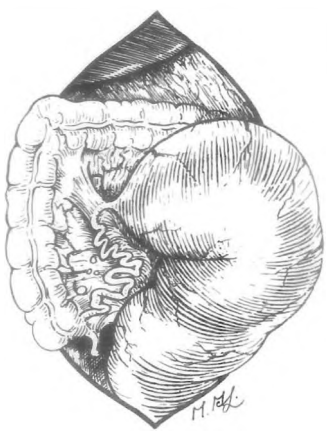

FIG. 1136.—Congenital atresia of the ileum. *(V. Swain, F.R.C.S., London.)*

Atresia or Stenosis of the Ileum (less frequently of the jejunum).—The overriding importance of very early diagnosis of this condition lies in the fact that the distension immediately above the septum soon becomes so great that the vascular supply of the intestinal wall becomes jeopardised, and gangrene and perforation result. In atresia of the ileum (fig. 1136) the infant is either born with central distension of the abdomen, or distension appears within twenty-four hours of birth. In jejunal atresia early distension is lacking, but vomiting soon occurs. In both conditions some meconium is likely to be evacuated.

FIG. 1137.—Plain radiograph in a case of congenital atresia of the ileum. *(Professor J. H. Louw, Cape Town.)*

Radiography.—Obstruction of the small intestine cannot be diagnosed by plain X-rays unless fluid levels are seen in addition to the distended coils (fig. 1137). By the time fluid levels are present, the obstruction is in an advanced state.

Treatment.—In the case of the ileum a satisfactory procedure is a Paul-Mikulicz anastomosis (fig. 1138). After the abdomen has been closed, and the wound protected with a dressing, a small Paul's tube is tied into the proximal and a small catheter into the distal limb of the spur. Four days later the tubes are removed and a crushing clamp is applied to the septum. When the stenosis lies in the jejunum, so devastating are the effects of even a temporary jejunal fistula that primary end-to-end anastomosis (fig. 1139), although a more difficult operation, must be recommended.

Friedrich Oskar Witzel, 1856–1925. Surgeon, Bonn, Germany.
Frank Thomas Paul, 1851–1941. Surgeon, Liverpool Royal Infirmary, Liverpool, England.
Johann von Mikulicz-Radecki, 1850–1905. Professor of Surgery, Breslau, Germany (now Wroclaw, Poland).

Arrested Rotation.—The cæcum remains in the left hypochondrium, and a peritoneal band is found running from the cæcum to the right side of the abdomen, and then across the second part of the duodenum. This is the transduodenal band of Ladd (fig. 1140). The symptoms (repeated vomiting), due to pressure on the duodenum, and the radiographic appearances are identical with those of duodenal stenosis.

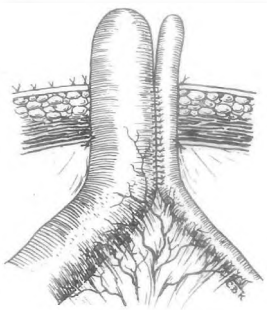

FIG. 1138.—Spur enterostomy for ileal atresia. (*After the late G. H. McNab, F.R.C.S., London.*)

Treatment.—Early laparotomy is essential. The pressure on the duodenum can be relieved immediately by dividing the attachment of the band near the parietal peritoneum. Often there is a second peritoneal band, extending from the middle line to the commencement of the jejunum; this also must be divided. The cæcum and colon are brushed over to the *left* side of the abdomen, leaving the small bowel on the right side (Nixon). The abdomen is closed. The results of timely operation in these cases are excellent.

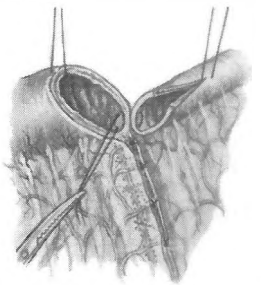

FIG. 1139.—End-to-end anastomosis. (*After the late Sir Denis Browne, F.R.C.S., London.*)

Volvulus of the Midgut (*syn.* Volvulus Neonatorum).—Arrested rotation described above, predisposes to volvulus of the midgut. The floating cæcum, together with the whole of the small intestine, which has a narrow attachment, revolves. Broadly speaking, the clinical features are similar to, and the radiological findings are identical with, those of arrested rotation which, indeed, is present. The onset, however, is more catastrophic, and dehydration occurs more rapidly than in arrested rotation *per se*. In addition, abdominal distension is often evident.

Treatment.—When the abdomen is opened, only distended coils of small intestine (which may or may not be cyanotic) and the stomach are seen. The whole of the mid-gut must be delivered on to the surface, where the intestine is protected with warm, moist abdominal packs. Only after this step has been taken is it possible to see the volvulus (fig. 1141) which usually takes place in a clockwise direction. Untwisting is only half the operation; of equal importance is to divide the second obstructive lesion—the transduodenal band of Ladd—which is often present.

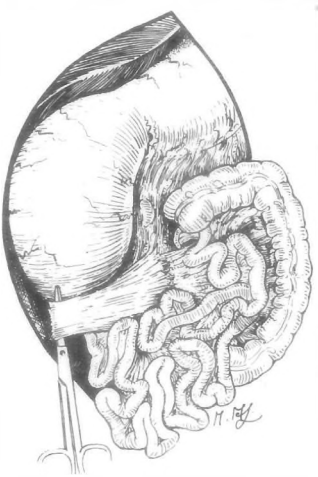

FIG. 1140.—Incomplete rotation of the cæcum and Ladd's transduodenal band compressing the duodenum. (*V. Swain, F.R.C.S., London.*)

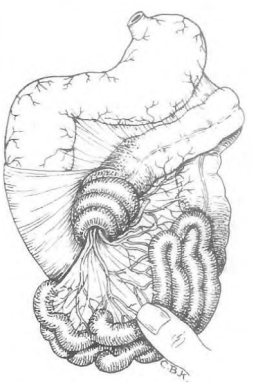

FIG. 1141.—Volvulus of the midgut.

Meconium ileus is the neonatal manifestation of mucoviscidosis (fibrocystic disease of the pancreas, p. 939). The terminal ileum becomes filled with meconium mixed with viscid mucus, notably from the pancreas, and during the latter months of fœtal life this mixture becomes progressively inspissated (fig. 1142). The infant is born with intestinal obstruction. At times the coil filled with inspissated meconium can be felt as a rubbery mass. A typical radiograph shows distended small intestine, some of which is mottled. Unlike ileal atresia, there is no abrupt termination of the gas-filled intestine.

Pathognomonic Test.—Into a bowl of vomitus is placed a piece of exposed X-ray

William Edwards Ladd, 1880–1967. Professor of Child Surgery, Harvard University, Boston, Massachusetts, U.S.A.
Harold Homewood Nixon, Contemporary. Surgeon, Hospital for Sick Children, Great Ormond Street, London.

film, and there it is left for half an hour. If trypsin is present, the gelatine that constitutes the sensitised coat of the film will be digested. In meconium ileus the film is merely rendered a little soft.

40% of cases are associated with complications, either volvulus neonatorum, atresia or meconium peritonitis (Dickson). When the physical signs of an acute intra-abdominal process are present, laparotomy must be carried out. When this is not so, however, a Gastrografin or Hypaque enema should be given to make the diagnosis. Since the colon is much reduced in size the radio-opaque fluid passes easily upwards to the ileum, and may on occasion disperse the obstructing meconium and relieve the condition.

Treatment.—For laparotomy, adequate pre-operative preparation is essential. The only condition with which meconium ileus can be confounded is Hirschsprung's disease (p. 991) affecting the whole colon, but in the latter condition (*b*) and (*c*) of fig. 1142 are lacking. The method of loosening the tenacious meconium from the mucous membrane by injections of 1% hydrogen peroxide through the bowel wall is not as satisfactory as believed previously. Recourse has to be made to a definitive resection of the most dilated segment. The proximal ileum is anastomosed end-to-side of collapsed colon beyond the obstruction (the Bishop-Koop operation). The distal ileal opening is formed into an ileostomy, through which the thick meconium can be eased postoperatively, assisted by irrigation of suitable lubricant solutions. The ileostomy becomes a mucous fistula which may need to be closed later.

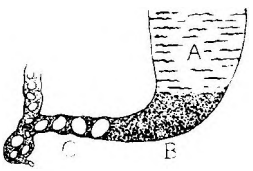

FIG. 1142.—Operative findings in meconium ileus: (*a*) Treacly fluid; (*b*) Putty-like material; (*c*) Meconium pellets.

Post-operative Complications.—In the section dealing with fibrocystic disease of the pancreas (p. 939), attention has been directed to the susceptibility of infants with mucoviscidosis to pulmonary complications. Oxygen and antibiotic therapy, in addition to the usual post-operative regimen for intestinal obstruction, are given.

ACUTE INTUSSUSCEPTION

One portion of the gut becomes invaginated into another immediately adjacent; almost always it is the proximal into the distal. Very rarely indeed is an intussusception retrograde.

Ætiology.—In a few cases there is some obvious cause, e.g. a polypus,

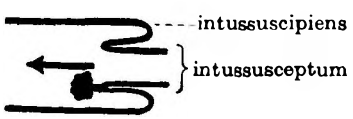

FIG. 1143.—The mechanism of the production of an intussusception.

a papilliferous carcinoma, a submucous lipoma, or an inverted Meckel's diverticulum (p. 997). Obviously such a protrusion invites intussusception (fig. 1143). In intussusception of infants it is generally agreed that:

1. Idiopathic intussusception occurs most often between the sixth and ninth months.
2. There is a change in diet—the infant is weaned.
3. An idiopathic intussusception usually commences in some part of the last 50 cm. of the small intestine.
4. The maximum aggregation of Peyer's patches is in the lower ileum.

Theory—1. Change of diet brings about a change of intestinal flora. 2. This predisposes to inflammation of the intestinal tract. 3. Which in turn causes inflammation and swelling of Peyer's patches. 4. A swollen Peyer's patch produces an elevation protruding into the lumen of the gut comparable to one of the known causes of intussusception.

Another theory:—Intussusception often shows a seasonal incidence related to attacks of upper respiratory infection. After an operation for intussusception antibodies to

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Harry Craden Bishop, Contemporary. Surgeon, Philadelphia, U.S.A.
Chas. Everett Koop, Contemporary. Surgeon, Philadelphia, U.S.A.
Johann Conrad Peyer, 1653–1712. Professor of Logic, Rhetoric, and Medicine, Schaffhausen, Switzerland.

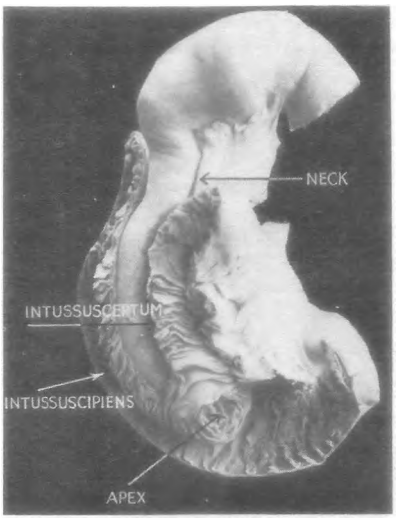


FIG. 1144. — An intussusception dissected to show its constituent parts.

certain viruses have been isolated and it is presumed that these cause swelling of Peyer's patches.

Pathology.—An intussusception is composed of three parts:

1. The entering, or inner, tube.
2. The returning, or middle, tube
3. The sheath, or outer, tube.

The outer tube is called the *intussuscipiens*, The inner and middle tubes together form the *intussusceptum*. The neck is the junction of the entering layer with the mass. That part which advances is the apex, and the mass which constitutes the intussusception (fig. 1144) increases as it advances.

The blood supply of the inner layers of the intussusception is liable to be impaired. The onset of early gangrene is dependent upon the tightness of the invagination. Because of the great pressure exerted upon it by passing through the ileo-cæcal valve, an ileo-colic intussusception provides most examples of early gangrene.

Types of Intussusception (After Gross) (In 702 cases)

	% of Series		% of Series
Ileo-ileal	5	Multiple	1
Ileo-colic	77	Retrograde	0·2
Ileo-ileo-colic	12	Others	2·8
Colo-colic	2		

A definite cause was found at operation in only 43 cases

Clinical Features of Intussusception in Infants.—Usually the patient is a fine, lusty male child between six and nine months of age. The onset is sudden. The child has a paroxysm of abdominal pain, draws up his legs, and screams. He may vomit shortly after the onset of the attack, but this is not a constant occurrence; not until the intussusception has been present for about twenty-four hours is vomiting a conspicuous feature. The attacks, which last a few minutes and recur about every fifteen minutes, are accompanied by *facial pallor*. They become progressively more severe. In between the attacks he lies listless and looks somewhat drawn. In the early stages a normal stool is frequently passed; later blood and mucus are evacuated—the well-known ' red-currant jelly ' stool.

On Examination.—The abdomen is not distended. It should be palpated, between the spasms, with a warm hand slipped beneath the bedclothes, care being taken not to arouse the dozing infant. Typically a lump is felt which may harden on palpation (fig. 1145), but if the lump is lying under the right or left costal margins, it may not be possible to feel it, even under an anæsthetic. There is said to be a feeling of emptiness in the right iliac fossa. On rectal examination, if the intussusception has travelled far enough, its apex, a conical mass which is aptly likened to the cervix uteri, will be felt,

Robert Edward Gross, Contemporary. Ladd Professor of Children's Surgery, Harvard University Medical School, Boston, Massachusetts, U.S.A.

especially on bimanual examination. In the large majority of cases the intussusception has not advanced far enough for the apex to be felt per rectum, but in most cases blood-stained mucus will be found on the examining finger.

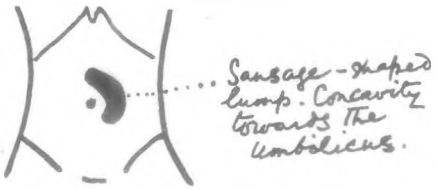


FIG. 1145.—The physical signs recorded in a typical case of intussusception in an infant.

In a few instances the intussusception actually protrudes through the anus. This does not necessarily imply that the intussusception is of long standing, but rather that the patient is possessed of a long mesentery, rendering the small intestine unduly mobile.

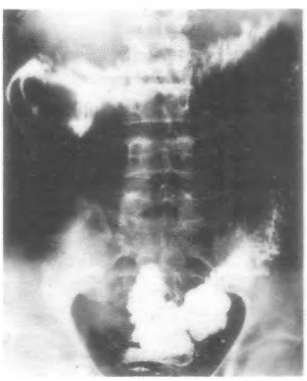


FIG. 1146.—'Claw' sign of ileo-colic intussusception. The barium in the intussuscipiens is seen as a claw around the negative shadow of the intussusception. (*R. S. Naik, M.S., Durg, India.*)

Unrelieved, the pain becomes continuous. After twenty-four to thirty-six hours the abdomen commences to distend, and vomiting becomes copious. Absolute intestinal obstruction follows, and death from this cause, or from peritonitis secondary to the gangrene, is probable. Once in a while a natural cure occurs due to sloughing of the intussusceptum.

Radiography.—A plain film usually reveals increased gas shadows in the small intestine, and at times an absence of the cæcal gas shadow. A barium enema (fig. 1146) gives positive evidence of the presence of an ileo-colic intussusception ('claw' sign), but if the intussusception is ileo-ileal, and the ileo-cæcal valve is competent, this form of examination is negative.

Differential Diagnosis

1. *From Acute Entero-colitis.*—Unlike intussusception, diarrhœa is a leading symptom. As in intussusception, abdominal pain and vomiting often occur; likewise blood and mucus may be passed, but in entero-colitis fæcal matter or bile is always present in the stools.
2. *From Purpura with Intestinal Symptoms* (*syn.* Henoch's Purpura).—There is likely to be the characteristic rash, which might be mistaken for flea-bites. Intussusception is a not uncommon accompaniment of this form of purpura, consequently the differential diagnosis is not of vital importance, for exploratory laparotomy must be performed in suspicious cases.
3. *From Prolapse of the Rectum.*—This can be eliminated readily. In prolapse the projecting mucosa can be felt continuous with the perianal skin. In intussusception protruding from the anus, the finger passes indefinitely into the depths of a sulcus (figs. 1220 and 1221).

Intussusception in adolescence is nearly always caused by an inverted Meckel's diverticulum (p. 997).

Intussusception in adults is most often due to a papilliferous carcinoma; con-

Eduard Heinrich Henoch, 1820–1910. Professor of Diseases of Children, Berlin.

sequently, the colo-colic type is frequent. Rarer causes are a papilloma or a submucous lipoma. Idiopathic intussusception is extremely uncommon, except in Egypt immediately after the Mohammedan fasting seasons (Mooro).

Preliminary Treatment.—If necessary, the general condition of the patient is improved by the administration of dextrose-saline solution. Especially in cases of more than twelve hours' duration, gastric aspiration should be carried out and continued during and after the operation.

Reduction by Hydrostatic Pressure.—In early cases, Australasian surgeons[1] in particular favour reduction by hydrostatic pressure. In the operating theatre an unlubricated catheter is passed into the rectum of the anæsthetised infant. The catheter is connected to the tubing of a reservoir filled with saline solution, and elevated to the height of 1 metre. The solution is allowed to run into the bowel for four minutes, while the buttocks are pressed together to prevent escape of the fluid. The catheter is then removed, and the fluid is allowed to escape into a bowl. Several such injections are made. After the first injection the fluid returned is blood-stained; subsequently, if reduction of the intussusception is effected, flatus and fæcal matter are passed. If there is the *slightest* doubt as to whether the intussusception has been reduced completely, laparotomy is performed but, as a result of the hydrostatic pressure, in most instances the intussusception is so reduced in size, and is so near its place of origin, that a grid-iron incision in the right iliac fossa is all that is necessary. Scandinavian surgeons favour the use of a barium enema to accomplish the reduction (fig. 1146). The more usual method of treatment is to proceed with the operation, without resorting to these preliminary measures.

Operative Reduction.—The abdomen is opened through a right lower paramedian incision. The first part of the reduction is accomplished by squeezing the lowest part of the sausage-like mass, if possible without exteriorising the bowel, and little by little the intussusception is reduced (fig. 1147). The last part of the intussusception is the most difficult to reduce and should be withdrawn and gently compressed in a warm, saline-soaked pack, to lessen the œdema. In the majority of instances reduction is completed easily by squeezing the apex of the intussusception, as shown in fig. 1148. After reduction is completed, the terminal portion of small intestine, the caput cæci, and the appendix will be seen reddened and stiffened with œdema.

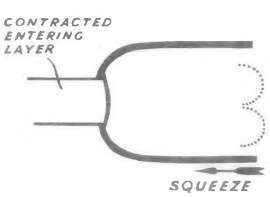


FIG. 1147. — Diagram showing the method of reducing an intussusception.

Owing to earlier diagnosis, the number of irreducible intussusceptions is decreasing; and the mortality is correspondingly lower.

FIG. 1148.—Reducing the terminal part of the intussusception. (*After R. E. Gross.*)

Methods of effecting Reduction in Difficult Cases

1. The little finger is inserted into the neck of the intussusception and an endeavour is made to separate adhesions between the intussuscipiens and intussusceptum, after which reduction is re-attempted (Cope's method).

[1] Intussusception is common in the urban districts of Australia.

Abdel Mooro, Contemporary. Professor of Surgery, Cairo, Egypt.
Sir Zachary Cope, 1881–1975. Surgeon, St. Mary's Hospital, London.

2. The thumb and forefinger are placed as shown in fig. 1148, and gentle pressure is exerted. Gradually the pressure is increased. In this way œdema is squeezed from the region of the ileo-cæcal valve.

Resection of an Irreducible or Gangrenous Intussusception.—Previous methods consisted of exteriorising the ileum and colon by various manœuvres. With exact methods of electrolyte balance and of fluid replacement, these have been abandoned and resection with end-to-end anastomosis is the rule.

After-treatment.—Gastric aspiration should be continued for twelve to twenty-four hours and dextrose-saline administered intravenously, or subcutaneously with hyaluronidase. On the second day the gastric tube is removed and sips of water are given. A few hours later, feeding is commenced with the mother's milk if the infant is breast-fed. In cases where resection has been necessary, intravenous alimentation is required.

Recurrent intussusception occurs in only 2 per cent. of cases of idiopathic intussusception. If a second operation is necessary, the last few inches of the ileum should be anchored to the ascending colon by sutures in order to avoid further recurrence.

VOLVULUS

A volvulus results from axial rotation of a portion of the alimentary tract.

(*a*) **Volvulus Neonatorum** (p. 1040).

(*b*) **Volvulus of the small intestine,** other than the above, usually occurs in the lower ileum, and is favoured by the presence of an adhesion passing from the antimesenteric border of an intestinal loop (fig. 1149) to the parietes or to the female pelvic organs. In Africans, volvulus involving many feet of small intestine without causative adhesions occurs rather commonly. The consumption of a large meal of maize and vegetables seems to predispose to the condition.

FIG. 1149.

Treatment is to untwist the loop, if possible. A causative adhesion should be divided and the stump of its intestinal attachment buried in the intestinal wall by a purse-string suture.

(*c*) **Volvulus of the cæcum** occurs occasionally, especially when the right half of the colon is lax and mobile. Volvulus of this part of the large intestine occurs nearly always in a clockwise direction. The first twist obstructs the ascending colon; if a second twist occurs, it obstructs the ileum also. Cases are recorded between 14 and 88 years. It is about twice as common in females. Presentation is usually acute with abdominal pain 90%, nausea and vomiting 70%, constipation 60%. Less commonly the presentation is chronic often with associated medical illnesses. In about 25 per cent of cases there is a palpable tympanitic swelling not, as a rule, in the right iliac fossa, for in process of torsion the mobile cæcum moves out of the right iliac fossa into the mid-abdomen, or even to the left side. A plain radiograph shows loops of gas-filled ileum, and sometimes an especially large gas shadow which can be recognised as the cæcum. At first, the obstruction is not absolute; fæces and flatus may be expelled after an enema, but unless spontaneous untwisting occurs, the attacks of intestinal colic continue. Barium enema is most effective in diagnosis.

Treatment.—Laparotomy is performed, and in early cases it is usually possible to untwist the bowel. Sometimes before untwisting can be accomplished, it is necessary to deflate the ballooned cæcum by the insertion of a needle. Untwisting should be followed by cæcostomy, which serves two purposes—it relieves distension and it fixes the organ to the abdominal wall, thereby preventing a recurrence. If the cæcum is gangrenous or its viability is not assured, right hemicolectomy (p. 1017) is carried out.

(*d*) **Volvulus of the pelvic colon** is common in Eastern Europe, India, Scandinavia, and Peru. The predisposing causes are indicated in fig. 1150. The loop may

rotate half a turn, in which event spontaneous rectification sometimes occurs. After the loop has rotated 1½ turns the veins involved in the torsion are compressed, and the loop becomes greatly congested. If, as is sometimes the case, it rotates more than 1½ turns, the blood supply is cut off entirely and the loop becomes gangrenous. The rotation nearly always occurs in an *anti-clockwise* direction.

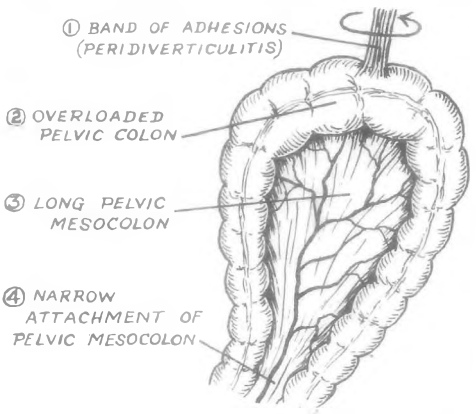
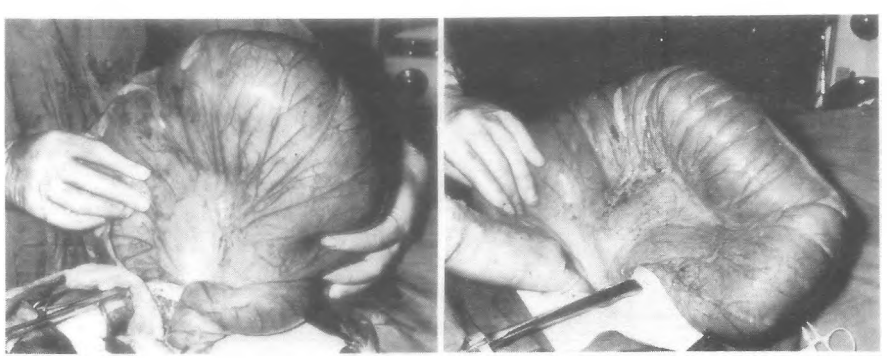

① BAND OF ADHESIONS (PERIDIVERTICULITIS)
② OVERLOADED PELVIC COLON
③ LONG PELVIC MESOCOLON
④ NARROW ATTACHMENT OF PELVIC MESOCOLON

FIG. 1150.—The predisposing causes of volvulus of the sigmoid colon.

Clinical Features. — Males are more commonly affected than females, and the sufferers are usually middle-aged or elderly. There is often a history of acute attacks of left-sided abdominal pain, probably due to a partial volvulus, that untwists itself and is followed by the passage of large quantities of flatus and fæces. As a rule the onset of volvulus of the pelvic colon is sudden and is characterised by severe abdominal pain, often coming on while the patient is straining at stool. Abdominal distension soon follows; in no other condition does extreme abdominal distension, partly due to the diffusion of CO_2 from distended veins, occur so quickly. If the patient is examined two or three hours after the commencement of the attack, the distension is mainly left-sided. In a matter of six hours the whole abdomen becomes distended. Hiccough and retching occur early; vomiting is late. Constipation is absolute, but an enema may be returned blood stained. A plain X-ray film of the abdomen shows massive distension of the colon with gas.

Treatment.—Sigmoidoscopy should be carried out and when the obstruction is reached an attempt is made to coax a soft rectal tube into the twisted gut. This will immediately deflate the gut and operation can be delayed for a few days until the patient is more fit. If deflation does not succeed, laparotomy must be performed immediately. An attempt is made to untwist the gas-filled viscus (fig. 1150);



(*a*) (*b*)

FIG. 1151.—Volvulus of the sigmoid colon (*a*) before and (*b*) after untwisting.
(*S. U. Rahman, F.R.C.S., Manchester.*)

meanwhile a rectal tube is passed by a nurse and guided into position by the surgeon with his hand inside the abdomen. When the colon is deflated, a resection and end-to-end anastomosis can be carried out. Some surgeons prefer to exteriorise and resect the volvulus by the Paul-Mikulicz procedure. This method is specially valuable if there is any suggestion of impending gangrene. Redundancy of the colon makes the performance of this procedure simple (fig. 1152).

OBSTRUCTION BY ADHESIONS AND BANDS

By Adhesions

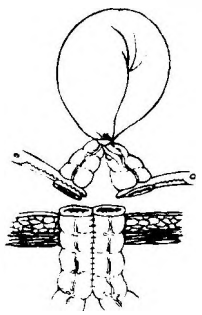


FIG. 1152.—The Paul-Mikulicz operation applied to volvulus of the pelvic colon.

Peritoneal irritation from whatever cause results in local outpouring of fibrin which produces adhesions between apposed peritoneal surfaces. The fibrinous adhesions may disappear when the source of irritation is removed, or they may become vascularised and be replaced by mature fibrous tissue.

Many studies have been made to determine the factors that cause adhesions. Infection is a potent but variable cause. Foreign materials—particularly talc, silk thread, barium sulphate and asbestos—stimulate granuloma formation resulting in fibrous adhesions. Trauma, vascular occlusion—and particularly venous occlusion—generate adhesions.

Numerous substances have been instilled into the peritoneal cavity in the hope of preventing adhesion formation. Hyaluronidase, hydrocortisone, silicone, fibrinolysin, Dextran, polyvinylpyrrolidine (PVP), chondroitin, and streptomycin solutions have all been tried. No single substance has been demonstrated to be completely effective and safe in clinical use.

Type 1.—Post-operative *fibrinous* (' bread and butter ') adhesions are unlikely to produce complete intestinal obstruction unless paralytic ileus is also present. This type of obstruction commences between the third and sixth post-operative days. After some months fibrinous adhesions completely disappear.

Type 2.—Post-operative *fibrous* adhesions are strong bands and occur at a site where an abdominal organ is deficient of blood supply and becomes adherent to the omentum or parietal peritoneum in order to gain an additional blood supply. They can give rise to intestinal obstruction at any time after an abdominal operation.

Type 3.—Adherence of a loop of intestine to an inflamed intraperitoneal structure, e.g. a tuberculous mesenteric lymph node (fig. 1070). The adhesive area occurs at the site of previous granulation tissue.

Type 4 follows chemical irritation from materials such as talc glove powder entering the peritoneal cavity by accident, or sulphonamide or penicillin placed there by design.

Post-operative adhesions giving rise to intestinal obstruction usually involve the lower ileum. Operations for acute appendicitis and gynæcological procedures are the most common precursors of this condition and demand early re-operation.

Treatment.—Gastro-intestinal suction drainage, combined with intravenous fluid therapy, is extremely beneficial; occasionally it is curative, but only in *Type* 1. On this account especial vigilance is necessary, for strangulation, if not present initially, is liable to develop during the course of such treatment. When, as is usually the case, operation is required, although many adhesions are often present, frequently only one of them is found to be the cause of the obstruction, in which case the condition can easily be remedied by dividing this adhesion. At other times the intestine is angulated by adherence to the parietes, to the mesentery, to another coil of

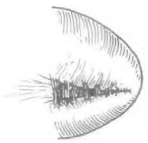


FIG. 1153.—Adhesions causing intestinal obstruction by angulation.

intestine (fig. 1153) or, in the female, to the uterus or adnexa. In these circumstances it is sometimes possible to free the obstructed intestine by dissection. In order to prevent recurrence the bare areas should be covered with omental grafts.

In *Type* 1 the adhesions can usually be separated with a finger, thereby releasing the obstruction. When adhesions are widespread and the small intestine so matted that a definite point of obstruction cannot be found, it may be best to do a Noble plication.

Noble's Plication Operation (fig. 1162).—All involved intestine is freed by dissection, which may include the whole of the small bowel. Adjacent coils (average length 15 to 20 cm) are sutured together along their mesenteric borders. If only a portion of the small intestine is plicated, the resulting folds of mesentery must be united to prevent internal herniæ.

Childs-Phillips' Transmesenteric Plication.—A recent modification of Noble's operation is reported to be a more reliable procedure. It consists of placing three long synthetic sutures through the mesentery of the plicated bowel, each doubled back upon itself and then tied loosely so that the pattern of the intestinal segments becomes laid out ' like sausages in a box '. A long needle, such as a liver needle or a modification of this, simplifies the procedure. The stitch should pass a few centimetres from the bowel wall and not be adjacent to it.

By a Band

A band (usually one band only is culpable) is occasionally the cause of acute obstruction to the small intestine. Such a band may be:

(*a*) Congenital: often an obliterated vitello-intestinal duct (p. 1128).

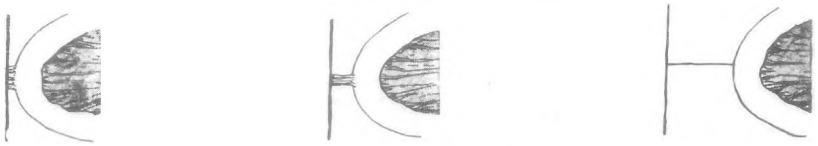


FIG. 1154.—Metamorphosis of an inflammatory peritoneal band from an adhesion.

(*b*) A string-like, frequently thin and fragile, band (fig. 1154) following previous bacterial peritonitis.

(*c*) A portion of greater omentum, adherent usually to the parietes, constitutes an obstructing band of relative stoutness (fig. 1155).

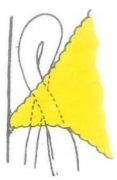


FIG. 1155.—Intestinal obstruction occurring under a portion of greater omentum adherent to the parietal peritoneum.

Treatment.—Provided the involved intestine is viable, division of the band and release of the obstructed loop is the simplest and one of the most satisfactory operations in surgery. Entrapment of a coil of small intestine beneath a string-like band is notorious for producing early gangrene. Even when the imprisoned intestine, after release, regains its pristine hue, if the narrow areas that have suffered direct compression by the band show any colour changes, they should be invaginated. Gangrenous intestine must be resected.

Thomas Benjamin Noble, 1895–1958. Surgeon, Community Hospital, Indianapolis, Indiana, U.S.A.
Wesley A. Childs, Contemporary. Surgeon, Albuquerque, New Mexico, U.S.A.
Richard V. Phillips, Contemporary. Surgeon, Albuquerque, New Mexico, U.S.A.

OBSTRUCTION DUE TO AN INTERNAL HERNIA

A portion of the small intestine passes into one of the retroperitoneal fossæ or into a congenital defect of one of the mesenteries, there to be imprisoned. The potential internal herniæ are as follows:

1. * **The Foramen of Winslow.**—The portal vein, common bile duct, and hepatic artery lie in its free border.
2. * **A Hole in the Mesentery.**
3. **A Hole in the Transverse Mesocolon.**
4. **A Defect in the Broad Ligament.**
5. **Congenital or Acquired Diaphragmatic Hernia** (p. 753).
6. **One of the retroperitoneal fossæ,** of which the following are most important:

Fossæ about the Duodenum

(*a*) * *The Left Paraduodenal Fossa.*—The inferior mesenteric vein lies in its free border.

(*b*) * *The Right Duodeno-jejunal Fossa.*—The superior mesenteric artery runs in its free border.

Fossæ about the Cæcum and Appendix (fig. 1156).

(*a*) *Superior Ileo-cæcal Fossa.*—Between the general mesentery and a fold of peritoneum raised by the anterior cæcal branch of the ileo-colic artery.

(*b*) *Inferior Ileo-cæcal Fossa.*—Between the 'bloodless' fold of Treves and the mesentery of the appendix.

(*c*) *The retro-cæcal fossa* behind the cæcum.

The intersigmoid fossa is situated in the base of the pelvic mesocolon.

The supravesical fossa is bounded by the median and lateral umbilical folds (obliterated urachus and umbilical arteries). A loss of weight with coincident loss of prevesical fat deepens the fossa.

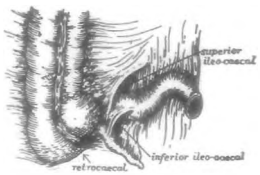


FIG. 1156.—Peritoneal fossæ around the cæcum and appendix.

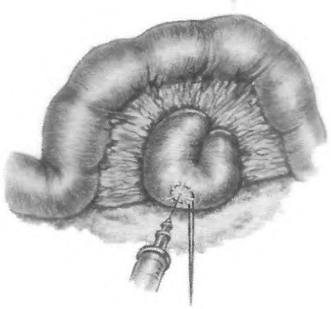


FIG. 1157.—Strangulation through a hole in the mesentery. Deflating the obstructed coil before attempting reduction.

An internal hernia is an uncommon cause of intestinal obstruction; a pre-operative diagnosis can be only a guess.

Treatment.—In those herniæ above marked with a ★ an important blood-vessel runs in the edge of the hernial orifice, and to divide the constricting agent (which is the correct treatment for the other varieties) may cause serious bleeding. When confronted with the difficult problem of how to release imprisoned intestine without dividing the constricting agent, the reader is referred to fig. 1157.

OBSTRUCTION FROM STRICTURE OF THE SMALL INTESTINE

Cicatricial contracture is usually an aftermath of tuberculous ulceration or Crohn's disease (p. 1010). Multiple strictures are usually present. Malignant stricture is rare, although both carcinoma and sarcoma occur from time to time (p. 1016). Gradual contraction of a stricture causes a chronic obstruction of the small intestine with dilatation and hypertrophy of the intestinal wall. Clinically there is some colic and distension, associated with the features of the blind-loop syndrome (p. 996), i.e. steatorrhœa, loss of weight, anæmia, avitaminosis, and cachexia.

Treatment.—Except in emergency, a simple stricture should never be by-passed by a lateral anastomosis. A blind-loop syndrome is sure to follow. A simple stricture

Jacob Benignus Winslow, 1669–1760. Professor of Anatomy, Physic and Surgery, Paris.
Sir Frederick Treves, 1853–1923. Surgeon, The London Hospital.

should be excised locally, and a malignant stricture widely, and the continuity of the intestine restored by end-to-end anastomosis.

OBSTRUCTION BY OBTURATION OF THE SMALL INTESTINE

Obstruction by a gall-stone[1] usually occurs in elderly obese women because the gall-stone, which is 2·5 cm or more in diameter, requires some years to reach this dimension, and more years elapse before it ulcerates through the gall-bladder into the duodenum (fig. 1012). It passes down the small bowel, becoming impacted about 60 cm from the ileo-cæcal valve (fig. 1158), because this is the narrowest part of the small gut. The symptoms are elusive. The patient has recurrent mild colic, accompanied by copious vomiting. As the obstruction is incomplete, there is often some result from an enema, and remissions of symptoms are frequent, but the vomiting returns, and by this time it is bilious; as time goes on it becomes fæculent. An X-ray may reveal the stone, and the small intestine will be seen to be distended with gas. It may actually be felt in the Pouch of Douglas on rectal examination. There is no abdominal distension until late, and late intestinal obstruction in an old person is an almost hopeless condition. Rarely, a large stone ulcerates into the transverse colon, and from time to time it remains in the large intestine long enough to increase in size, by the deposition of fæcal matter upon it, so that eventually it becomes impacted in the rectum.

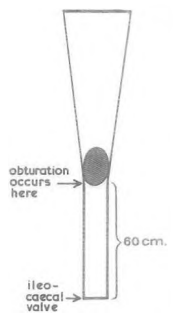


FIG. 1158.—Site of impaction of gall-stone.

Treatment.—Through a lower right paramedian incision the loop containing the stone is delivered and packed off most carefully. Occasionally, it is possible to crush the stone between the fingers while it is still in the lumen. If not, the intestine is opened and the stone removed. It is an advantage to sew up the incision in the intestine transversely, to avoid constriction. A gall-stone that causes intestinal obstruction is nearly always barrel-shaped and unfaceted (fig. 1159). If there is a facet, it is essential to palpate the intestine above the obstruction for a possible fellow-calculus. The area of the gall bladder should be left strictly alone, as even gentle manipulation can break down a cholecyst-duodenal fistula.

FIG. 1159. — Gall-stone removed from the lower end of the ileum.

Bolus obstruction is particularly liable to occur after partial gastrectomy (p. 855), when insufficiently masticated articles of food will be hurried into the small intestine where they become impacted at the narrowest portion of the intestine, i.e. about 60 cm (fig. 1158) from the ileo-cæcal valve. Normally they would be retained in the stomach until they had become partially digested. Brussels sprouts, dried fruit, and, particularly, unmasticated orange pulp have caused intestinal obstruction in these circumstances.

Treatment.—Timely laparotomy is required. An attempt should be made to squeeze the bolus gently onward into the cæcum, and there to break it up by kneading. Should the bolus be impacted so firmly that this manœuvre proves impracticable—and such is likely only in cases of obstruction

[1] Formerly called gall-stone 'ileus'. Ileus, from ἰλεος (Greek) = colic.

James Douglas, 1675–1742. Scottish Anatomist and Obstetrician.

by dried fruit or when a stricture of the small intestine is present—enterotomy must be performed. After the obstructing material has been removed the opening in the intestine is sutured, the coil mechanically cleansed before returning it to the abdomen, which is then closed.

Obstruction by stercolith gives rise to symptoms identical with those of obstruction by a gall-stone, for a stercolith, contrary to what might be expected, is usually formed and found in the small intestine, particularly in cases where a jejunal diverticulum or a stricture in the ileum is present. On careful dissection of a stercolith it is not unusual to find a nucleus of recognisable material, e.g. tomato skins.

Obstruction due to Worms.—An aggregation of *ascaris lumbricoides* is sometimes the cause of low small intestinal obstruction in children, usually under ten years of age, and especially those living in or near the tropics. There is debility out of proportion to that produced by the obstruction. The obturation is inclined to follow the ingestion of an anti-helminthic. If it is not known that the patient is suffering from ascaris infestation, a worm in the vomitus or the presence of eosinophilia may be the means of making a correct pre-operative diagnosis. In this form of intestinal obstruction laparotomy must be performed, and if possible the tangled mass should be kneaded along the ileum into the cæcum.

EMBOLISM AND THROMBOSIS OF THE MESENTERIC VESSELS

Arterial embolism is more common than spontaneous thrombosis, and the superior mesenteric vessels are implicated much more frequently than the inferior; the latter is often silent due to the better collateral circulation.

Embolism of the Superior Mesenteric Artery.—Possible sources of emboli include: the left auricle (especially in atrial fibrillation); a mural myocardial infarct; an atheromatous plaque or aneurysm of the aorta; a vegetation on the mitral valve; pulmonary vein thrombosis secondary to septic infarct; or from a left atrial myxoma.

Primary thrombosis of the superior mesenteric artery is the result either of arteriosclerosis or thrombo-angiitis obliterans.

Primary thrombosis of the superior mesenteric vein or its tributaries occurs occasionally in portal hypertension, portal pyæmia (pylephlebitis), sickle cell disease, and in women taking the contraceptive pill.

No matter whether the occlusion is arterial or venous, hæmorrhagic infarction occurs although, in the case of embolism, a short-lived pallor has been observed at early laparotomy. The intestine and its mesentery become swollen and œdematous, demarcation between infarcted and healthy intestine being gradual. Blood-stained fluid is exuded into the peritoneal cavity and the lumen of the infarcted intestine becomes filled with blood. When the main trunk of the superior mesenteric artery becomes occluded, infarction of nearly the whole of the small intestine, the cæcum, and part of the ascending colon occurs (fig. 1160). More frequently a branch of the main vessel is implicated and the area of infarction is proportionately less.

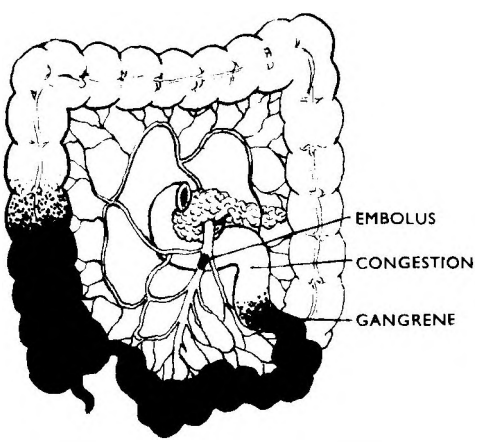


FIG. 1160.—Embolus lodged in the main trunk of the superior mesenteric artery. Showing the widespread gangrene that results.

Clinical Features.— (i) *Pain.* The most important clue to early diagnosis is the sudden onset of severe abdominal pain in a patient with atrial fibrillation or atherosclerosis. Pain is typically central abdominal and out of proportion to the physical findings.

(ii) *Gastro-intestinal emptying* with persistent vomiting and bowel evacuation

occurs early. In late cases altered blood may be passed per rectum. Shock due to fluid and later actual blood loss into the bowel develops rapidly.

(iii) *Abdominal tenderness* may be mild initially but later rebound tenderness and rigidity supervene.

(iv) X-ray examination of the abdomen usually shows no gas in the small intestine. A neutrophilic leucocytosis occurs early and almost always exceeds 15,000.

Provided that the predisposing factors are considered, the diagnosis of mesenteric thrombosis or embolism is not difficult. Mesenteric vascular occlusion carries a higher mortality than any other type of intestinal obstruction. This is not surprising when one considers the type of patient prone to this disaster.

Treatment.—In the early embolic case, when the diagnosis has been confirmed at laparotomy, superior mesenteric embolectomy should be attempted. In the later cases the affected bowel should be resected. In all cases, blood transfusion is required and anti-coagulant treatment should be commenced twelve to twenty-four hours after operation. In patients who recover, intestinal obstruction secondary to ischæmic fibrosis of an area of small bowel may occur.

Infarction of the large intestine is rare. Lodgement of an embolus in the middle colic artery should be treated by resection of the transverse colon with exteriorisation of both ends. If the patient survives and is deemed fit for a further operation, the bowel ends can be joined; otherwise the colostomy must be permanent.

Ischæmic Colitis is the term given to describe the structural changes in the colon as a result of deprivation of blood. Such episodes are similar to those in the heart or brain and occur specially in the region of the splenic flexure whose blood supply is particularly precarious. They are classified into gangrenous (see above) stricturing and transient forms (Marston). The presenting syndrome is lower abdominal pain of abrupt onset, vomiting, fever and the passage of bright red blood per rectum. The

FIG. 1161.—Ischæmic colitis—site of lesion in a woman of 67 with six months' rectal bleeding; occasional colicky pain and vomiting. Lesion resected with recovery. (*Adrian Marston, F.R.C.S., London.*)

age is that expected in degenerative vascular disease and at operation the length of colon has the appearance of a hæmorrhagic infarct which may require immediate resection. In the cases where no operation is performed, a stricture may appear on a barium enema as a tubular narrowing over several inches of bowel most often in the region of the splenic flexure. The differential diagnosis is from carcinoma, Crohn's disease (p. 1010) and ulcerative colitis. An aortogram is usually conclusive.

PARALYTIC ILEUS (*syn.* ADYNAMIC OBSTRUCTION)

This has already been defined as a state in which the intestine fails to transmit peristaltic waves, and is due to failure in the neuromuscular mechanism—i.e. in the myenteric plexus (Auerbach) and the submucous plexus (Meissner). This results in a collection of fluid and gas in the intestine, with resulting distension, vomiting, absent or high 'tinkling' bowel sounds, and failure to pass flatus.

The following varieties are recognised:

1. *Post-operative.*—Some degree of ileus, either local or general, may follow any abdominal operation. In the absence of infection, it is not serious. Indeed, it has been shown that intestinal motility and absorption commonly returns to normal in about sixteen hours—well ahead of gastric and colonic activity. Post-operative ileus may be prolonged if there is hypoproteinæmia, or latent renal failure (*vide infra*), or if gastrointestinal suction is continued beyond the point at which effective bowel sounds have returned.

Jeffrey Adrian Priestley Marston, Contemporary. Surgeon, Middlesex Hospital, London.
Leopold Auerbach. 1828–1897. Professor of Neuropathology, Breslau, Germany (now Wroclaw, Poland).
Georg Meissner, 1829–1905. Professor of Physiology, Göttingen, Germany.

2. *Infective.*—Peritonitis gives rise to prolonged ileus, but several factors may be involved. At the outset, peristalsis ceases as a normal response to prevent dissemination, but afterwards, bacterial toxins prevent the normal activity of the nerve plexuses. When the bowel begins to recover, the early, feeble peristaltic waves may not be able to overcome the obstructive effect of the newly formed slender adhesions between loops of intestines, and so further quiescence of activity follows. In this kind of ileus, there are therefore mechanical as well as neurogenic factors to be considered.

3. *Reflex.*—This form of ileus may occur following fractures of the spine or ribs, retroperitoneal hæmorrhage, or even the application of a plaster jacket.

4. *Uræmia.*—This type, with distension, vomiting, and hiccoughs is well recognised, and is seen in renal failure. It may follow prostatectomy.

5. *Hypokalæmia.*—A low serum potassium may cause ileus.

Clinical Features.—Ileus is suspected if, 48 hours after laparotomy:

(*a*) There has been no passage of flatus.

(*b*) There is no return of normal bowel sounds on auscultation.

Abdominal distension becomes more marked and drum-like (tympanitic); in the absence of gastric aspiration, there is effortless vomiting of large volumes of dirty fluid. There is no colic, and often no abdominal pain at all. There may be respiratory distress from the abdominal distension and the pulse-rate increases. Prolonged distension increases the risk of wound dehiscence. Radiologically, the abdomen shows gas-filled loops of intestine with multiple fluid levels.

It is important to recognise three types of bowel sounds:

(1) The *normal* low-pitched borborygmi each lasting about a second and occurring every twenty seconds or so.

(2) The prolonged, rapidly recurring and noisy borborygmi of *dynamic obstruction.*

(3) The high-pitched tinkling note 'like bells at evening pealing', which occurs every ten to thirty seconds, and is distinctive of *paralytic ileus.* It is due, not to peristalsis, but to the overflow of fluid from one distended loop to another.

Management.—The essence of this is prevention and the incidence has been greatly reduced by routine naso-gastric suction and withholding fluids by mouth after laparotomy until normal bowel sounds and/or the passage of flatus returns. Further, electrolyte balance should be achieved before, and maintained after operation. Specific treatment is directed to the cause, but there are some principles which have general application:

(1) The primary cause must be removed.

(2) Normal bowel activity will return if distension is relieved by adequate gastro-intestinal decompression. It is probably the distension of the gut (possibly exacerbated by the air swallowing of anxiety), that causes the ileus, rather than the ileus that causes the distension. Thorough decompression is therefore essential (p. 1036). If a post-operative indwelling gastric tube is used, it must not be spigoted but be kept open to permit swallowed air to be evacuated.

(3) Morphine or pethidine in repeated small doses are well proved and valuable sedatives in these cases.

(4) Close attention to the fluid and electrolyte balance, especially the serum potassium and the blood urea, is essential (Chapter 8).

(5) Peristaltic stimulants have no place in treatment. The object is to *rest* the bowel, not to stimulate it, i.e. don't flog a tired horse!

Measurement of abdominal girth at the umbilicus is the only way of being certain of increase or decrease of distension. This should be done four- or six-hourly. As recovery occurs, segmentation returns before peristalsis. This may cause 'wind pains', but is soon followed by the passage of flatus.

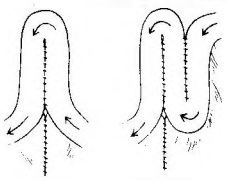


FIG. 1162.—Noble's plication operation.

Resolution is often accompanied by a short period of diarrhœa. No treatment is necessary for this.

If paralytic ileus is prolonged and threatens life, laparotomy should be carried out and the bowel decompressed with a Savage's sucker (p. 1036).

CHRONIC INTESTINAL OBSTRUCTION

The various abnormalities and diseases giving rise to chronic intestinal obstruction are described in Chapters 49 and 52. There remains one condition to be considered here.

Fæcal impaction in the rectum and the colon occurs principally in elderly, and often bed-ridden, patients. It is all too often overlooked, in which case the patient suffers unnecessary distress.

The symptoms are those of chronic intestinal obstruction, and attacks of spurious diarrhœa are a common accompaniment. A fæcal accumulation may form a palpable abdominal mass which can be indented. When the mass is situated in the rectum, it can be indented by the palpating finger.

Treatment.—Enemas are usually insufficient. Repeated bowel wash-outs and the use of various proprietary fæcal plasticisers (e.g. Dioctyl) by mouth or by enema, may result in disimpaction. Otherwise the anal sphincter must be dilated under general anæsthesia and the mass removed with the aid of fingers and tablespoon.

CHAPTER 51

THE VERMIFORM APPENDIX

SURGICAL ANATOMY

THE vermiform appendix is present only in man, certain anthropoid apes, and the wombat.[1] Morphologically, it is the undeveloped distal end of the large cæcum found in many lower animals. It is true that many herbivores are provided with a wide-lumened cæcal diverticulum in which bacteriolytic breakdown of cellulose takes place. However, the walls of this diverticulum lack the heavy deposition of lymphoid tissue that characterises the *vermiform* appendix (Last).

The appendix varies considerably in length and circumference. The average length is between 7·5 and 10 cm. Specimens of over 30 cm. in length have been recorded. The appendix averages 0·5 cm. longer in the male than in the female. The lumen, which should admit a matchstick, is irregular, being encroached upon by the multiple longitudinal folds of mucous membrane.

From without inwards, the structure of the appendix is as follows. There is a peritoneal coat which completely invests it, except along the narrow line of attachment of the mesoappendix. The muscular coat resembles that of the small intestine. There is a well-developed submucous coat containing, especially in childhood and youth, a large number of lymphoid follicles. The mucous membrane resembles that of the large intestine, but there are fewer crypts of Lieberkühn.

The **mesoappendix** which springs from the lower surface of the mesentery is subject to great variations. Sometimes as much as the distal one-third of the appendix is bereft of mesoappendix. Especially in childhood, the mesoappendix is so transparent that the contained blood-vessels can be seen. In many adults it becomes laden with fat, which obscures these vessels.

The **appendicular artery**, a branch of the lower division of the ileo-colic artery, passes behind the terminal ileum to enter the mesoappendix a short distance from the base of the appendix. It then comes to lie in the free border of the mesoappendix; but for a variable distance from the tip, where the mesoappendix is lacking, the artery lies directly on the muscle wall beneath the peritoneal coat. **An accessory appendicular artery** may be present (fig. 1163).

The appendicular vein is a radicle of the ileo-colic vein, which drains into the portal system.

Lymphatic Vessels.—Four, six, or more lymphatic channels traverse the mesoappendix to empty into the ileo-cæcal lymph nodes.

McBurney's point lies at the junction of the lateral third with the medial two-thirds of a line joining the anterior superior iliac spine and the umbilicus (fig. 1164). McBurney's point is the classical site of greatest tenderness in appendicitis, and also a most useful point to have in mind when a grid-iron incision is made.

Inconstancy of Position.—The relative frequency of the more usual positions occupied by the organ is depicted in fig. 1165. In addition, the appendix must necessarily share in abnormalities in position of the cæcum. The most frequent of these is failure of the cæcum to descend, which results in the base of the appendix being situated in the right hypochondrium. Very occasionally the cæcum and

[1] Wombat — a nocturnal, burrowing Australian marsupial.

Raymond Jack Last, Contemporary. Formerly Professor of Applied Anatomy, Royal College of Surgeons of England, London.
Johann Nathaniel Lieberkühn, 1711–1756. Physician and Anatomist, Berlin.
Charles McBurney, 1845–1913. Professor of Surgery, Columbia University College of Physicians and Surgeons New York, U.S.A.

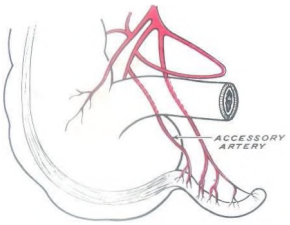


FIG. 1163.—In nearly 50 per cent. of cases there is an accessory appendicular artery, a branch of the posterior cæcal. (*After T. Seshachalam.*)

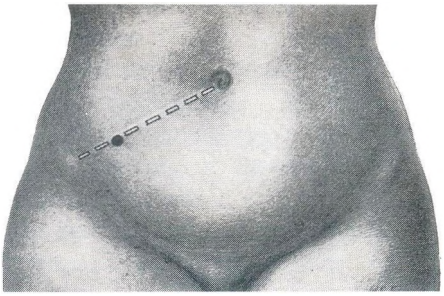


FIG. 1164.—McBurney's point, the surface marking of the base of the appendix.

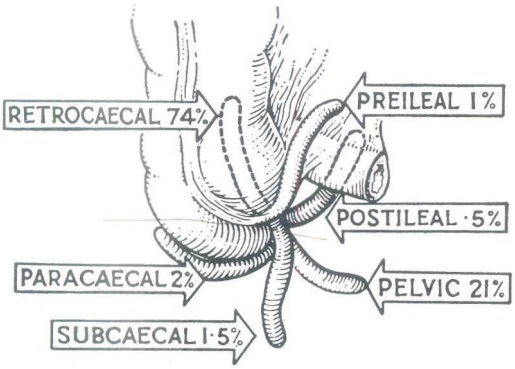


FIG. 1165.—The various positions of the appendix. (*After Sir Cecil Wakeley London, formerly P.R.C.S.*)

appendix are situated in the left iliac fossa or in the left hypochondrium (see below).

Locating the Appendix.—The traditional method consists of following one of the tæniæ coli downwards till the appendix is reached. If the organ is still not visible and it is certain that it has not been removed, it will probably be found adherent to posterior cæcal wall.

CONGENITAL ABNORMALITIES

Agenesis.—Once in 100,000 persons the vermiform appendix is absent; possibly some of these are congenital, but most are due to sloughing of an appendix that had intussuscepted previously (p. 1073).

Duplication.—A few cases of double appendix have been reported; in some instances one of the twin appendices has been found acutely inflamed and the other uninvolved.

Left-sided Appendix.—*Situs inversus viscerum*, a congenital abnormality where there is complete transposition of thoracic and abdominal viscera, occurs once in 35,000 individuals, and is more common in males. In such cases, of course, the vermiform appendix is situated on the left as it is also in some cases of non-rotation of the mid-gut (fig. 1166).

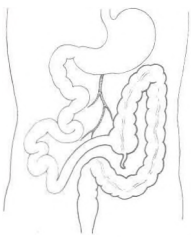


FIG. 1166.—Left-sided cæcum and appendix due to incomplete intestinal rotation. (*After Findlay and Humphreys.*)

ACUTE APPENDICITIS

During recent years the mortality from acute appendicitis has been falling. Earlier diagnosis and appendicectomy while the inflammation is still confined to the appendix, the recognition by the general public that it is dangerous to take or give to a child a purgative in the presence of undiagnosed abdominal

pain, more discrimination in performing immediate appendicectomy in late cases, a greater appreciation of the importance of accurate fluid and electrolytic balance, better anæsthesia, and the control of infection by antibiotics have all played a part in bringing about this improvement. Nevertheless, hospital statistics[1] show that in cases where the inflammation is no longer limited to the appendix, the mortality rate is 5 per cent. in males and 6 per cent in females. In men over sixty-five, the mortality may be as much as 25 per cent. owing to the general effects of arteriosclerosis.

All are agreed that early diagnosis with prompt appendicectomy is the goal. On the other hand, some patients fail to seek medical advice until a late hour. In such cases there must be no rule of thumb, and when to operate and when not to operate becomes a matter of refined judgment. If the attack has been present for some days, the patient's condition is satisfactory and if there is a lump, it is often wise to adopt the delayed treatment (p. 1069).

Ætiology

Until the close of the nineteenth century appendicitis remained unrecognised. Unquestionably, before this time it was a comparatively rare disease, but there can be no doubt that it existed even in remote times, for an acutely inflamed, perforated appendix was found preserved in the mummy of a young royal princess of Egypt (Spencer).

The riddle of appendicitis—its actual cause and its meteoric rise from an insignificant disease to the most common serious intra-abdominal inflammatory affection of Western civilised races—has been a matter for much speculation. So far no satisfactory explanation has been forthcoming. The following ætiological factors are important, but for the most part they are purely contributory.

Race and Diet.—Appendicitis is particularly common in the highly civilised European, American, and Australasian countries, while it is rare in Asiatics, Africans, and Polynesians. Rendle Short showed that if individuals from the latter races migrate to the countries where appendicitis is common, they soon acquire the local susceptibility to the disease. Even apes in captivity appear to acquire the human liability to appendicitis. These significant facts satisfy many that the rise of appendicitis amongst the highly civilised is due to departure from a simple diet rich in cellulose to one relatively rich in meat. But this cannot be the whole explanation, for acute appendicitis occurs in lifelong vegetarians and even in babes at the breast.

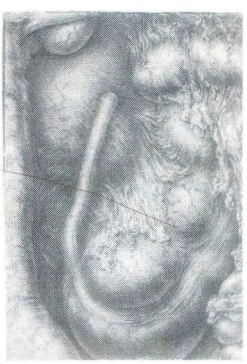


FIG. 1167.—Long retrocæcal appendix. This type tends to be familial.

Social Status.—In England, acute appendicitis is more common among the upper and middle classes than in those belonging to the so-called working class. Thus the mortality from acute appendicitis is about 20 per cent. higher in men of social classes I and II (professional and managerial workers) than it is in social class V (unskilled labourers) (Registrar-General, 1954).

Familial Susceptibility.—This unusual but generally accepted fact can be accounted for by an hereditary abnormality in position of the organ, which predisposes to infection. Thus the whole family may have long rectrocæcal appendices (fig. 1167) with comparatively poor blood supply.

Obstruction of the Lumen of the Appendix.—When an acutely inflamed appendix has been removed, some form of obstruction to its lumen can be demon-

[1] Statistics from the Social Medical Research Unit of the Medical Research Council, The London Hospital, 1957.

Arthur Morgan Spencer, Contemporary. Formerly Medical Superintendent, Powick Hospital, nr. Worcester, England.
Arthur Rendle Short, 1880–1953. Professor of Surgery, Bristol, England.

strated in a large percentage of cases. The obstructing agent is usually a fæcolith or a stricture; exceptionally, a foreign body or a round worm or thread-worms are found.

FIG. 1168.—Fæcoliths. X-ray of an appendix after removal.

Fæcoliths (fig. 1168) vary in size and have a laminated structure. They are composed of inspissated fæcal material, calcium and magnesium phosphates and carbonates, bacteria and epithelial debris; rarely, a foreign body is incorporated in the mass. The presence of a fæcolith or fæcoliths postulates some form of appendicular stasis.

Worms. — Worms (fig. 1169) and other intestinal parasites can injure the appendicular mucous membrane and occasionally block its lumen.

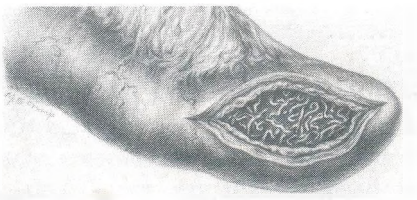


FIG. 1169.—An appendix filled with oxyuria vermicularis.

The Abuse of Purgatives.—It is abundantly clear that the ingestion of purgatives, particularly castor oil, by patients with ' stomach ache ', and the violent peristaltic action which results, favours, and often determines, perforation of an inflamed appendix. ' Purgation means perforation ' is a wise adage.

Bacteriology.—Cultures from inflamed appendices usually reveal that the infection is mixed and there is hardly a pyogenic organism which has not been isolated from such specimens. The most common organisms present are a mixture of *Esch. coli* (found in 85 per cent of cases), enterococci (30 per cent), non-hæmolytic streptococci, anærobic[1] streptococci, together with *Cl. welchii* (30 per cent) and bacteroides (p. 967). In most instances the infecting organisms are normal inhabitants of the lumen of the appendix.

Pathology—The menace of acute appendicitis lies in the frequency with which the peritoneal cavity is infected from this focus, 1. By perforation, 2. By transmigration of bacteria through the appendicular wall.

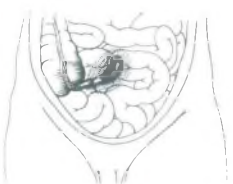


FIG. 1170.—When an inflamed appendix lies amidst coils of small intestine the risk of peritonitis is high.

The greater omentum, the ' abdominal policeman ', attempts to arrest the spread of peritoneal invasion, whilst violent peristalsis from ingested purgatives tends to spread it. Obviously, if the inflamed appendix lies dangling amidst coils of small intestine (fig. 1170), the threat of peritonitis is increased; should early perforation occur, diffusing peritonitis is inevitable.

It is of great importance to recognise two types of acute appendicitis.

(*a*) **Non-obstructive Acute Appendicitis.**—The inflammation usually commences in the mucous membrane; less often in the lymph follicles. Like any inflammatory process, it terminates in one of the following ways: (1) Resolution; (2) Ulceration; (3) Suppuration; (4) Fibrosis; (5) Gangrene. All grades of inflammation occur and once infection reaches the loose submucous tissues it progresses rapidly. The organ becomes turgid, dusky red, and hæmorrhages occur into the mucous membrane. The vascular supply of the distal part of the appendix is often in jeopardy because it is intra-

[1] The foul odour of exudates connected with appendicitis with perforation is caused by anærobic streptococci or anærobic bacilli, and not by *Esch. coli*, as is so commonly believed.

mural and liable to occlusion by inflammation or thrombosis. This may lead to gangrene of the tip. Non-obstructive appendicitis may progress sufficiently slowly for protective adhesions to form, and the resulting peritonitis is localised. In many instances the infection never progresses beyond the mucous lining (i.e. catarrhal inflammation) but although the attack passes off, it is unlikely that a *status quo ante* is ever regained. Because the tip suffers most, fibrosis usually occurs therein and this is a classical finding in recurrent appendicitis (fig. 1184).

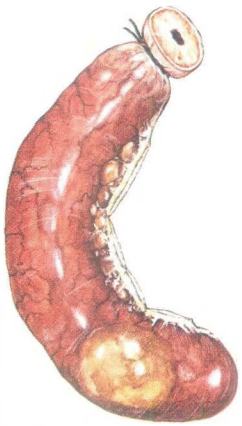


FIG. 1171.—Acute appendicitis. Perforation imminent.

(*b*) **Obstructive Acute Appendicitis.**—About one-third of cases of acute appendicitis belong to this group. The obstruction can be in the lumen (fæcolith, foreign body, or parasites); in the wall (almost invariably inflammatory, but exceptionally by a carcinoma of the cæcum); outside the wall (adhesions and kinking). Of these, much the most common is a fæcolith. On occasions the appendix becomes strangulated in an inguinal or femoral hernia. A kink is unlikely to cause complete obstruction.

In obstructive appendicitis the products of inflammation become pent up (fig. 1172) so that the inflammation proceeds more rapidly and more certainly to gangrene or perforation. Often within twelve to eighteen hours the appendix distal to the obstruction becomes gangrenous (fig. 1173). Close examination of gangrenous appendices directly after their removal shows conclusively that they usually belong to the obstructive group (Wilkie). Perforation occurs most often at the site of an impacted fæcolith before protective adhesions have had time to form. The escaping purulent and gaseous contents are under high pressure and early widespread peritonitis is liable to ensue.

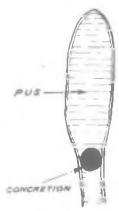


FIG. 1172.—Acute obstructive appendicitis.

FIG. 1173.—Acute obstructive appendicitis with gangrene. There is a large fæcal concretion impacted in the proximal end of the lumen of the organ.

Clinical Features (Fitz)

Age Incidence.—Rare before the age of two, acute appendicitis becomes increasingly common during childhood and adolescence. The maximum incidence is between the ages of twenty and thirty; thereafter there is a gradual decline, but no age is exempt.

The patient often gives a history of similar slight attacks. The attack can commence at any time, but frequently it does so in the early hours of the morning, awakening the patient from sleep. Recent constipation is usual.

Sir David Percival Dalbreck Wilkie, 1882–1938. Professor of Surgery, Edinburgh.
Reginald Heber Fitz, 1843–1913. Professor of Medicine, Harvard University, Boston, Massachusetts, U.S.A. Published a classical paper on appendicitis in 1886 which drew attention to the disease and framed the clinical features.

Non-obstructive Acute Appendicitis.—Typically there are three main features:

1. **Abdominal Pain which Shifts.**—Usually the first symptom is pain around the umbilicus, in the epigastrium, or it may be generalised. This is *visceral* pain and is therefore somewhat vague (p. 990). It is due to distension of the appendix. In non-obstructive cases the pain is constant but in obstructive cases it is colicky. After a few hours the pain shifts to the point where the inflamed appendix irritates the parietal peritoneum, which is very sensitive. This pain is *somatic* or *peritoneal*, accurately localised and constant. A clinical and often valuable observation is that coughing causes pain in acute appendicitis but the pain is absent in the case of a stone in the ureter (p. 1064).

2. **Upset of Gastric Function.**—Protective pylorospasm occurs and this may be manifested by anorexia, nausea, vomiting a brown-furred tongue, and a foul breath. Typically the vomiting is of short duration and stops as soon as the stomach is empty. In the majority of instances the patient is constipated but occasionally diarrhœa occurs, especially in the very young or when the appendix lies in the post-ileal or pelvic position.

3. **Localised Tenderness at the Site of the Appendix.**—As soon as the pain has shifted, there is localised tenderness either at McBurney's point or elsewhere, as determined by the site of the appendix (fig. 1165). This tenderness may be confined to the pelvis and thus *rectal examination must be done in every case of lower abdominal pain.*[1] The identification of the site of the appendix is important; it will determine the operative approach. With the passage of time, accurate localisation becomes more difficult as muscular rigidity becomes evident in addition to the tenderness.

General Features.—During the first six hours there is rarely any alteration in the temperature or pulse-rate; after that time slight pyrexia[2] (37·2° to 37·7° C) with corresponding increase in the pulse-rate to 80 or 90, is usual. In severe cases, as time passes the temperature rises to about 38·3° C but seldom more, and the pulse-rate becomes correspondingly elevated. In 90 per cent. of cases the white cell count is greater than 10,000 cells per cu mm (10×10^9/l).

In **Obstructive Appendicitis** the sequence of clinical events occurs much more quickly and early diagnosis and treatment are accordingly much more urgent. The onset is abrupt and there may be severe generalised abdominal colic from the start. The temperature is often normal, but vomiting is common, so that the clinical picture mimics acute intestinal obstruction. In a few hours, however, tenderness appears in the right iliac fossa and the diagnosis will become clear.

Special Features, according to position.

Retrocæcal.—Rigidity is often absent ('silent' appendix) and even on deep pressure tenderness may be lacking, the reason being that the cæcum,

[1] "If you don't put your finger in the rectum, you may put your foot in it."
[2] In 20 per cent. of cases the temperature is 37° C or less.

distended with gas, prevents the pressure exerted by the hand from reaching the inflamed structure, and gurgling may even be elicited. However, deep tenderness is often present in the loin, and rigidity of the quadratus lumborum may be in evidence. Psoas spasm, due to the inflamed appendix being in contact with that muscle, may be sufficient to cause flexion of the hip joint; to extend the joint causes abdominal pain. Hyperextension of the hip joint may induce abdominal pain when the degree of psoas spasm is insufficient to cause flexion of the hip.

Pelvic.—When the appendix lies entirely within the pelvis there is usually complete absence of abdominal rigidity, and often tenderness over McBurney's point is lacking as well. In some instances deep tenderness can be made out just above and to the right of the symphysis pubis. In either event a rectal examination reveals tenderness in the recto-vesical pouch or the pouch of Douglas, especially on the right side. Psoas spasm may also be present when the appendix is in this position: alternatively, spasm of the obturator internus is sometimes demonstrable when the hip is flexed and internally rotated. If an inflamed appendix is in contact with the obturator internus, this manœuvre will cause pain in the hypogastrium (Zachary Cope). An inflamed appendix in contact with the bladder may cause frequency of micturition. A child sometimes postpones micturition as this causes pain (McFadden). Occasionally early diarrhœa results from an inflamed appendix being in contact with the rectum.

Post-ileal.—Although this is rare, it accounts for some of the cases of 'missed appendix'. Here the inflamed appendix lies behind the terminal ileum. It presents the greatest difficulty in diagnosis because the pain may not shift, diarrhœa is a feature, marked retching may occur and tenderness, if any, is ill-defined, though it may be present immediately to the right of the umbilicus. As the appendix irritates the lower ileum, the patient usually passes small loose stools soon after eating or drinking.

Maldescended (*subhepatic*).—The tenderness is in the subhepatic region (p. 1062). It is sometimes mistaken for acute cholecystitis.

Special Features, according to age.

Acute Appendicitis in Infants.—In infants under thirty-six months of age the incidence of perforation is over 80 per cent. (Fields), and the mortality is considerably higher than the general mortality; indeed, when acute appendicitis occurs during the first year of life, only 50 per cent. of the patients reach their first birthday. One of the reasons for the rapid onset of diffuse peritonitis is that the greater omentum, being comparatively short and undeveloped, is unable to give much assistance in localising the infection[1]. Even more important is the difficulty in arriving at an early diagnosis, and particularly in differentiating the condition from enteritis; also acute appendicitis can complicate enteritis. In addition, acute appendicitis may be associated with acute respiratory infection or one of the exanthemas.

Acute Appendicitis in Children.—It is rare to find a child with appen-

[1] The omentum is an apron of fat in the adult, but it is a mere bib in children.

James Douglas, 1675–1742. Anatomist and Obstetrician, London.
Sir Vincent Zachary Cope, 1881–1975. Surgeon, St. Mary's Hospital, London.
George Dixon Fisher McFadden, Contemporary. Late Senior Surgeon, Belfast City Hospital, Belfast, Northern Ireland.
Irving Anatole Fields, Contemporary. Associate Professor of Surgery, Loma Linda University, Los Angeles, U.S.A.

dicitis who has not vomited and they usually have complete aversion to food. In addition, they do not sleep during the attack and very often bowel sounds are completely absent in the early stages.

Acute Appendicitis in the Aged.—Gangrene and perforation occur much more frequently in elderly patients. Elderly patients with lax abdominal walls or obesity may harbour a gangrenous appendix with little evidence of it. In addition, the picture may simulate sub-acute intestinal obstruction and if enemas are given, peritonitis may be spread more widely.

Acute Appendicitis in Pregnancy

In pregnancy the appendix shifts to the upper abdomen, thus favouring peritonitis: the nearer to term, the greater the danger, even in cases without perforation. After the sixth month there is a maternal mortality of 20 per cent—ten times greater than in the first three months (Parker). As pregnancy advances the pain becomes higher and more lateral. Microscopical examination of specimens of urine will help to exclude pyelonephritis, but in doubtful cases it is best to perform early appendicectomy. The pregnant patient with acute perforated appendicitis aborts or goes into premature labour in 50 per cent of cases, while in acute non-perforated appendicitis this figure is reduced to 30 per cent.

THE DIFFERENTIAL DIAGNOSIS OF ACUTE APPENDICITIS

Although acute appendicitis is the commonest abdominal emergency the diagnosis at times can be extremely difficult. It is wise to consider carefully possible diseases of the chest, the abdomen, the pelvis, the genito-urinary system, the central nervous system, and the spine[1]. For these purposes it is helpful to visualise the body as a house (fig. 1174) and compare six parts of the house to the appropriate anatomical regions.

1. **The Attic** (i.e. *The Naso-pharynx and Thorax*)

TONSILLITIS. In children abdominal colic may arise from swallowed exudate (' Tonsil tummy ').

PNEUMONIA AND PLEURISY, especially at the right base, give rise to right-sided abdominal pain, but they are associated with an increased respiration rate, and the pain prevents deep inspiration. Pleural friction or altered breath-sounds on auscultation, and a chest X-ray may be helpful.

2. **The Upper Storey** (i.e. *Diaphragm to the level of the Umbilicus*)

PERFORATED PEPTIC ULCER (notably a perforated duodenal ulcer with duodenal contents passing along the paracolic gutter to the right iliac fossa).—As a rule there is a history of dyspepsia and a very *sudden* onset of pain which starts in the epigastrium and passes down the right paracolic gutter. In appendicitis the pain starts in the umbilical region. Rigidity

[1] " Distension, rigidity, vomiting, pain,
Are actors abdominal which often deign
To act on behalf of the chest, spine or brain,
Or general ills of which typhoid's the main."
From " The Acute Abdomen in Rhyme " by ' Zeta ' (Sir Zachary Cope)

Robert Bernard Parker, Contemporary. Obstetrician and Gynæcologist, Selly Oak Hospital, Birmingham, England.

and tenderness in the right iliac fossa are present in both conditions, but in perforated duodenal ulcer the rigidity is usually greater in the right hypochondrium. X-ray may show gas under the diaphragm (fig. 885, p. 836).

ACUTE CHOLECYSTITIS.—Murphy's sign and especially the radiation of pain through to the right scapula are important features (Boas' sign, p. 922). Retching and noisy vomiting are common. Jaundice may be present. In cases of difficulty in differentiating acute cholecystitis from acute appendicitis, the history and location of physical signs are of prime importance.

CYCLICAL VOMITING.—The patient is an infant or a young child, and there is a history of previous similar attacks. Rigidity is absent and acetone is found in the urine, but acetonuria may accompany starvation.

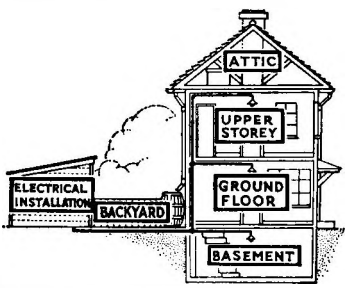


FIG. 1174.—See text. Note the water butt in the backyard (bladder).

3. **The Ground Floor** (i.e. *Umbilicus to the Brim of the Pelvis*)

ENTERO-COLITIS.—In this condition there is intestinal colic together with diarrhœa and vomiting but localised tenderness does not usually occur. There is often a history of a local epidemic. Post-ileal appendicitis may mimic this condition almost completely and it is better to look and see than to wait and see.

NON-SPECIFIC MESENTERIC LYMPHADENITIS.—The patient, usually a child, is completely free from pain in between attacks, which last a few minutes. Cervical lymph nodes may be enlarged. Shifting tenderness when the child turns on to his left side, if present, is convincing evidence. This condition is a common difficulty in children and if doubt exists a laparotomy is advisable. Some hold the view that appendicectomy may help resolution of the lymph nodes.

INTESTINAL OBSTRUCTION.—Here there is persisting colicky pain around the umbilicus with vomiting first of the stomach, then of the intestinal contents. The bowel-sounds are noisy and a plain erect X-ray shows fluid levels.

REGIONAL ILEITIS in its acute form may be indistinguishable from acute appendicitis unless a doughy mass of inflamed ileum can be felt. A history of diarrhœa suggests regional ileitis rather than appendicitis.

CARCINOMA OF THE CÆCUM (p. 1066), when obstructed, may very well mimic appendicitis in patients in the carcinoma age-group.

MECKEL'S DIVERTICULITIS (p. 997).

4. **The Basement** (i.e. *The Pelvis*)

It is in women of the child-bearing age that pelvic disease so often mimics acute appendicitis:

SALPINGITIS.—Unlike early acute appendicitis, early salpingitis should be treated by non-operative measures. The history of a vaginal discharge, of menstrual irregularities and dysmenorrhœa, or burning pain on micturition, are all helpful differential diagnostic points. The onset of symptoms usually follows a period and the pain *starts* low down and remains there (fig. 1175). On rectal or vaginal examination the enlarged tender Fallopian tubes

John Benjamin Murphy, 1857–1916. Professor of Surgery, Northwestern University, Chicago, U.S.A.
Ismar Isidor Boas, 1858–1938. Gastro-enterologist, Augusta Hospital, Berlin.
Gabriele Falloppio (Fallopius), 1523–1563. Professor of Anatomy, Surgery and Botany, Padua, Italy.

FIG. 1175.—Typical distribution of abdominal tenderness in acute salpingitis.

may be palpated. A bacteriological cervical swab is cultured and will provide information about the sensitivity of the organism. When the condition is mainly right-sided, the differential diagnosis is so difficult that it is wiser to explore the abdomen.

ECTOPIC GESTATION.—It is unlikely that a *ruptured* ectopic pregnancy, with its well-defined signs of hæmoperitoneum, will be mistaken for acute appendicitis, but the same cannot be said for a right-sided tubal abortion, or still more for a right-sided unruptured tubal pregnancy. In the latter, the signs are very similar to those of acute appendicitis, except that the pain *commences* on the right side and stays there. The pain is severe and continues unabated till operation. Usually there is a history of a missed period. The cervix is softened and often severe pain is felt when it is moved on vaginal examination. In addition, in tubal abortion, the signs of intraperitoneal bleeding usually become obvious after a while. The patient should be questioned specifically regarding referred pain in the shoulder, especially a quarter of an hour after the foot of the bed has been raised on blocks.

RUPTURED OVARIAN FOLLICLE (*syn.* mittelschmerz) occurs about the fourteenth to the sixteenth day between a period, especially in early womanhood. The signs are similar to those of very early tubal abortion, but the history of a missed period and the sign of a soft cervix are absent. There is often a history of such attacks and the condition does not progress.

TWISTED RIGHT OVARIAN CYST.—Here the pain is severe, is often referred to the loin, and is made worse when the patient rolls over. The pulse-rate progressively rises while the temperature remains normal. Sometimes the cyst is not easy to feel. If examination of the pelvis under anæsthesia is practised preparatory to laparotomy, a mistake will seldom be made.

DIVERTICULUM OF CÆCUM (solitary) (p. 1003).—This is rare. When inflamed it is indistinguishable from acute appendicitis.

5. **The Backyard** (i.e. *The Retroperitoneal Structures*)

RIGHT URETERIC COLIC.—In typical ureteric colic, pain commences in the loin and passes to the groin; this symptom, combined with the presence of urinary symptoms, serves to distinguish many cases from acute appendicitis. When ureteric colic is due to a stone in the right ureter there is often considerable tenderness in the right iliac fossa. Coughing causes pain in acute appendicitis, but not with a ureteric calculus. A plain X-ray may show a stone in the line of the right ureter and microscopy of the urine will reveal red cells. If an intravenous pyelogram is done, it may show dilatation or diminished excretion in the right urinary tract. However, if early acute retrocæcal appendicitis cannot be ruled out, it is safer to operate.

RIGHT-SIDED ACUTE PYELONEPHRITIS is accompanied and often preceded by increased frequency of micturition. It may cause difficulty in diagnosis, especially in women. The leading features are tenderness confined to the

loin, fever (temperature 39° C) and possibly rigors and pyuria. It presents special difficulty in pregnancy.

6. **The Electrical Installation** (i.e. *Central Nervous System*)

Pre-herpetic pain of the right tenth and eleventh dorsal nerves is localised over the same area as that of appendicitis. It does not shift and is associated with marked hyperæsthesia. There is no intestinal upset nor rigidity. The herpetic eruption may be delayed for thirty-six to forty-eight hours.

Tabetic crises are now rare. Severe abdominal pain and vomiting usher in the crisis. Other signs of tabes confirm the diagnosis.

Spinal conditions are sometimes associated with acute abdominal pain especially in children and the elderly. These may include Pott's disease of the spine, secondary carcinomatous deposits, senile osteo-porosis, and myelomatosis. The pain is essentially due to compression of nerve roots and may be precipitated by movement. There is rigidity of the lumbar spine and intestinal symptoms are absent. In a doubtful case spinal X-rays will often be a guide.

Other conditions to be remembered are:

The abdominal crises of porphyria which may simulate appendicular colic (p. 1060) are characterised by violent intestinal colic with constipation, and are liable to be precipitated by the administration of barbiturates, the symptoms being produced by areas of intestinal spasm. The urine of these patients is usually orange in colour, which is often dismissed as 'concentrated'. If the specimen of urine is left exposed to daylight for even a short space of time, it becomes amber in colour, particularly near the surface. There are several conclusive laboratory tests for porphyrinuria. A plain X-ray of the abdomen often displays short segments of intestinal spasm with related gaseous distension of the small and large intestine. In obscure cases of intestinal colic it is well to remember this condition, especially when it is associated with mental or neurological symptoms.[1]

'**Diabetic abdomen**' denotes the severe abdominal pain and vomiting which occasionally precedes coma. The urine should be tested in every abdominal emergency.

Acute Pancreatitis.—In early cases there may be vomiting and pain in the right iliac fossa.

PERFORATION AND GANGRENE

When perforation or gangrene occurs within twelve to twenty-four hours after the commencement of the attack, as is sometimes the case in acute appendicular obstruction, diffuse peritonitis is liable to occur. In non-obstructive appendicitis particularly, and in obstructive appendicitis when perforation or gangrene develops after a period of twenty-four hours, the resulting peritonitis often becomes localised, especially when the appendix lies in a relatively secluded portion of the peritoneal cavity.

LOCAL, DIFFUSING AND DIFFUSE PERITONITIS are discussed in Chapter 48.

THE APPENDIX MASS (*syn.* PERI-APPENDICULAR PHLEGMON)

On the third day (rarely sooner) after the commencement of an attack of acute appendicitis, a tender mass can frequently be felt in the right iliac fossa beneath some rigidity of the overlying musculature, the other quadrants of the abdomen being free from rigidity or tenderness. Alternatively, the mass is situated within the pelvis. The mass, which at this time is not yet an appendix abscess, and may never become one, is composed mainly of the greater omentum, œdematous cæcal wall, and œdematous portions of the small intestine. In its midst is a perforated or otherwise inflamed vermi-

[1] It is almost certain that the bouts of mental instability from which King George III (who died in 1820) suffered were due to porphyria.

Percivall Pott, 1714–1788. Surgeon, St. Bartholomew's Hospital, London.

form appendix. By the fourth or fifth day the mass becomes more circumscribed. As the rigidity passes off its periphery can be defined clearly and should be outlined with a skin pencil. During the ensuing days (fifth to tenth day) the swelling either becomes larger, and an appendix abscess results, or it becomes smaller, and subsides slowly as the inflammation resolves.

Appendix Abscess.—Accompanying an abscess there is variable pyrexia, but the pulse-rate is usually under 100. There is an increased leucocyte count with a relative increase of polymorphonuclear cells. To a great extent the location of the abscess is governed by the position of the appendix. Thus

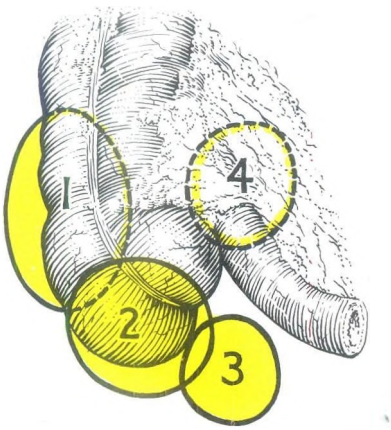


FIG. 1176.—Positions of an appendix abscess palpable from the abdomen. 1. Retrocæcal. 2. Subcæcal. 3. Retrorectus (behind the rectus abdominis muscle). 4. Post-ileal (pre-ileal occupies the same position as 4, but lies in front of the ileum).

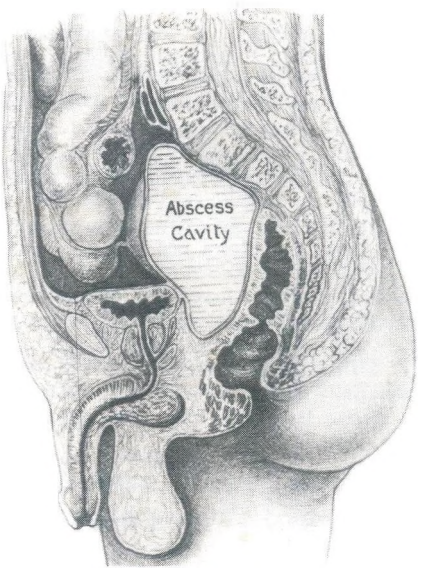


FIG. 1177.—Appendix abscess invading the pelvis. Note the relationship to the rectum.

the commonest site of the abscess is in the lateral part of the iliac fossa (extension of retrocæcal suppuration) (fig. 1176) and the second most common is in the pelvis (fig. 1177). Notwithstanding, an abscess centred beneath McBurney's point is not so unusual as the percentages of the anatomical positions of the appendix would indicate. This is because perforation often implicates the proximal half of an inflamed appendix.

Differential Diagnosis of an Appendix Mass

Carcinoma of the Cæcum.—Here the lump is rarely tender, it has appeared slowly and there is usually a secondary anæmia with occult blood in the stools. There is often progressive deterioration in health over months.

Crohn's Disease (p. 1010). The diarrhœa, loss of weight, and a lump nearer the midline merits careful barium studies of the small bowel in order to make a firm diagnosis which may otherwise be difficult. Occult blood in the stools

and a raised erythrocyte sedimentation rate may help, especially in a young adult.

Parametritis.—Here there is usually a history of recent parturition, the lump is over the medial third of the inguinal ligament and appears to be continuous with it.

Actinomycosis (p. 1015).

Tuberculosis (p. 1013).

Twisted Ovarian Cyst (p. 1064).—The essential step in diagnosis is the bimanual examination.

Iliac Lymphadenitis.—When right-sided, this condition sometimes simulates an appendix abscess. In the early stage psoas spasm is often in evidence. There is tenderness, some rigidity, and a palpable swelling above the inguinal ligament (fig. 1178). Often the inguinal lymph nodes are unaffected. Suppuration of the iliac lymph nodes leads to an extraperitoneal abscess. There is usually a focus of infection, e.g. a scratch or a blister on the skin of the lower limb.

FIG. 1178.—Tender mass connected with suppurating deep iliac lymph nodes.

TREATMENT OF ACUTE APPENDICITIS

The treatment of acute appendicitis is appendicectomy.[1] If the diagnosis is made at an early stage in the attack, and particularly in the absence of a localised mass, all are agreed that the appendix should be removed urgently.

Appendicectomy.—When the diagnosis is certain the grid-iron incision is the best one to be employed. When the diagnosis is in doubt the lower right paramedian incision is preferable because it gives good access to the pelvic organs in the female and, if necessary, it can be readily extended upwards to deal with a perforated duodenal ulcer.

The Grid-iron[2] Incision.—An adequate incision, according to the age, musculature, or obesity of the patient, is made with its centre over McBurney's point, at right angles to a line joining the anterior superior iliac spine to the umbilicus. In the subcutaneous tissues an arterial twig from the superficial circumflex iliac artery usually requires ligation. The external oblique is incised in the length of the incision. The fibres of the internal oblique and transversus abdominis are separated, and after suitable retraction the peritoneum is opened. If it is found that more room is required, it is better to cut the deeper muscles in the line of the incision thus converting the grid-iron into a Rutherford Morison incision (vide infra). The lowest mortality following appendicectomy for acute appendicitis is associated with the grid-iron incision.

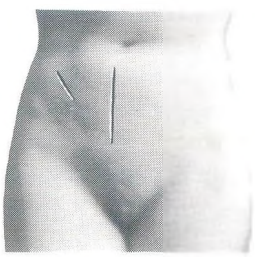


FIG. 1179.—Grid-iron incision and paramedian incision.

The paramedian incision is a vertical incision lying parallel to and 1·25 to 2·5 cm. to the right of the middle line. It commences 2·5 cm. below the level of the umbilicus and ends just above the pubis. The anterior rectus sheath is incised in the line of the incision and the rectus muscle is retracted laterally. Branches of the inferior epigastric vessels may require ligation. The transversalis fascia and the peritoneum are incised together, the peritoneal cavity being opened through the length of the

[1] The first surgeon to perform deliberate appendicectomy for acute appendicitis was Lawson Tait, in May 1880. The patient recovered. It is recorded in 1736 that Claudius Amyand successfully removed an acutely inflamed appendix from the hernial sac of a boy.

[2] Grid-iron — a frame of cross-beams to support a ship during repairs. The grid-iron incision was described first by McArthur.

Robert Lawson Tait, 1845–1899. Surgeon, Hospital for Diseases of Women, Birmingham, England.
Claudius Amyand, ?1685–1740. Surgeon, St. George's Hospital, London.
Lewis Linn McArthur, 1858–1934. Surgeon, St. Luke's Hospital, Chicago, U.S.A.
James Rutherford Morison, 1853–1939. Professor of Surgery, Durham, England.

incision. Care must be taken not to injure the bladder inferiorly. The advantages of the incision have been referred to already. Its disadvantages are (*a*) that it gives poor access to a retrocæcal appendix (it should be possible to diagnose retrocæcal appendicitis pre-operatively); (*b*) if the incision becomes infected its 'trap-door' nature harbours infection.

Rutherford Morison's incision is useful if the appendix is para- or retrocæcal and fixed. It is essentially an oblique muscle-cutting incision with its lower end over McBurney's point and extending obliquely upwards and laterally as necessary. All layers are divided in the line of the incision.

Removal of the Appendix.—It will be assumed that the abdomen has been opened by a grid-iron incision. A retractor is placed under the medial side of the peritoneum and the abdominal wall is lifted up. After removing any pus or serous exudate with a sucker, a pack is inserted into the wound on the medial side. Using a swab, the cæcum is withdrawn. A finger may be inserted into the wound to aid delivery of the appendix. Once the appendix has been delivered the cæcum is grasped by an assistant. A tissue-holding forceps (e.g. Lane's) are applied around the appendix in such a way as to encircle the organ and yet not damage it (fig. 1180). The base of the mesoappendix is clamped in a hæmostat, tied and severed. Sometimes only one such manœuvre frees the whole of the mesoappendix. When the mesoappendix is broad, the procedure must be repeated with a second, or rarely, a third hæmostat. The appendix, now completely freed, is crushed near its junction with the cæcum in a hæmostat, which is removed and reapplied just distal to the crushed portion. A catgut ligature is tied around the crushed portion close to the cæcum and an atraumatic catgut purse-string suture is inserted into the cæcum about 1·25 cm. from the base. This stitch passes through the muscle coat, especially picking up the tæniæ coli. It is left untied until the appendix has been amputated with a scalpel below the hæmostat. The stump is invaginated (fig. 1181) while the purse-string suture is tied, thus burying the appendix stump.

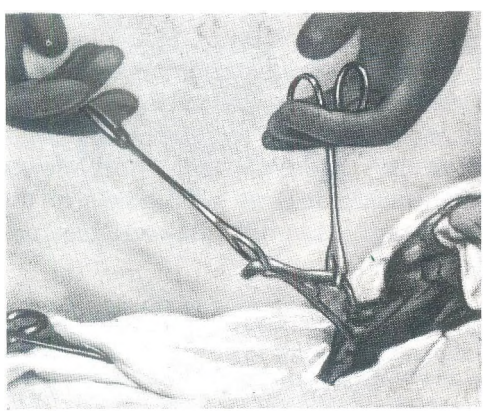


FIG. 1180.—Showing the appendix delivered and the mesoappendix displayed.

Methods to be Adopted in Special Circumstances.—When the cæcal wall is œdematous, the purse-string suture is in danger of cutting out. If the œdema is of limited extent, this can be overcome by inserting the purse-string suture into more healthy cæcal wall at a greater distance from the base of the appendix. Occasions may arise when, because of extensive œdema of the cæcal wall, it is better not to attempt invagination, in which case the stump of the appendix should be ligated and the cut surface touched with the diathermy in an attempt to reduce infection.

When the base of the appendix is inflamed, it should not be crushed, for fear of distributing infection by way of the lymphatics or bloodstream. It should be ligated close to the cæcal wall just tightly enough to occlude the lumen, after which the appendix is amputated and the stump invaginated.

Sir William Arbuthnot Lane, 1856–1943. Surgeon, Guy's Hospital, London.

Should the base of the appendix be gangrenous, neither crushing nor ligation must be attempted. Two stitches are placed through the cæcal wall close to the base of the gangrenous appendix, which is amputated flush with the cæcal wall, after which these stitches are tied. Further closure is effected by means of a second layer of interrupted sero-muscular sutures.

Retrograde Appendicectomy.—When the appendix is retrocæcal and adherent, it is an advantage to divide the base of the organ between hæmostats. After the vessels have been dealt with and the stump has been ligated and invaginated, gentle traction on the cæcum will enable the surgeon to deliver the organ which is removed from base to tip.

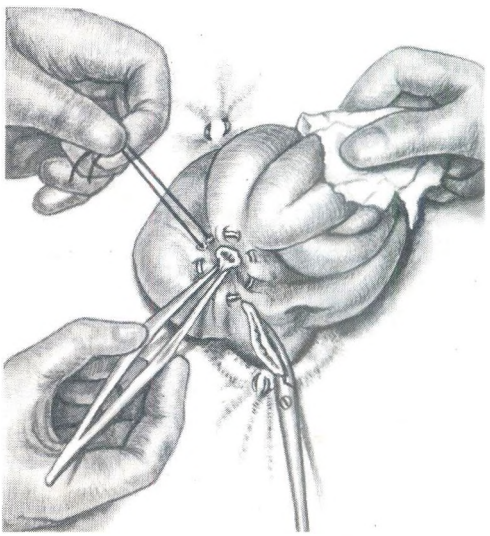


FIG. 1181.—Appendicectomy. Inverting the stump of the appendix.

Drainage of the Peritoneal Cavity.—This is usually unnecessary provided adequate peritoneal toilet has been done. If, however, there is considerable purulent fluid in the retrocæcal space or the pelvis, or if there is persistent oozing, it is wise to bring out a Penrose drain[1] through a separate stab incision.

Drainage of the Parietes.—This is indicated if there is any soiling of the wound, especially in the obese and in children.

The rule is: ' If in doubt drain, and especially the parietes '.

THE MANAGEMENT OF AN APPENDIX MASS

If an appendix mass is present and the condition of the patient is satisfactory, the standard modern treatment is conservative, i.e. the *Ochsner-Sherren* regimen. This decision is based on the fact that Nature has already localised the lesion and it is foolish to disturb these barriers. Inadvertent surgery at this time is difficult, bloody, and dangerous. It may be impossible to find the appendix and, occasionally, a fæcal fistula may form. For these reasons it is wise to observe a rigid non-operative programme, but to be prepared to operate at any time should Nature fail to control the infection.

The treatment is not merely a postponement of operation; it is not a substitute for operation, but a preparation for it—essentially a surgeon's treatment, to be undertaken only in a hospital, or a correspondingly equipped nursing home. Although the treatment should be conducted on the threshold of an operating theatre, there are circumstances—for instance, in an ill-equipped ship at sea—where conservative treatment would be less dangerous than to attempt operation.

The history is taken, and particular note is made of the number of hours since the onset. The history begins ' ten, twenty-six, fifty-five *hours* ago ', not ' last Thursday ' or ' three days ago '. The physical signs are then recorded in the clinical notes in diagrammatic form. The extent of the

[1] Penrose drain. Extremely thin (latex) rubber tubing with a gauze wick.

Charles Bingham Penrose, 1862–1925. Professor of Gynæcology, University of Pennsylvania, Philadelphia, U.S.A.
Albert John Ochsner, 1858–1925. Professor of Clinical Surgery, University of Illinois, Chicago, U.S.A.
James Sherren, 1872–1945. Surgeon, The London Hospital.

rigidity is marked by shading; the presence of a lump is drawn as near as possible to scale.

In this connection it should be noted especially that sometimes when a patient is first admitted overlying muscular rigidity renders an appendix mass indefinite, or even impalpable. In the majority of such instances, should a lump be present, if the patient is re-examined in two hours' time, lessened apprehension and the warmth of being in bed will reduce guarding of the abdominal wall sufficiently to permit the lump to be felt.

Charts.—As a routine, the pulse is recorded every hour on a special chart. Temperature is recorded every four hours. Instructions are given to the nurse to report if the patient vomits and to save the specimen for inspection. Unless the vomitus is a small quantity of clear fluid, no time should be lost in passing and retaining a transnasal gastric aspiration tube, in order to keep the stomach empty.

Diet.—Water, 30 ml. hourly, may be given by mouth. Mouth-washes are given frequently. Desire for food, usually about the fourth or fifth day, is an indication that satisfactory progress is being made and that oral feeding may be started. The first feeds should be fluids only and progression to solid food can take place over the next few days.

Intravenous fluids with fluid balance chart and daily assay of electrolytes must be instituted.

Drugs.—It should be particularly noted that no morphine or its derivatives are given in border-line cases that are being watched closely for a few hours in order to observe whether the pulse-rate and other signs are tending to settle. Once it has been decided definitely to treat the patient by conservative measures, morphine may be given. Pain, as opposed to tenderness, is very seldom complained of after the first twelve hours of the treatment.

Antibiotic therapy is, of course, employed by both schools. Parenteral ampicillin, gentamicin and metranidazole are given. When the patient is permitted to receive nourishment by mouth, the antibiotic therapy is changed to the oral route.

Bowels.—If the bowels are not opened naturally by the fourth or fifth day, a glycerine suppository will encourage normal evacuation. No purgatives of any kind are given until resolution is complete—that is, until the temperature and pulse have been normal for a week and pain and physical signs are absent.

Criteria for Stopping 'Delayed' Treatment

(1) a rising pulse-rate, (2) vomiting or copious gastric aspirate, (3) increasing or spreading abdominal pain, and, increasing size of the abscess.

A rising pulse-rate in the early stages is the most reliable single sign that it is dangerous to proceed with the delayed method.

Vomiting (or copious gastric aspirate) after the first few hours should always be regarded seriously, and this by itself may be a sufficient indication to operate.

A patient undergoing delayed treatment should not complain of pain, as opposed to tenderness, after the first six hours of such treatment. If he does, there is usually something amiss, and there is a strong indication for operation.

Contraindications to the 'Delayed' Treatment

1. The diagnosis cannot be made between acute appendicitis and some other intra-abdominal catastrophe normally requiring immediate operation.
2. The signs indicate that the inflammation is still confined to the appendix.
3. When the patient is under ten years of age (poor development of the greater omentum and early perforation of the appendix), (p. 1061).

4. When the patient is over the age of sixty-five years more than ordinary bias is directed towards immediate operation, because of the frequency of peritonitis with minimum clinical signs.

The outcome of cases suitable for delayed treatment is that 90% resolve without incident. The appendix, however, must be removed later to avoid further attacks. The originators of this treatment suggested that this should be done after an interval of three months. Present-day practice, however, favours appendicectomy as soon as convenient after *complete* resolution of the mass.

FIG. 1182.—This fourteen-year-old boy had symptoms for three days. The confines of the mass are outlined. He responded to conservative treatment and returned three months later for appendicectomy.

THE TREATMENT OF APPENDIX ABSCESS

Failure of resolution of an appendix mass usually indicates that there is pus within the mass (p. 1066).

Indications for Opening an Appendix Abscess.[1]—(1) When the swelling is not diminishing in size after the fifth day of treatment; (2) when the temperature is swinging above 37·8° C on several successive days; (3) a pelvic abscess seldom resolves—repeated rectal examinations are required to determine when it is ready for opening into the rectum (p. 971).

Opening an Appendix Abscess. — The swelling is palpated under the anæsthetic.

A retrocæcal appendix abscess should be opened extraperitoneally. An incision from 2·5 to 5 cm long, depending on the thickness of the abdominal wall, is made over the centre of the swelling, rather nearer the lateral than the medial aspect. The external oblique is incised and the fibres of the deeper muscles are divided, instead of being separated, so as to give freer exit to the contents of the abscess. When the peritoneum has been reached the extraperitoneal tissues are separated in an outward and backward direction, until the abscess cavity is entered. In cases where the abscess cavity lies at some distance from the incision, more direct drainage is afforded by a counter-incision in the flank, in which case the original incision is closed.

A subcæcal abscess can be opened in the same manner, the incision being placed nearer the anterior superior iliac spine.

A pre- or post-ileal abscess can be reached only through the peritoneal cavity. When the peritoneum has been opened, gauze packing is inserted so as to isolate the region from the general peritoneal cavity before opening the abscess.

A pelvic abscess is opened into the rectum as described on p. 971.

When it is necessary to drain an appendix abscess, no prolonged attempt should be made to perform appendicectomy unless the appendix is lying free in the abscess cavity; usually the appendix is incorporated in the walls of the abscess.

Interval Appendicectomy.—Following successful drainage of an appendix abscess, arrangements should be made for the patient to return for appendicectomy three months after the wound has healed. It is highly important to explain to the patient that drainage of an appendix abscess is no safeguard against future attacks of appendicitis. Sometimes carcinoma of the cæcum may co-exist. In the carcinoma age group, all patients should have barium studies or colonoscopy to exclude this.

[1] The first recorded operation for an appendix abscess was by Henry Hancock (1809–1880) of Charing Cross Hospital, London, in 1848.

THE PERILS OF PROLONGED ANTIBIOTIC THERAPY IN INTRA-ABDOMINAL SEPSIS

In addition to the general danger of antibiotics (p. 20) there are certain dangers peculiar to their use in intra-abdominal sepsis. These are:

1. The masking of the general signs (especially the raised temperature) of an intra-abdominal abscess behoves the clinician to make a daily abdominal and pelvic examination, lest an enlarging abscess bursts its confines, perhaps into the general peritoneal cavity.

2. Pus that has been sterilised by antibiotics remains and behaves as a foreign body, and as an irritant. For instance, cases have been reported where one or more sterile abscesses lay among the coils of small intestine, causing obscure subacute intestinal obstruction.

3. Cases have been reported whereby a pelvic abscess, by reason of antibiotic therapy, has been converted into granulation tissue, leading to a 'frozen' pelvis with consequent stricture of the rectum (Stammers).

FIG. 1183.—Following appendicectomy a sinus persisted for 6 months until this fæcolith appeared on the dressing. (*Roger Mitchell, F.R.C.S.*)

COMPLICATIONS ARISING AFTER APPENDICECTOMY FOR ACUTE APPENDICITIS

The complications vary with the degree of peritonitis that was present, and with the resistance of the patient to the infection. These complications include:

Early.—Ileus (p. 1052), Wound sepsis, Residual abscess (local, fig. 1183, pelvic, paracolic, subphrenic, pp. 971 to 973), Intestinal obstruction from adhesions (p. 1047), Fæcal fistula (p. 1025), Pylephlebitis (p. 884), Post-operative thrombosis and embolism (pp. 175 and 153), Actinomycosis (p. 1015), Pulmonary complications (pulmonary collapse or pneumonitis) (p. 749).

Late.—Intestinal obstruction from adhesions (p. 1047), Incisional hernia, Right inguinal hernia following the grid-iron incision (especially if a drain is brought through the wound) (p. 1154). Sterility in the female from 'frozen' pelvis.

★ ★ ★

It is advisable to include the following practical problem:

After an operation for acute appendicitis the condition of the patient is unsatisfactory. The temperature is swinging and the pulse rate is elevated—signs which foretell pocketing of pus. How would you investigate the case?

1. *Examine the wound and the abdominal wall* for an abscess.
2. *Consider a pelvic abscess, and perform a rectal examination* (p. 971).
3. *Palpate the left iliac fossa* for an abscess in this situation.
4. *Examine the loin* for retrocæcal swelling and tenderness.
5. *Examine the legs*—to exclude the possibility of phlebitis.
6. *Examine the conjunctivæ for an icteric tinge and the liver for enlargement, and enquire if the patient has had rigors*—pylephlebitis.
7. *Examine the lungs*—pneumonitis or collapse.
8. *Examine the urine for organisms (pyelonephritis).*
9. *In children consider tonsillitis and otitis media.*
10. *Ensure that the i.v. apparatus is sterile. If you can't be sure, change it, needle and all.*
11. Lastly, *suspect the possibility of a subdiaphragmatic abscess* (p. 972).

Francis Alan Roland Stammers, Contemporary. Emeritus Professor of Surgery, Birmingham, England.

RECURRENT ACUTE APPENDICITIS

Appendicitis is notoriously recurrent. This is perhaps the commonest form of appendicitis—mild subacute attacks which are so often attributed to 'biliousness' or a 'chill on the liver' or dyspepsia which does not respond to alkalis. The attacks vary in intensity, may occur every few months, and the majority of cases ultimately culminate in severe acute appendicitis. If careful histories are taken from patients with acute appendicitis, over two-thirds remember having had milder but similar attacks of pain. The appendix in these cases shows obliterative appendicitis (fig. 1184).

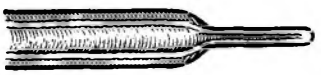


FIG. 1184.—Recurrent attacks of appendicitis lead to fibrosis spreading from the tip with gradual obliteration of the lumen.

SUBACUTE APPENDICITIS

Subacute appendicitis is but a mild form of acute appendicitis, and requires no detailed consideration.

CHRONIC APPENDICITIS

Chronic appendicitis, *per se*, does not exist. Patients labelled thus are usually examples of the recurrent form of the disease.

LESS COMMON PATHOLOGICAL CONDITIONS OF THE APPENDIX

Mucocele of the appendix may occur when the proximal end of the lumen slowly becomes completely occluded, usually by a fibrous stricture, and the pent-up secretion remains sterile. The appendix is greatly enlarged; sometimes it contains several ounces of mucus. The symptoms produced are those of mild subacute appendicitis unless infection supervenes, when the mucocele is converted into an empyema. Rupture of a mucocele of the appendix is a cause of pseudomyxoma peritonei (p. 981).

Diverticula of the Appendix.—Diverticulosis occurs once in about 200 appendices removed by operation (Edwards). These diverticula are not merely extensions of diverticulosis of the colon; some are congenital (all coats); most are acquired (no muscularis layer). Diverticula of the appendix can occur in conjunction with a mucocele. The intramural pressure rises sufficiently to cause herniation of the mucous membrane through the muscle coat at several points. More often diverticula (fig. 1185) are not found in association with a mucocele, and often there is no demonstrable obstruction to the lumen. Usually the patient gives a history of previous recurrent attacks of appendicitis. If encountered during the course of an operation for another condition, a diverticula-bearing appendix should always be removed, because if perchance such an appendix becomes the seat of inflammation, perforation will occur very easily.

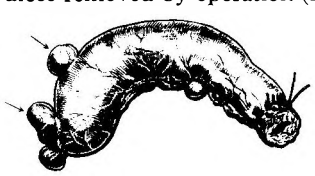


FIG. 1185.—Appendicular diverticulosis.

Intussusception of the appendix is rare and occurs mostly in childhood. It can be diagnosed only at operation. The symptoms usually are not acute. Untreated, the condition may pass on to an appendiculo-colic intussusception. The appendix may slough, and this accounts for most of the very rare cases in which the appendix is absent (p. 1056). The treatment is appendicectomy.

Endometriosis of the Appendix.—About 150 cases have been reported. The tumour—a miniature uterus in so far as endometrium is concerned—gives rise to monthly melæna. The loss of blood per rectum may be severe.

Primary Crohn's Disease (p. 1066) **of the Appendix.**—About 40 cases have been reported. Fistula has not been reported following appendicectomy in this location of the disease.

Harold Clifford Edwards, Contemporary. Consulting Surgeon, King's College Hospital, London.

Neoplasms of the Appendix

Carcinoid tumour (*syn.* Argentaffinoma), which arises in argentaffin tissue (Kulchitsky cells of the crypts of Lieberkühn) can occur anywhere in the gastro-intestinal tract, but most commonly it is situated in the vermiform appendix; indeed, the tumour is found once in about 300 to 400 appendices subjected to histological examination. Carcinoid tumours are distributed evenly among appendices removed from patients between the ages of ten and sixty years. Most of the patients are females (80 per cent).

In many instances the appendix is removed because of symptoms of subacute or recurrent appendicitis. The tumour can occur in any part of the appendix, but it frequently does so in the distal third of the organ. The neoplasm feels moderately hard, and on slitting up the appendix it can be seen between the intact mucosa and the peritoneum, replacing the muscular coats. There is no mistaking it, because it is of a bright yellow colour, due to contained lipoid. Microscopically the chief cells are spheroidal in type and contain granules that stain with ammoniacal silver salts. Unlike carcinoid tumours arising in other parts of the intestinal tract (p. 1016) in only 4 per cent. of cases does the tumour give rise to metastases, and it is most exceptional for metastases from an argentaffin carcinoma of the vermiform appendix to secrete sufficient hormone to produce the characteristic symptomatology described on p. 1017. Carcinoid tumour is ten times more common than other forms of carcinoma of the vermiform appendix. Where the mesoappendix is invaded hemicolectomy is indicated.

Primary Adenocarcinoma.—Less than 200 cases are recorded. These are mostly of the colonic type and should be treated by right hemicolectomy; as a second stage procedure if the condition is not recognised at the first operation. Less frequently, a malignant mucocele occurs and here appendicectomy alone is probably sufficient if the tumour is in the distal part of the viscus and is confined to the submucosa; otherwise right hemicolectomy is again indicated. The five-year survival rate after right hemicolectomy is of the order of 60%.

Nicholas Kulchitsky, 1856–1925. Professor of Histology, Kharkov, Ukraine, U.S.S.R.

CHAPTER 52

THE ANAL CANAL AND RECTUM

Surgical Anatomy

The anal canal commences at the level where the rectum passes through the pelvic diaphragm and ends at the anal verge.[1] The muscular junction between the rectum and anal canal can be felt with the finger as a thickened ridge—the 'ano-rectal bundle' or 'ring'.

Anal Canal Musculature:

The internal sphincter is a thickened continuation of the circular muscle coat of the rectum. This involuntary muscle commences where the rectum passes through the pelvic diaphragm, and ends at the anal orifice, where its lower border can be felt. The internal anal sphincter is 2·5 cm long and 2 to 5 mm thick. When exposed during life, it is pearly-white in colour, and its individual transversely placed fibres can be seen clearly. Spasm and contracture of this muscle play a major part in fissure and other anal affections.

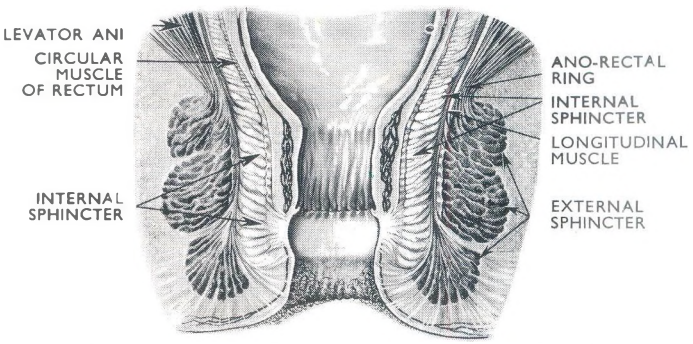


FIG. 1186.—The musculature of the anal canal. (*After Sir Clifford Naunton Morgan, F.R.C.S., London.*)

The longitudinal muscle is a continuation of the longitudinal muscle coat of the rectum intermingled with fibres from the pubo-rectalis. Its fibres fan out through the lowest part of the external sphincter, to be inserted into the true anal and perianal skin, thus constituting the **corrugator cutis ani of Ellis.**

The external sphincter, formerly subdivided into a deep, superficial, and subcutaneous portion is now considered to be one muscle (Goligher). Some of its fibres are attached posteriorly to the coccyx, while anteriorly they are inserted into the mid-perineal point in the male, whereas in the female they fuse with the sphincter vaginæ. In life the external sphincter is pink in colour, and homogeneous. Unlike the pale internal sphincter muscle, which is involuntary, the red external sphincter is composed of voluntary (somatic) muscle.

The longitudinal muscles, by traversing the internal and external sphincters to reach their insertions, serve to brace these sphincters.

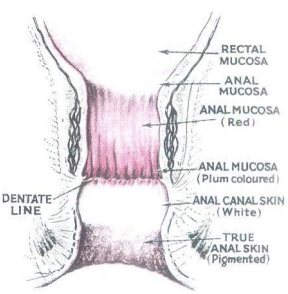


FIG. 1187. — The lining membrane of the anal canal. (*After Sir Clifford Naunton Morgan, F.R.C.S., London.*)

[1] Anal verge = the external or distal boundary of the anal canal.

George Viner Ellis, 1812–1900. Professor of Anatomy, University College, London.
John Cedric Goligher, Contemporary. Emeritus Professor of Surgery, Leeds, England.

The Mucous Membrane.—The *pink* columnar epithelium lining the rectum extends through the ano-rectal ring into the surgical anal canal. The mucosa of the surgical anal canal is attached loosely to the underlying structures, and covers the internal rectal plexus. Passing downwards where it clothes the series of 8 to 12 longitudinal folds known as the columns of Morgagni, the mucous membrane becomes cubical, and *red* in colour (fig. 1187); above the anal valves the mucous membrane becomes *plum coloured.* Just below the level of the anal valves there is an abrupt, albeit wavy, transition to squamous epithelium, which is *parchment colour.* This wavy junction constitutes the dentate line. The squamous epithelium lining the anatomical anal canal is thin and shiny, and is known as the anoderm. This squamous epithelium differs from true skin in that it has no epidermal appendages, i.e. hair, sweat glands. The anoderm passes imperceptibly into the pigmented skin of the anus. At the dentate line the anoderm is attached very firmly indeed to deeper structures.

The dentate line is a most important landmark both morphologically and surgically It represents (1) the site of fusion of the proctodæum and post-allantoic gut, and (2) the position of the anal membrane, remnants of which may frequently be seen as anal papillæ situated on the free margin of the anal valves. The dentate line separates:

Above	*Below*
Cubical epithelium	from squamous epithelium
Autonomic nerves (insensitive)	from spinal nerves (very sensitive)
Portal venous system	from systemic venous system

The anal valves of Ball are a series of transversely placed semilunar folds linking the columns of Morgagni. They lie along and actually constitute the waviness of the dentate line. They are functionless remnants of the fusion of the post-allantoic gut with the proctodæum.

The crypts of Morgagni (*syn.* anal crypts) are small pockets between the inferior extremities of the columns of Morgagni. Into several of these crypts, mostly those situated posteriorly, opens one **anal gland** by a narrow duct. This duct bifurcates, and the branches pass outward to enter the internal sphincter muscle, in 60 per cent. of people (fig. 1188). Issuing from this ampulla there are three to six tubular subbranches that extend into the inter-muscular connective tissue, where they end blindly. In some lower animals these glands secrete an odoriferous substance during the rutting season; in man their function, if any, is obscure. Some of their cells have been shown to give a positive staining reaction for mucin, but as the lining epithelium is mainly cubical, the mucus-secreting propensity of the anal glands must be extremely small. Infection of an anal gland can give rise to an abscess, and in the opinion of a number of surgeons, infection of an anal gland is the most common cause of ano-rectal abscesses and fistulæ.

FIG. 1188.—Anal gland with duct opening into a crypt of Morgagni.

FIG. 1189.—Houston's valves as seen through a sigmoidoscope.

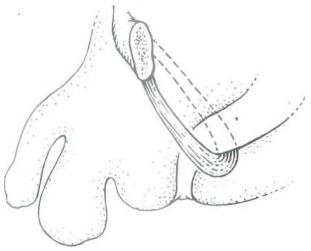


FIG. 1190.—The disposition of the pubo-rectalis muscle.

The Rectum[1].—The rectum begins above at the junction of the sigmoid colon with the rectum and ends at the ano-rectal bundle. The recto-sigmoid junction usually lies in front of the lumbar-sacral junction. It describes three lateral curves, two concave to the left (hence the *left* lateral position for sigmoidoscopy) and one concave to the right. The

[1] The word rectum means ' straight '. It is straight only in infants and the elderly.

Giovanni Battista Morgagni, 1682–1771. Professor of Anatomy, Padua, Italy.
Sir Charles Bent Ball, 1851–1916. Regius Professor of Surgery, Dublin.

relative shortness of the longitudinal muscle coat forms the **valves of Houston** that are so much in evidence in sigmoidoscopy (fig. 1189).

The ano-rectal ring marks the junction between the rectum and the anal canal. It is formed by the fusion of the pubo-rectalis muscle (fig. 1190), external sphincter, conjoined longitudinal muscle, and internal sphincter. The ano-rectal ring can be clearly felt digitally, especially on its posterior and lateral aspects. Division of the ano-rectal ring results in permanent incontinence of fæces.

THE BLOOD SUPPLY OF ANAL CANAL AND RECTUM

The superior rectal artery (fig. 1191) is the direct continuation of the inferior mesenteric artery and constitutes the chief arterial supply to the rectum. Opposite the third sacral vertebra the artery divides into a right and a left branch. About half-way down the rectum the right branch subdivides into an anterior and posterior branch. The terminal branches run straight downwards, each in a column of Morgagni.

The middle rectal artery arises on each side from the internal iliac artery and passes in close proximity to the lateral ligament of the rectum to supply the muscle coat and mucosa of the mid-rectum. Often it is a comparatively small vessel. After division of the inferior mesenteric artery, in the operation of anterior resection, the middle and inferior rectal arteries can maintain an adequate blood supply as high as the recto-sigmoid junction.

The inferior rectal artery arises on each side as a branch of the internal pudendal artery, as this artery enters Alcock's canal. Crossing the upper part of the ischio-rectal fossa, it breaks up into branches which supply the anal sphincters, anal canal, and the skin of the anal margin.

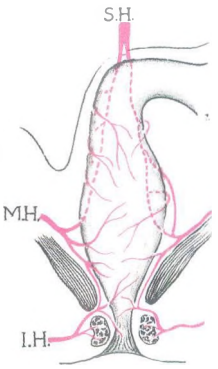


FIG. 1191.—The arterial supply of the ano-rectum S.H., superior rectal artery; M.H., middle rectal artery; I.H., inferior rectal artery.

The internal rectal venous plexus lies in the loose submucosa of the anal canal and extends from the level of the dentate line to that of the ano-rectal ring. The plexus drains into about six collecting veins which are situated in the submucosa of the rectum. About half-way up the rectum these branches pass through the rectal wall, and having reached the outside of the rectum, they unite to form the **superior rectal vein,** an important tributary of the portal vein. The **middle rectal veins** are small and drain into the internal iliac veins.

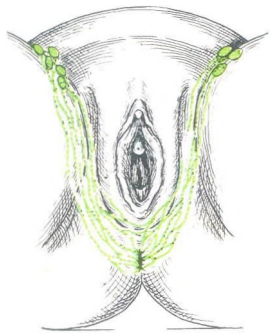


FIG. 1192.—Lymphatic trunks from the anus and the lower part of the anal canal draining into the inguinal lymph nodes. (*After M. P. C. Sappey.*)

The external rectal venous plexus lies under the skin of the anal canal below the dentate line and beneath the skin of the anal margin. Communicating veins pass from the external rectal plexus to the internal rectal plexus beneath the anoderm. The lower part of the external rectal plexus drains into the internal pudendal veins and thence into the internal iliac veins, thus providing a link between the portal and systemic venous systems.

LYMPHATIC DRAINAGE OF ANAL CANAL AND RECTUM

The lymphatics of the mucocutaneous lining intercommunicate freely with those of the muscle coats. This free anastomosis accounts for the occasional spread of malignant cells of the rectum into the inguinal lymph nodes. There are, however, three main sets of lymph nodes:

1. **Superior Rectal Lymph Nodes.**—The lymph vessels and nodes keep close to the superior rectal vessels. A special group lie just above the levator ani and close to the rectal wall in the region of the ampulla—they are the pararectal lymph nodes of Gerota. There are larger lymph nodes at the bifurcation of the superior rectal artery and also up to the origin of the inferior mesenteric artery from the aorta.

John Houston, 1802–1845. Physician, City of Dublin Hospital and Lecturer in Surgery, Dublin.
Benjamin Alcock. Professor of Anatomy, Cork, Ireland (1849–1855). He was dismissed from his post for a breach of the Anatomy Acts, and disappeared in America.
Dumitru Gerota, 1867–1939. Professor of Surgery, Bucharest, Rumania.

2. **Middle Rectal Lymph Nodes.**—Along the lateral ligaments of the rectum lie lymph vessels and nodes close to the middle rectal vessels. From here they pass to the lymph nodes around the internal iliac vessels, which are frequently involved in malignant lesions so that wide excision of the lateral ligaments is indicated.

3. **Inguinal Lymph Nodes.**—The anus and lower portion of the anal canal are drained by lymphatics which pass up to the inguinal lymph nodes (fig. 1192).

There is seldom, if ever, metastasis along the lateral or inferior lymphatic pathways from a carcinoma situated *above* the ampulla of the rectum. This justifies restorative resection in cases of carcinoma high in the rectum.

RECTAL FASCIAS

The rectum is separated in front from the bladder and prostate in the male by a condensation of connective tissue—the rectovesical fascia—known as Denonvilliers Fascia.

In both sexes the rectum is separated posteriorly from the last two sacral vertebræ by another condensation of connective tissue known as Waldeyer's Fascia.

These fascial layers are important in that they provide a temporary barrier to the direct invasion of the bladder and prostate in front and the sacrum behind by rectal carcinomas.

SYMPTOMS OF ANO-RECTAL DISEASE

In every case, careful enquiry should be made for pain, prolapse, pruritus, bleeding, discharge, alteration in bowel habit, and loss of weight.

EXAMINATION OF THE ANUS AND RECTUM

Rectal examination should be conducted systematically as follows:

Inspection, e.g. prolapsed hæmorrhoids, sentinel pile, fistula, pruritus ani.

Digital Examination.—Intraluminal, e.g. polyp, fæcal impaction.
Intramural, e.g. carcinoma.
Extramural: Male, e.g. prostate.
Female, e.g. contents of pouch of Douglas.

Discharge.—On wiping the fingerstall mucus, pus, or blood may be seen.

Proctoscopy.—(fig. 1193) is of great importance and value. The key to good visualisation is to examine the patient in the knee-elbow position (fig. 1194) or in the left lateral position with the buttocks elevated on a small cushion. This permits air to distend the rectum and causes the pelvic colon to drop away from the proctoscope so that a good view can be obtained.

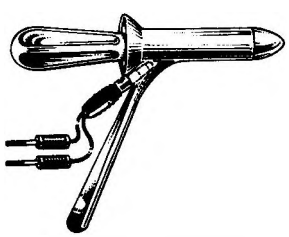


FIG. 1193.—An illuminated proctoscope.

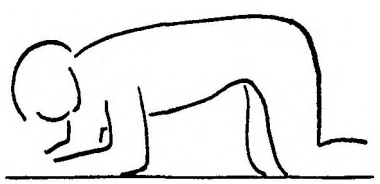


FIG. 1194.—Knee-elbow position for proctoscopy.

Charles Pierre Denonvilliers, 1808–1872. French Surgeon.
Heinrich Wilhelm Gottfried Waldeyer-Hartz, 1836–1921. Professor of Pathological Anatomy, Berlin.
James Douglas, 1675–1742. Anatomist and Obstetrician, London.

Sigmoidoscopy is necessary to visualise the whole length of the rectum and the recto-sigmoid junction. It can be carried out either in the knee-elbow or in the left lateral position.

CONGENITAL ABNORMALITIES

Early in embryonic life there is a common chamber—the cloaca—into which open the hind gut and the allantois. The cloaca becomes separated into the bladder and post-allantoic gut (rectum) by the down-growth of a septum (fig. 1195). About this time an epiblastic bud, the proctodæum, grows in towards the rectum. Normally fusion between these two structures occurs during the third month of intrauterine life.

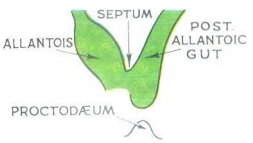


FIG. 1195.

Imperforate Anus[1].—One infant in 4,500 is born with an imperforate anus, or with imperfect fusion of the post-allantoic gut with the proctodæum. The condition is divided into two main groups—the high and the low, depending on whether the termination of the bowel is above or below the pelvic floor. The low varieties are easy to diagnose, simple to treat, and the outlook is good. The high varieties often have a fistula into the urinary tract together with a deficient pelvic floor and the prognosis is not good.

Low Abnormalities (fig. 1196).—(*a*) The *covered anus*: the underlying anal canal is covered by a bar of skin with a track running forward to the perineal raphe. The track should be opened with scissors, followed by routine

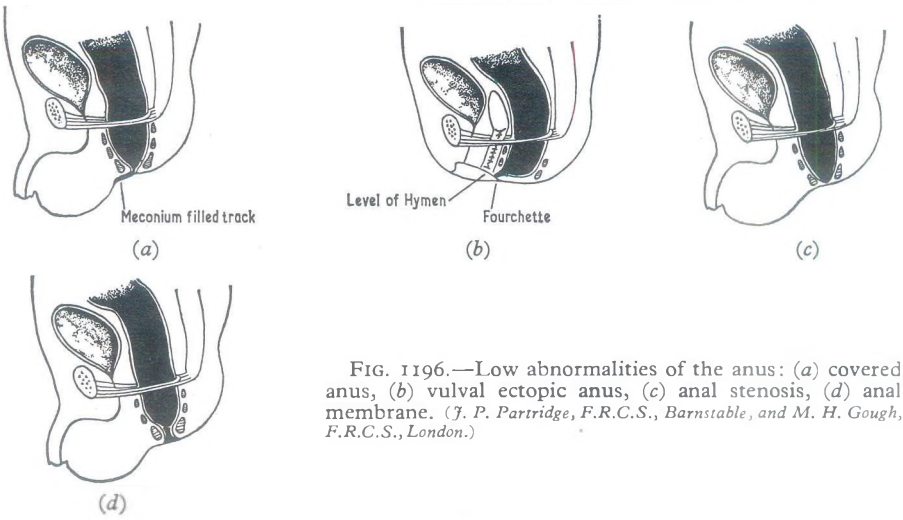


FIG. 1196.—Low abnormalities of the anus: (*a*) covered anus, (*b*) vulval ectopic anus, (*c*) anal stenosis, (*d*) anal membrane. (*J. P. Partridge, F.R.C.S., Barnstable, and M. H. Gough, F.R.C.S., London.*)

dilatation of the anus. (*b*) The *ectopic anus*: the anus is situated anteriorly and may open in the perineum in boys or more commonly in the vulva in girls, or rarely into the vagina. A plastic ' cut-back ' operation is required. (*c*) The *stenosed anus*: the anus is microscopic but careful examination usually reveals a minute opening which responds to regular dilatation. (*d*) *Membranous stenosis*: here the anus is normally sited but is covered with a thin membrane

[1] The term imperforate anus is used as a well-recognised description. Strictly it should be ' agenesis ' and ' atresia ' of the rectum and anus.

which bulges with retained meconium. It is rare, and an incision will cure the condition.

High Abnormalities (fig. 1197).—These are often associated with a fistulous connection between the blind rectal stump and the bladder, or other abnormalities of the pelvic structures. (1) *Ano-rectal Agenesis.*—A blind rectal pouch lies just above the pelvic floor—its anterior aspect in the male is attached to the bladder and often there is a recto-vesical fistula manifested by the passage of gas or meconium in the urine. In the female the fistula is usually into the posterior fornix. (2) *Rectal Atresia.*—The anal canal is normal but ends blindly at the level of the pelvic floor. The bowel also ends

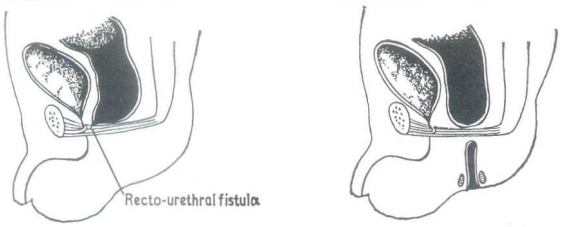


FIG. 1197.—High abnormalities: (*a*) ano-rectal agenesis with recto-urethral fistula, (*b*) rectal atresia. (*J. P. Partridge, F.R.C.S., Barnstaple, and M. H. Gough, F.R.C.S., London.*)

blindly above the pelvic floor without a fistulous opening. This anomaly is rare but must be treated by mobilisation of the rectum and excision of the stricture. After that, end-to-end anastomosis of the anus and rectum must be attempted. More conservative measures are followed by an intractable stricture. (3) *Cloaca.*—This occurs only in females and here the bowel, urinary and genital tracts all open into a common wide cavity (fig. 1195). Commonly severe malformations of the area are associated with other developmental abnormalities (e.g. tracheo-bronchial fistula).

Clinical Management.—As congenital abnormalities are frequently multiple, very careful general examination of the baby must be made to exclude any other anomalies. It is urgent and important to determine whether the abnormality is high or low and an X-ray will help.

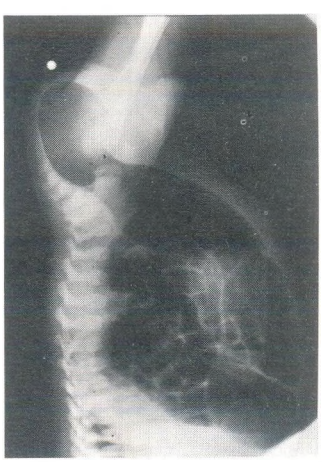


FIG. 1198.—X-ray of neonate while held upside down to show gas in rectum. Anal dimple marked by piece of lead shot. (*Graham Airth, Bristol.*)

X-ray Examination.—Six hours after birth sufficient air may have collected in the large intestine to cast an X-ray shadow. With a metal button or a coin strapped to the site of the anus, or a metal bougie inserted into the blind anal canal, the infant is held upside down for three to four minutes and radiographed in the inverted position (fig. 1198). The gas in the rectum will rise to the top and indicate the distance between the site of the metal indicator and the blind end of the rectum. If the distance is over 2·5 cm, the abnormality is ' high '. This method, though useful, is sometimes vitiated by a plug of meconium in the rectum causing an apparent gap far in excess of that actually present. It may be necessary to wait till the baby is twenty-four hours old before rectal gas appears.

Where a ' high ' lesion is suspected an effort must be made to obtain a specimen of urine—the presence of *proteus* or *pyocyaneus* usually signifies that a fistula is present. An excretory radiograph is recommended by some, even though there is a definite radiation risk. There may be gas in the bladder. A diagnostic perineal exploration is usually unwise—it may prejudice the chances of further surgery.

Treatment.—In the *low* abnormalities this is usually simple and has been outlined when each condition was described above. The *high* abnormalities present a very difficult problem and each case must be considered on its merits. On the whole, newborn babies stand surgery very well, provided compatible blood is available and a clear airway is maintained post-operatively and inhalation of vomit prevented by naso-gastric suction. The presence of other congenital abnormalities is also a most important factor to consider. The possibilities are:

1. Laparotomy, division of recto-urethral fistula, and transverse colostomy. A rectal ' pull-through ' operation can be done later.
2. Laparotomy, division of fistula, and ' pull-through ' operation in one stage.
3. Division of the fistula and rectal ' pull-down ' operation through the (*a*) perineal or (*b*) sacral route (this method is now rarely used).
4. Colostomy only (for the cloacal variety).

For the ' pull-through ' operation the lower bowel is mobilised, and a new passage is created through the pelvic floor by passing a pair of curved forceps through it, keeping close to the urethra, to the site of the future anus. This is dilated by Hegar's dilators so that the bowel can be pulled down and its mucosa stitched to the skin of the newly formed anus. (For details the reader is referred to the standard textbooks of operative surgery.)

In general, daily dilatation will be required for at least three months and it may be necessary for years.

In a high percentage of cases, imperforate anus is associated with other congenital abnormalities, especially of the urinary organs, and nearly half the deaths in cases of imperforate anus are due to other malformations.

Sacro-coccygeal teratoma, although rare, is among the most common of the large tumours seen during the first three months of life. The frequency of the pre-coccygeal region for the development of a teratoma is explained by the fact that this area is the site of the ' primitive knot ', a group of totipotent cells that retain their totipotentiality longer than any others save the sex anlage. Females are more often affected than males.

The tumour, which arises between the sacrum and the rectum, is firmly attached to the coccyx, and occasionally to the last piece of the sacrum. At the time of birth some of these tumours are huge, and in 20 per cent. of cases the infant is still-born. The tumour tends to be large (figs. 1199 and 48), but it can be small enough to pass unnoticed until it enlarges or a complication ensues. It is this variety that is prone to become malignant, usually at about ten months of age.

FIG. 1199.—Congenital sacro-coccygeal teratoma. The tumour was excised successfully. *(Maj. Gen. A. C. Bose, A.V.S.M., M.S., F.A.C.S., Senior Consultant (Surgery), Armed Forces, India.)*

Treatment.—Removal soon after birth; delay is liable to result in fatal ulceration, infection, rectal or urinary obstruction, or a malignant change.

Alfred Hegar, 1830–1914. Professor of Obstetrics and Gynæcology, Freiburg, Germany.

Operation.—Excision is undertaken through a longitudinal elliptical incision, the coccygeal attachment being left until the last. The coccyx must always be excised; occasionally the last piece of the sacrum must be removed also. There may be a fistula between the tumour and the rectum but as a rule this is small, and can be closed safely without performing a colostomy. The dead space in the pelvis is drained, the skin is united, and a pressure dressing applied.

When the operation is undertaken soon after birth, the prognosis is good.

Post-anal Dermoid.—The space in front of the lower part of the sacrum and coccyx is occupied by a soft, cystic swelling—a post-anal dermoid cyst—which is regarded as a simple form of teratoma. Hidden in the hollow of the sacrum, it is unlikely to be discovered unless a sinus communicating with the exterior is present, or develops as a result of inflammation. Such a cyst usually remains symptomless until adult life, when it is prone to become infected. Exceptionally, by its very size, it gives rise to difficulty in defæcation. The cyst is easily palpable on rectal examination.

Differential Diagnosis.—Especially in a child, an anterior sacral meningocele must be excluded. The latter enlarges when the child cries, and is frequently associated with paralysis of the lower limbs, and incontinence. When a discharging sinus is present, a post-anal dermoid will probably be mistaken for a pilonidal sinus, unless pressure over the sacro-coccygeal region with a finger in the rectum causes a flow of sebaceous material; and/or injection of lipiodol and radiography reveals a bottle-necked cyst in front of the coccyx (fig. 1200).

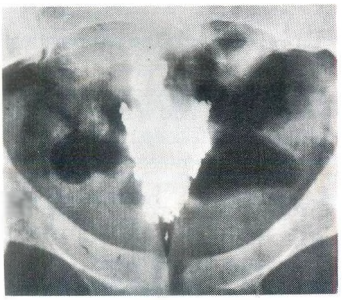


FIG. 1200.—Post-anal dermoid cyst with sinus. Injected with lipiodol. Sinus and cyst excised.

Treatment is complete excision of the cyst, and sinus if present. In the case of large cysts it is necessary to remove the coccyx in order to gain access.

Post-anal Dimple (*syn.* Fovea Coccygea).—A dimple, sometimes amounting to a short, blind pit, in the skin beneath the tip of the coccyx is noticed from time to time in the course of a clinical examination; Klass observed it in 9 per cent. of a large number of recruits. A dimple in this situation has the same significance as a dimple at other sites; it represents nothing more than a local fixation of skin by collagen fibres to underlying structures. Such a dimple is a possible starting-point of a pilonidal sinus.

PILONIDAL[1] SINUS

Ætiology.—The army of supporters of the congenital theory of the origin of pilonidal sinus has become reduced to a corporal's guard.

That, in rare instances, a sinus in the ano-coccygeal area *is* congenital must be allowed, but in these cases of proven congenital origin the sinus is not necessarily pilonidal. It could be (*a*) a sinus connected with a post-anal dermoid, referred to above, or (*b*) a sinus resulting from a persistent caudal remnant of the original neural canal. The latter occurs in the sacral rather than the coccygeal region, and is definitely connected with the spinal theca. On this account, meningitis from an extradural abscess may occur in a child.

The reasons which support the acquired theory of origin of pilonidal sinus can be summarised thus:

1. Interdigital pilonidal sinus is an occupational disease of men's hairdressers, the hair within the interdigital cleft or clefts being the customers'. Also pilonidal sinuses of the axilla and umbilicus have been reported.

[1] Appertaining to a nest of hair. (Latin—*pilus* = hair, *nidus* = nest.)

Alan Arnold Klass, Contemporary. Surgeon, Winnipeg General Hospital, Canada.

2. The age incidence of the appearance of pilonidal sinus (82 per cent. occur between the ages of twenty and twenty-nine years) is at variance with the age of onset of congenital lesions.

3. Hair follicles have never been demonstrated in the walls of the sinus.

4. The hairs projecting from the sinus are dead hairs, with their pointed ends directed towards the blind end of the sinus.

5. The disease mostly affects men, and hairy men most frequently.

6. Recurrence is common, even though adequate excision of the track is carried out.

The mode of origin of a pilonidal sinus is now believed to be as follows:

On sitting, the buttocks take the weight of the body, and move independently, or together. Hairs broken off by friction against clothing and shed short hairs, whether originating from the nape of the neck, back, or buttocks, tend to collect in the cleft of the nates and/or a post-anal dimple. Furthermore, it is suggested that the use of toilet paper may contribute to hair entangled in fæcal matter being swept into the cleft; pilonidal sinus is extremely rare in those races that employ ablution after defæcation. By reason of the shearing action of the buttocks, which is increased by sitting on a hard seat, and especially by vibration of a vehicle, loose hair travels down the intergluteal furrow, to penetrate the skin or the open mouth of a sudoriferous gland, such glands being more active in early manhood. It is not yet clear whether the initial entry of hairs through the skin is a primary event, or follows the softening of the skin due to pustular or other forms of dermatitis. Once a sinus has formed, intermittent negative pressure of the area may suck other loose hairs into the pit. So common was pilonidal sinus among jeep[1] riders in the 1939–45 war that it became known as ' jeep bottom '.

Pathology.—The sinus extends into the subcutaneous planes as an infected track. Branching side channels are not infrequent. A stratified squamous epithelial lining, of varying degrees of integrity, is found in about half the cases. Hair shafts are found either (*a*) lying loose in the sinus, (*b*) embedded in granulation tissue, or (*c*) deep in mature scar tissue in three-quarters of the cases. Foreign body giant cells are common.

Clinical Features.—There is a chronic or recurring sinus in the middle line about the level of the first piece of the coccyx (fig. 1201). Typically a tuft of hairs projects from its mouth. The discharge from the sinus or sinuses is often blood-stained, contains foul sebum, and sometimes hairs. Secondary openings may be present on either side of the midline, often far out on to the buttocks or in the perineum.

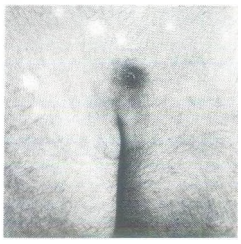


FIG. 1201.—Pilonidal sinus. (*R. S. Laurie, F.R.C.S., London.*)

As has been indicated already, symptoms usually commence during the third decade; patients presenting later in life nearly always give a history dating back to this period.

Males with this condition outnumber females by four to one, the females being on an average three years younger than the males; this corresponds to the earlier maturation of the female. The condition rarely occurs in blondes; many of the patients are exceptionally hairy and are usually obese. In spite

[1] Jeep—U.S. Army reconnaissance motor vehicle.

of the preponderance in dark-haired persons, whose hair is stiffer than the silky blonde (Oldham), the condition is practically confined to white races. The complaint is of a discharge, pain, or a tender swelling at the bottom of the spine. Even at the height of an attack of inflammation the constitutional symptoms are slight. Often there is a history of repeated abscesses in the region that have discharged spontaneously or have been incised. The primary sinus may have one, or as many as six openings, all of which are strictly in the middle line between the level of the sacrococcygeal joint and the tip of the coccyx. Unlike a fistula-in-ano, the sinus passes upwards and forwards towards the sacrum. It does not reach bone, but ends blindly near the bone. When an abscess forms it may discharge through a primary sinus; more frequently it points and bursts, or is incised to one side of the middle line (usually the left), thus forming a secondary sinus.

Conservative Treatment.—Patients reporting for the first time with mild symptoms can sometimes be cured by conservative measures, which consist of cleaning out the track, removing all hairs from the area, followed by frequent washing of the parts with a detergent and water, and applying equal parts of witch hazel (Liq. Ext. Hamamelis) and alcohol. Long sitting, e.g. driving a car, is avoided if possible. These measures, tried on a large scale in the U.S. Army, proved tolerably successful—more successful than similar attempts in civil life, because the sufferer could be relegated to duties that were unlikely to aggravate the condition.

Treatment of an Acute Exacerbation.—If rest, baths, local antiseptic dressings, and the administration of a broad spectrum antibiotic fail to bring about resolution, the abscess should be opened through a comparatively small incision. Provided all hairs and granulation tissue are removed from the abscess cavity, there is some prospect of curing the lesion (Millar). After it has been cleaned out, the track can be destroyed by careful installation of pure phenol solution (Maurice). In all other circumstances an elective operation must be planned.

Operation should be performed only when the inflammation has been controlled by the measures indicated already.

The patient is placed on the operating table, for preference in the 'jack-knife' position. Methylene blue is injected into the sinus to colour all the tracts, the nozzle of the syringe being pressed against the opening to obtain some pressure. Variations in operative technique include the following:

1. Lay open the tracts, remove all debris and hair, and suture the edges to the skin, thus marsupializing the sinus. This procedure yields a good hairless scar.
2. Excise all the tracts as stained by the blue dye and, after securing haemostasis by diathermy and catgut ligature, pack the wound. The following day the whole dressing is removed, and daily baths and moist dressings are instituted until the wound heals by granulation. Epithelialisation can sometimes be speeded up by skin grafting.
3. Excise all the tracts, secure haemostasis as above, and, using sutures coapt the subcutaneous fat and skin very accurately and institute a drain and suction (i.e. Redivac) for 48 hours to remove blood and serum. In cases of extensive sinus formation primary cover may be achieved by rotating a flap of skin and fat.

Immediately after operation the patient should avoid sitting on the wound. Subsequently, the scar may require protection from further incursions of hair by selective hair-trimming.

James Bagot Oldham, 1899–1977. Surgeon, United Liverpool Hospitals, Liverpool, England.
Douglas Malcolm Millar, Contemporary. Surgeon, Essex County Hospital, Colchester.
Brian Armstead Maurice, Contemporary. Surgeon, Tunbridge Wells, Kent.

Recurrent Pilonidal Sinus.—Three possibilities account for this disappointment. (1) A diverticulum of the main channel has been overlooked at the primary operation. (2) New hairs enter the skin or the scar. (3) When the natal fold is deformed by scarring, the least trauma causes tearing of the scar, and the resulting crevice becomes contaminated with coliform and cutaneous bacteria.

FIG. 1202.—Rectal impalement by a broken shooting-stick.

INJURIES

The rectum or anal canal may be injured in a number of ways, all uncommon.

1. By falling in a sitting posture on to a spiked or blunt-pointed object. The up-turned leg of a chair, handle of a broom, floor-mop, pitch-fork, or a broken shooting-stick (fig. 1202) have all resulted in rectal impalement.
2. By the fœtal head during childbirth.
3. During the administration of an enema by a syringe fitted with a bone, glass, or vulcanite nozzle.
4. During sigmoidoscopy, usually when examining a patient suffering from ulcerative procto-colitis or amœbic dysentery. Sigmoidoscopy performed under general anæsthesia is especially dangerous.
5. ' Split Perineum '. A lacerated wound of the perineum, involving the anal canal, is an occasional pillion-riding accident.
6. Compressed-air rupture (p. 995).

Diagnosis.—When there is a history of rectal impalement, the first interrogation should be, ' Has the patient passed urine since the accident? ' The anus having been inspected, the abdomen should be palpated. If rigidity or tenderness is present, early laparotomy is imperative. Prior to the operation an urethral catheter is passed. If the urine is blood-stained and/or the quantity recovered is unexpectedly small, it is wise to suspect ruptured bladder or urethra (pp. 1225 and 1288).

Treatment.—After the patient has been anæsthetised, the rectum is examined with a finger and a speculum, especial attention being directed to the anterior wall. Left lower laparotomy is then performed. If an intraperitoneal rupture of the rectum is found, the perforation is closed with sutures. Should blood be present beneath the pelvic peritoneum, it is necessary to mobilise the recto-sigmoid, which allows the rectum to be drawn upwards, thus permitting the perforation below the pelvic diaphragm to be closed securely. A perforation in the bladder also can be sutured *via* this avenue. After closing the laparotomy wound, a left iliac colostomy is performed through a separate grid-iron incision. In cases where the bladder has been injured a self-retaining urethral catheter is placed in position.

If the rectal injury is below the pelvic floor, wide drainage from below is indicated. A 'protective' colostomy is advisable. Care must be taken to preserve sphincter function during the debridement of the perineal wounds. Antibiotic cover should be provided against both aerobic and anaerobic organisms.

FIG. 1203.—Pepper-pot in the rectum. On removal it was found to be inscribed 'A present from Margate '! *(Dr. L. S. Carstairs, Royal Northern Hospital, London.)*

FOREIGN BODIES IN THE RECTUM

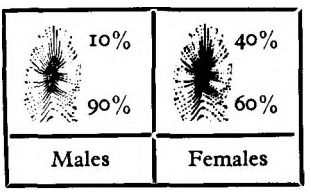
The variety of foreign bodies which have found their way into the rectum is hardly less remarkable than the ingenuity displayed in their removal (fig. 1203). A turnip has been delivered *per anum* by the use of obstetric forceps. A stick firmly impacted has been withdrawn by inserting a gimlet into its lower end. A tumbler, mouth looking downwards, has been extracted by filling the interior with a wet plaster of Paris bandage, leaving the end of the bandage protruding, and allowing the plaster to set.

If insurmountable difficulty is experienced in grasping any foreign body in the rectum, a left lower laparotomy is necessary, which allows that object to be pushed from above into the assistant's fingers in the rectum. If there is considerable laceration of the mucosa a temporary colostomy is advisable.

ANAL FISSURE (*syn.* FISSURE-IN-ANO)

Definition.—An elongated ulcer in the long axis of the lower anal canal.

Location.—The site of election for an anal fissure is the mid-line posteriorly. The next most frequent situation is the mid-line anteriorly. The relative distribution of fissure in these two sites is ——————————→



Ætiology.—The cause of anal fissure, and particularly the reason why the midline posteriorly is so frequently affected, is not completely understood. A probable explanation is as follows: the posterior wall of the rectum curves forwards from the hollow of the sacrum to join the anal canal, which then turns sharply backwards. During defæcation the pressure of a hard fæcal mass is mainly on the posterior ano-rectal angle (fig. 1204) in which event the overlying epithelium is greatly stretched, and, being relatively unsupported by muscle, is placed in a vulnerable position when a scybalous mass is being expelled. Possibly some cases are due to tearing down of an anal valve of Ball. As shown by the above percentages, an anterior anal fissure is much more common in women, particularly in those who have borne children. This can be explained by the lack of support of the anal mucous membrane by a damaged pelvic floor.

One cause of anal fissure is certain—an incorrectly performed operation for hæmorrhoids in which too much skin is removed. This results in anal stenosis and tearing of the stricture when a hard motion is passed.

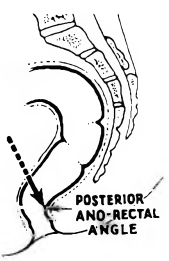


FIG. 1204.—Probable mechanism of tearing of the anal mucosa in the case of a posterior fissure.

Pathology.—An anal fissure is either acute or chronic. The upper internal end of the fissure stops at the dentate line. Because the fissure occurs in the stratified sensitive epithelium of the lower half of the anal canal, pain is the most prominent symptom (*vide infra*).

Acute anal fissure is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory induration or œdema of its edges. There is always spasm of the anal sphincter muscle.

Chronic anal fissure is characterised by inflamed indurated margins, and a base consisting of either scar tissue or the lower border of the internal sphincter muscle. The ulcer is canoe-shaped, and at the inferior extremity frequently there is a tag of skin, usually œdematous. This tag is known picturesquely as a sentinel pile—'sentinel' because it guards the fissure. There is usually spasm of the involuntary musculature of the internal sphincter. In long-standing cases this muscle becomes organically contracted by infiltration of fibrous tissue. Infection is common and may be severe, ending in abscess formation. A cutaneous fistula may follow.

Chronic fissure-in-ano may have a specific cause—often a granulomatous infection, *e.g.* Crohn's disease or syphilis. Biopsy examination is advisable of any tissue removed at operation for a chronic fissure. Specific fissures

of this type are often less painful than the appearances of the lesion would suggest.

Clinical Features.—The condition is more common in women, and generally occurs during the meridian of life. It is uncommon in the aged, because of muscular atony; on the other hand anal fissure is not rare in children, is sometimes encountered during infancy, and may cause acquired mega-colon (p. 994).

Pain is *the* symptom—sharp agonising pain starting during defæcation, often overwhelming in intensity and lasting an hour or more. As a rule it ceases suddenly, and the sufferer is comfortable until the next action of the bowel. Periods of remission occur for days or weeks. The patient tends to become constipated rather than go through the agony of defæcation.[1]

Stools are frequently streaked with blood.

Discharge.—A slight discharge accompanies fully established cases.

On Examination.—In cases of some standing, a sentinel skin tag can usually be displayed. This, together with a typical history and a tightly closed, puckered anus, is almost pathognomonic of the condition. By gently parting the margins of the anus, the lower end of the fissure can be seen.

Because of the intense pain it causes, digital examination of the anal canal should not be attempted at this stage unless (*a*) the fissure cannot be seen or (*b*) it seems imperative to exclude some intrarectal condition. In these circumstances the local application of a surface anæsthetic such as 5 per cent. xylocaine on a pledget of cotton-wool, left in place for about five minutes, will enable the necessary examination to be made. In early cases the edges of the fissure are impalpable; in fully established cases a characteristic crater which feels like a vertical buttonhole can be palpated. Proctoscopy is necessary. A general anæsthetic may be needed.

Differential Diagnosis:

Carcinoma of the anus in its very early stages easily simulates a fissure. If real doubt exists, the lesion must be excised under general anæsthesia and submitted to histological examination.

Multiple fissures in the peri-anal skin are seen as a complication of pruritus ani (p. 1097) and procto-colitis (p. 1102).

Anal chancre (fig. 1205) is becoming more common and may present as a *painful* rather than a painless ulcer (p. 45). The serous discharge contains spirochætes. A glass pipette is used to aspirate a few drops which are placed on a slide for examination by dark-ground illumination. Lubricating gel from the finger-stall may prevent adequate aspiration of serum from a chancre.

Tuberculous ulcer (p. 66) has an undermined edge.

Proctalgia Fugax (p. 1089) causes severe episodic pain.

FIG. 1205.—Anal chancre.

Treatment.—The pain of an anal fissure is so great that usually the patient demands relief, and consequently many patients with an acute fissure present

[1] One patient accustomed himself to take a generous dose of senna on Saturday night, and retire to the toilet on Sunday morning with a bottle of whisky and the newspaper.

early. The object of all treatment for this condition is to obtain *complete relaxation* of the internal sphincter. Provided the complications are dealt with, the fissure will slowly heal as soon as all spasm has disappeared.

Conservative Treatment.—In cases where the fissure is acute and superficial and where the inflammation is minimal, simple conservative measures will usually give relief. Xylocaine 5 per cent. in a water-soluble lubricant is introduced with a fine nozzle into the anal canal. After waiting a few minutes for the surface anæsthetic to act, relaxation may be sufficient to permit the passage of a well-lubricated finger into the canal. Following this a small anal dilator (fig. 1206) may be passed and, if the anæsthesia is adequate, it may be possible to introduce the largest dilator. Anal dilators are commonly made in three sizes and it may not be possible to use the largest dilator until several days have passed. The patient is supplied with xylocaine lubricant and instructed to pass a dilator twice a day for a month, by which time the fissure is usually healed. Laxatives are prescribed to ensure that the motions are soft, but the stools should not be made watery: Celevac 10 ml b.d. gives a soft stool of good bulk, which is ideal.

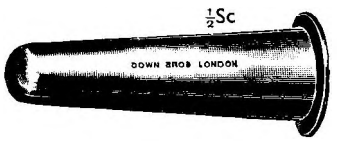


FIG. 1206.—St. Mark's Hospital (London), anal dilator.

Operative Measures

The simplest procedure is wide dilatation of the sphincter. Under general anæsthesia the index and middle finger of each hand are inserted simultaneously into the anus and pulled apart to give maximal dilatation. The patient can go home the same day but should be warned that there may be some fæcal incontinence lasting possibly for a week or ten days.

Should these measures prove ineffective, or if the fissure is chronic with fibrosis, a skin tag, or a mucous polypus, then operative measures are advisable. General anæsthesia is best, though some surgeons use a local anæsthetic in the form of xylocaine or proctocaine introduced into the ischio-rectal fossa on each side, in order to anæsthetise the nerves passing towards the rectum.

(i) *Dorsal fissurectomy and sphincterotomy.* With the patient in the lithotomy position a well greased Sims' speculum is introduced. This will permit a very good view of the fissure which becomes stretched to show the fibres of the internal sphincter running transversely in its floor. The essential part of the operation is to divide all of these transverse fibres so that the floor of the fissure is completely smooth. If a sentinel pile is present, this is excised. The ends of the divided muscle retract and a smooth wound is left. The after-treatment consists of attention to bowels, a daily bath, and the passage of an anal dilator until the wounds have healed, which usually takes about three weeks. Despite the presence of the wound there is little or no pain and the final results are excellent. The disadvantage of this operation is the prolonged healing time—usually not less than three weeks and often longer—and occasionally a mild, persistent and permanent mucus discharge.

(ii) *Lateral anal sphincterotomy* (Notaras). In this operation, the internal sphincter is divided away from the fissure itself—usually either in the right or the left lateral positions. The procedure can be done by an open or a closed method. Healing is usually complete within three weeks. The operation is more successful for acute than chronic fissures. 75% of cases are suitable for treatment by this method. The patients are able to leave hospital within three or four days, and the procedure can be done as an Out-Patient under local anæsthesia by an experienced surgeon.

HYPERTROPHIED ANAL PAPILLA

Anal papillæ occur at the dentate line, and are remnants of the ectodermal membrane that separated the hind-gut from the proctodæum. As these papillæ are present in

James Marion Sims, 1813–1883. Gynæcological Surgeon, State Hospital for Women, New York, U.S.A.
Mitchell James Notaras, Contemporary. Surgeon, Barnet General Hospital, London.

fully 60% of patients examined proctologically, they should be regarded as normal structures. Anal papillæ can become elongated, as they frequently do in the presence of an anal fissure. Occasionally an elongated anal papilla may be the cause of pruritus. An elongated anal papilla associated with pain and/or bleeding at defæcation, is sometimes encountered in infancy. Hæmorrhage into a hypertrophied anal papilla can cause sudden rectal pain. A prolapsed papilla may become nipped by contraction of the sphincter mechanism after defæcation. Occasionally a red œdematous papilla is encountered with local pain and a purulent discharge from the associated crypt. This condition of 'cryptitis' is more frequently seen abroad, but may be cured by laying open the mouth of the infected anal gland and removing the papilla.

Treatment.—Using a slotted proctoscope, elongated papillæ without hæmorrhoids should be crushed and excised after injecting the base with local anæsthetic. When elongated papillæ complicate internal hæmorrhoids, this is an indication for operative treatment of the hæmorrhoids, as well as excision of the elongated papillæ.

PROCTALGIA FUGAX

This disease is characterised by attacks of severe pain arising in the rectum, recurring at irregular intervals and apparently unrelated to organic disease. The pain is described as cramp-like, often occurs when the patient is in bed at night, lasts only a few minutes, and disappears spontaneously. It may follow straining at stool, sudden explosive bowel action or ejaculation. It seems to occur more commonly in patients suffering from anxiety or undue stress and also it is said to afflict young doctors. The pain may be unbearable—it is possibly due to segmental cramp in the pubo-coccygeus muscle. It is unpleasant, incurable, but fortunately harmless and gradually subsides.

HÆMORRHOIDS[1, 2] (*syn.* PILES[3])

Hæmorrhoids are dilated veins occurring in relation to the anus, and originating in the subepithelial plexus formed by radicles of the superior, middle, and inferior rectal veins.

Such hæmorrhoids may be **external** or **internal**—external or internal to the anal orifice. The external variety are covered by skin, while the internal variety lie beneath the mucous membrane. When the two varieties are associated, they are known as **intero-external** hæmorrhoids.

Hæmorrhoids may be '*symptomatic*' of some other condition, and this important fact must be remembered when they are found. Symptomatic hæmorrhoids may appear (*a*) in *carcinoma of rectum*. This, by compressing or causing thrombosis of the superior rectal vein, gives rise to hæmorrhoids (fig. 1207) sufficiently often to warrant examination of the rectum and the recto-sigmoid junction for a neoplasm in every case of hæmorrhoids. (*b*) During *pregnancy*. Pregnancy piles are due to compression of the superior rectal veins by the pregnant uterus and the effect of progesterone on the smooth muscle in the walls of the veins. (*c*) From *straining at micturition* consequent upon a stricture of the urethra—or an enlarged prostate. (*d*) From chronic constipation.

N.B. Contrary to the usual belief, in one hundred and twenty-eight consecutive cases of portal hypertension,

FIG. 1207.—Carcinoma of the rectum associated with hæmorrhoids. A not infrequent diagnostic pitfall.

[1] 'The common people call them piles, the aristocracy call them hæmorrhoids, the French call them figs—what does it matter so long as you can cure them?'—John of Arderne, 1370.
[2] Greek—*haima* = blood, *rhoos* = flowing. [3] Latin—*pĭla* = a ball.

John of Arderne, ?1306–?1390. The first English surgeon. He practised in Newark and later in London.

Macpherson did not encounter a single example of hæmorrhoids that could be attributed to portal cirrhosis, although bleeding œsophageal varices often complicate portal hypertension (p. 897).

The great majority of hæmorrhoids are not symptomatic, and the description that follows concerns hæmorrhoids that are *not* a manifestation of some underlying cause.

INTERNAL HÆMORRHOIDS

Internal hæmorrhoids, which include intero-external hæmorrhoids, are exceedingly common. Essentially the condition is a varicosity of the internal rectal plexus, but because of the communication between the internal and external rectal plexuses, if the former becomes varicose, the latter is liable to become involved also.

Ætiology: *Hereditary.*—The condition is so frequently seen in members of the same family that there must be a predisposing factor, such as a congenital weakness of the vein walls or an abnormally large arterial supply to the rectal plexus.

Varicose veins of the legs and hæmorrhoids often occur concurrently.

Morphological.—In quadrupeds, gravity aids, or at any rate does not retard, return of venous blood from the rectum. Consequently venous valves are not required. In man, the weight of the column of blood unassisted by valves produces a high venous pressure in the lower rectum, unparalleled in the body. Such a pressure may be resisted by a cushioning effect of the columns of mucous membrane (Thomson). Except in a few fat old dogs, hæmorrhoids are exceedingly rare in animals.

Anatomical.—(1) The collecting radicles of the superior rectal vein lie unsupported in the very loose submucous connective tissue of the rectum. (2) These veins pass through muscular tissue and are liable to be constricted by its contraction during defæcation. (3) The superior rectal veins, being tributaries of the portal vein, have no valves.

Exciting Causes.—Straining accompanying constipation or that induced by overpurgation is considered to be a potent cause of hæmorrhoids. Less often the diarrhœa of enteritis, colitis, or the dysenteries aggravates latent hæmorrhoids.

Pathology.—Internal hæmorrhoids in association with the terminal divisions of the superior rectal artery are arranged in three groups at 3, 7, and 11 o'clock with the patient in the lithotomy position (fig. 1208).[1] This distribution is due to the arterial supply of the anus whereby there are two subdivisions of the right branch of the superior rectal artery, but the left branch remains single. In between these three primary hæmorrhoids there may be smaller secondary hæmorrhoids. Each principal hæmorrhoid can be divided into three parts:

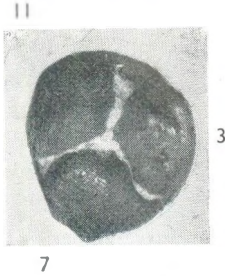


FIG. 1208.—Proctoscopy third-degree hæmorrhoids. Note their 3, 7, and 11 o'clock positions. (*E. T. C. Milligan, F.R.C.S., London.*)

The pedicle is situated in the rectum just above the ano-rectal ring. As seen through a proctoscope, it is covered with pale pink mucosa through which large tributaries of the superior rectal vein can be seen. Occasionally a pulsating artery can be felt in this situation.

The internal hæmorrhoid, which commences at the ano-rectal ring and ends at the dentate line. It is bright red or purple, and covered by mucous membrane.

[1] Lithotomy position = the position in which patients used to be put for the classical operation of 'cutting' for bladder stone via the urethral or the perineal route (see p. 1120).

Archibald Ian Stewart Macpherson, Contemporary. Surgeon, Royal Infirmary, Edinburgh.
William Hamish Fearon Thomson, Contemporary. Surgeon, Gloucestershire Royal Hospital, Gloucester, England.

An external associated hæmorrhoid lies between the dentate line and the anal margin. It is covered by skin, through which blue veins can be seen, unless fibrosis has occurred. This associated hæmorrhoid is present only in well-established cases.

Entering the pedicle of each internal hæmorrhoid is a terminal branch of the superior rectal artery. Very occasionally there is a hæmangiomatous condition of this artery—an ' arterial pile '.

Clinical Features.—*Bleeding*, as the name hæmorrhoid implies, is the principal and earliest symptom. At first the bleeding is slight; it is bright red and occurs during defæcation (a ' splash in the pan '), and it may continue thus for months or years. Hæmorrhoids that bleed but do not prolapse outside the anal canal are called *first-degree hæmorrhoids.*

Prolapse is a much later symptom. In the beginning the protrusion is slight and occurs only at stool, and reduction is spontaneous. As time goes on the hæmorrhoids do not reduce themselves, but have to be replaced digitally by the patient. Hæmorrhoids that prolapse on defæcation but return or need to be replaced manually and then stay reduced are called *second-degree hæmorrhoids.* Still later, prolapse occurs during the day, apart from defæcation, when the patient is tired or exerts himself. Hæmorrhoids that are permanently prolapsed are called *third-degree hæmorrhoids.* By now, the hæmorrhoids have become a source of great discomfort and cause a feeling of heaviness in the rectum.

Discharge.—A mucoid discharge is a frequent accompaniment of prolapsed hæmorrhoids. It is composed of mucus from the engorged mucous membrane, sometimes augmented by leakage of ingested liquid paraffin. *Pruritus* will almost certainly follow this discharge.

Pain is absent unless complications supervene.

Anæmia can be caused very rarely by persistent profuse bleeding from hæmorrhoids.

On inspection there may be no evidence of internal hæmorrhoids. In more advanced cases redundant folds or tags of skin can be seen in the position of one or more of the three primary hæmorrhoids. When the patient strains, internal hæmorrhoids may come into view transiently, or if they are of the third degree they prolapse and remain prolapsed.

Digital examination.—Internal hæmorrhoids cannot be felt unless they are thrombosed.

Proctoscopy.—A proctoscope is passed to its fullest extent and the obturator is removed. The instrument is then slowly withdrawn. Just below the ano-rectal ring internal hæmorrhoids, if present, will bulge into the lumen of the proctoscope.

Complications:

Profuse hæmorrhage is not rare. Most often it occurs in the early stages of the second degree. The bleeding occurs mainly externally, but it may continue internally after the bleeding hæmorrhoid has retracted or has been returned. In these circumstances the rectum is found to contain blood.

Strangulation.—One or more of the internal hæmorrhoids prolapse and become gripped by the external sphincter. Further congestion follows because the venous return is impeded. Second-degree hæmorrhoids are most often complicated in this way. Strangulation is accompanied by considerable pain, and is often spoken of by the patient as an 'acute attack of piles'.[1] Unless the internal hæmorrhoids can be reduced within an hour or two, strangulation is followed by thrombosis.

FIG. 1209. — An attack of piles. Prolapsed strangulated piles, as commonly seen, on the left. A less common mass on the right with a fibro-fatty covering.

Thrombosis.—The affected hæmorrhoid or hæmorrhoids become dark purple or black (fig. 1209) and feel solid. Considerable œdema of the anal margin accompanies thrombosis. Once thrombosis has occurred the pain of strangulation largely passes off, but tenderness persists.

Ulceration.—Superficial ulceration of the exposed mucous membrane often accompanies strangulation with thrombosis.

Gangrene occurs when strangulation is sufficiently tight to constrict the arterial supply of the hæmorrhoid. The resulting sloughing is usually superficial and localised. Occasionally a whole hæmorrhoid sloughs off, leaving an ulcer which heals gradually. Very occasionally massive gangrene extends to the mucous membrane within the anal canal and rectum.

Fibrosis.—After thrombosis, internal hæmorrhoids sometimes become converted into fibrous tissue. The fibrosed hæmorrhoid is at first sessile, but by repeated traction during prolapse at defæcation, it becomes pedunculated and constitutes a fibrous polypus that is readily distinguished by its white colour from an adenoma, which is bright red. Fibrosis following transient strangulation commonly occurs in the subcutaneous part of a primary hæmorrhoid. Fibrosis in an external hæmorrhoid favours prolapse of an associated internal hæmorrhoid.

Suppuration is uncommon. It occurs as a result of infection of a thrombosed hæmorrhoid. Throbbing pain is followed by perianal swelling, and a perianal or submucous abscess results.

Pylephlebitis (*syn.* Portal Pyæmia).—Theoretically, infected hæmorrhoids should be a potent cause of portal pyæmia and liver abscesses (p. 884). Although cases do occur from time to time, this complication is surprisingly infrequent.

TREATMENT OF INTERNAL HÆMORRHOIDS

Non-operative treatment is recommended when the hæmorrhoids are a symptom of some other condition or disease except, of course, when a carcinoma is present. The bowels are regulated by Isogel, and if necessary a small dose of Senokot at night. Various proprietary creams can be inserted into the rectum from a collapsible tube fitted with a nozzle, at night and before defæcation. Suppositories are also useful. **In cases of inflamed and permanently prolapsed hæmorrhoids** with a patulous anus, once the œdema has been reduced by repeated dressings of glycerine and tannic acid B.P. (Eisenhammer) or Lotio Plumbi B.P., surgery offers the only hope of permanent cure. However, a severe stretching of the anal sphincter ('manual dilatation of the anus') is frequently successful in relieving symptoms even in advanced cases of piles, probably by preventing congestion of the hæmorrhoidal veins (Lord). This procedure is unsuitable for cases with a patulous sphincter. It requires a general anæsthetic and after-care by

[1] An 'acute attack of piles' also embraces a thrombotic pile (p. 1096) or an inflamed anal skin tag.

Stephen Eisenhammer, Contemporary. Rectal Surgeon, Johannesburg, South Africa.
Peter Herent Lord, Contemporary. Surgeon, Wycombe General Hospital, High Wycombe, Bucks, England.

passing a special dilator to maintain sphincter relaxation; but the patient is usually hospitalised for less than 24 hours.

Active Treatment.—This consists of injection or treatment by elastic band applications to the base of each hæmorrhoid or formal operation, each with specific indications. Treatment should not be withheld because the patient is elderly or infirm.

Injection Treatment[1].—*Indications.*—This is ideal for first-degree internal hæmorrhoids which bleed. Early second degree hæmorrhoids are often cured by this method but a proportion relapse.

Technique.—The patient should have an empty rectum, but no special preparation is necessary. A proctoscope is introduced, and the hæmorrhoids are displayed. The proctoscope is introduced farther in until the hæmorrhoid has almost disappeared from the lumen and only its upper end is visible. The injection is made at this point above the main mass of each hæmorrhoid (fig. 1210) into the submucosa at or just above the ano-rectal ring. Using Gabriel's syringe (fig. 1211) with the bevel of the needle directed towards the rectal wall, from 3 to 5 ml. of 5 per cent. phenol in almond oil is injected. The injection should produce elevation and pallor of the mucosa. The solution spreads in the submucosa upwards to the pedicle, and downwards to the internal hæmorrhoid and to secondary hæmorrhoids if present, but it is prevented by the intermuscular septum from reaching the external hæmorrhoid. There is a slight, transient bleeding from the point of puncture. The injection is painless, but a dull ache is common for a few hours. There is no special after-treatment. If there is only one hæmorrhoid present, it may be cured by one injection; if all three hæmorrhoids are equally enlarged, each is injected at the same session. Often three sessions at six-weekly intervals are required. Care should be taken not to inject into the prostate anteriorly, for the resulting prostatitis can be crippling.

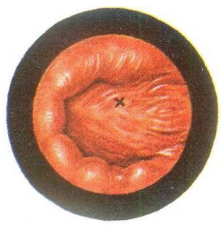


FIG. 1210.—Correct site for injecting a hæmorrhoid. (*After W. B. Gabriel, F.R.C.S., London.*)

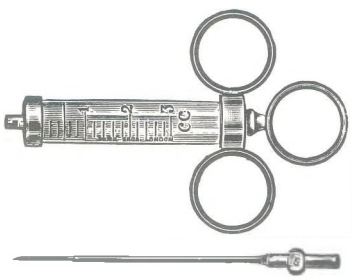


FIG. 1211.—Gabriel's syringe for injecting hæmorrhoids.

Banding Treatment (Barron).—For second-degree hæmorrhoids which are too large for successful handling by injections, treatment is available by slipping tight elastic bands on to the base of the pedicle of each hæmorrhoid with a special instrument (fig. 1212). The bands cause ischæmic necrosis of the piles, which slough off within a few days. The procedure should be painless if done properly, and can be

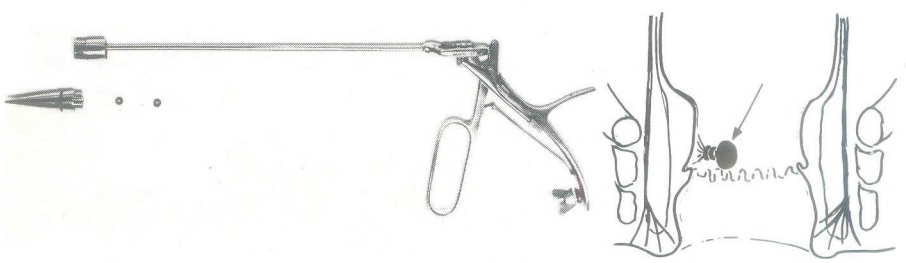


FIG. 1212.—Barron's banding apparatus with appearance of a typical 'banded' hæmorrhoid.

[1] In 1871, Mitchell of Clinton, Illinois, first used carbolic acid for injecting hæmorrhoids. Itinerant irregular practitioners exploited the method.

William Bashall Gabriel, 1893–1975. Surgeon, Royal Northern Hospital, London.
John Barron, Contemporary. Surgeon, Chicago, Illinois, U.S.A.

performed in the Out-Patient Department. Not more than two hæmorrhoids should be banded at each session and three weeks at least should elapse between each treatment.

Cryosurgery.—The application of *liquid nitrogen* is currently under active evaluation in some centres. The extreme cold (−196° C) of the application causes coagulation necrosis of the piles which subsequently separate and drop off. The procedure is painless and can be done in the Out-Patient Department. Some encouraging early reports have been issued (Williams).

Operation.—*Indications.*—The following cases are unsuitable for injection or banding treatment. (1) Third-degree hæmorrhoids; (2) failure of conservative treatments of second-degree hæmorrhoids; (3) fibrosed hæmorrhoids; (4) intero-external hæmorrhoids when the external hæmorrhoid is well defined. These are indications for hæmorrhoidectomy.

HÆMORRHOIDECTOMY

Forty-eight hours' pre-operative treatment is necessary. An aperient, a little more than the patient usually takes, or, if none is habitually taken, 2 ml. of cascara evacuant, is given on the evening forty-eight hours prior to the operation. On the following morning a small dose of effervescent saline is given before breakfast. On the evening before the operation a soap-and-water enema is administered, and the anal region is shaved. On the morning of the operation the rectum is washed out with water by means of a funnel and tube attached to a catheter.

Ligation and Excision.—With the patient in the lithotomy position the sphincter is widely stretched, a manœuvre which greatly reduces post-operative pain. The internal hæmorrhoids are then prolapsed by traction on the skin tags, or on the skin of the anal margin. Each hæmorrhoid is dealt with as follows: It is picked up with dissecting forceps and traction is exerted. Traction displays a longitudinal fold (the pedicle) above the hæmorrhoid. Each pedicle is grasped in a fine-pointed hæmostat, as also is each external hæmorrhoid or skin tag connected with each internal hæmorrhoid. These pairs of hæmostats, when held out by the assistants, form a triangle.

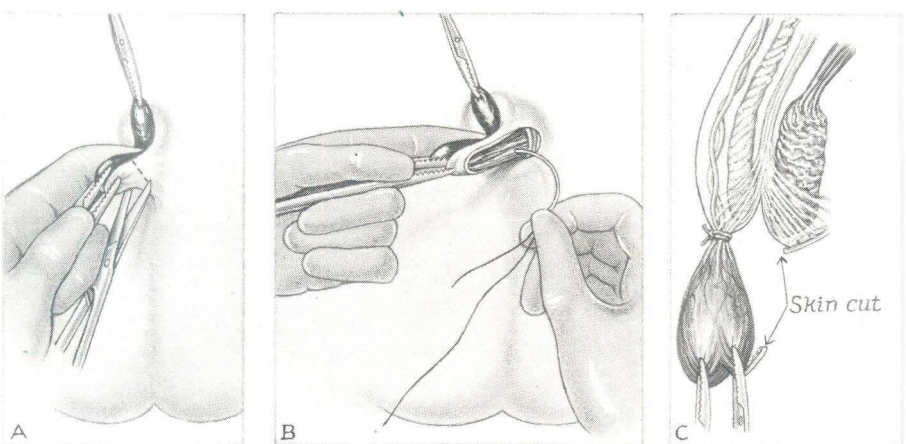


FIG. 1213.—Ligation and excision of hæmorrhoids: (A) The skin cut to the left lateral hæmorrhoid; (B) Transfixion of the pedicle; (C) Ligation.

The operator takes the left lateral pair of hæmostats in the palm of his hand and places the extended forefinger in the anal canal to support the internal hæmorrhoid. In this way traction is applied to the skin of the anal margin. With scissors, a V-shaped cut is made (fig. 1213 (A)), each limb of which is placed on either side of the skin-holding hæmostat. This cut traverses the skin and the corrugator cutis ani. Exerting further traction a little blunt dissection exposes the lower border of the internal sphincter. A transfixion ligature of No. 3 chromic catgut is applied to the

Kenneth Lloyd-Williams, Contemporary. Surgeon, Royal United Hospitals, Bath, England.

pedicle at this level (fig. 1213 (B)). Each hæmorrhoid having been dealt with in this manner (fig. 1213 (C)), they are excised 1·25 cm distal to the ligature, the ends of which are cut about 1 cm from the knot. The stumps of the ligated hæmorrhoids are returned to the rectum by tucking a piece of gauze into the anal canal with closed scissors.

The margins of the skin wounds are trimmed so as not to leave overhanging edges, viz.——————————[1]. Bleeding subcutaneous arteries having been secured, the corners of three pieces of petroleum-jelly gauze are tucked into the anus so as to cover the areas denuded of skin. A pad of gauze and wool, and a firmly applied T-bandage complete the operation.

Post-operative Treatment.—Twice daily baths are started from the first day and liquid paraffin in the evenings. The dressings float off in the bath; if not, they can be removed. An enema is rarely necessary as the bowels usually move on the fourth or fifth day. The baths and dry dressings (a sterile sanitary towel is best for this purpose) held in position by a T-bandage keep the area clean. On the seventh day a finger is passed into the rectum to assess the degree of spasm. If there is risk of stricture formation, a St. Mark's Hospital dilator (fig. 1206) is passed daily. The patient can leave hospital on the tenth to fourteenth day when the risk of secondary hæmorrhage has passed. In the later stages a dry dressing is employed during the day and a zinc and castor oil dressing at night. The paraffin should be stopped after the tenth day and the patient encouraged to pass solid motions in order to dilate the anal canal. The wounds heal in three to six weeks. Modifications of the hæmorrhoidectomy operation have been described by Ferguson and Parks. In these modifications, the anal wound is closed completely or partly to reduce pain and healing time.

Post-operative complications may be early or late.

Early. *Pain* may demand repeated pethidine. Xylocaine jelly introduced through a fine nozzle into the rectum, as necessary, is of considerable value.

Retention of urine is not unusual after hæmorrhoidectomy in male patients, and frequently it is precipitated by the presence of a rectal tube, or pack, or both. Before resorting to catheterisation, the patient should be reassured, given an analgesic, allowed to stand at the side of the bed in privacy or be assisted to a hot bath into which he may be able to void urine.

Reactionary hæmorrhage is much more common than secondary hæmorrhage. The hæmorrhage may be mainly or entirely concealed, but will become evident on examining the rectum.

Treatment.—A suitable dose of morphine is given intravenously. If the bleeding persists, the patient must be taken to the operating theatre and the bleeding-point secured by diathermy or under-running with a ligature on a needle. Should a definite bleeding-point not be found, suspected areas are under-run in this way.

Late.—Secondary hæmorrhage is uncommon; when it occurs, it does so about the seventh or eighth day after operation. It is usually controlled by morphia but if the hæmorrhage is severe, an anæsthetic should be given and a catgut stitch inserted to occlude the bleeding vessel.

Anal stricture.—This must be prevented at all costs. A rectal examination at the tenth day will indicate if stricturing is to be expected. It may then be necessary to give a general anæsthetic and dilate the anus to take four fingers. After that daily use of the dilator should give a satisfactory result.

Anal fissure (p. 1086) and submucous abscess (p. 1106) may also occur.

TREATMENT OF COMPLICATIONS OF HÆMORRHOIDS

In cases of strangulation, thrombosis, and gangrene, it was formerly believed that surgery would cause pylephlebitis. If an adequate antibiotic cover is given from the start, this is not found to be so. Bed rest with frequent hot sitz baths, warm saline compresses with firm pressure usually cause the

[1] If it looks like a clover, the trouble is over,
If it looks like a dahlia, it's surely a failure.

James A. Ferguson, Contemporary. Ferguson Clinic, Grand Rapids, Michigan, U.S.A.
Sir Alan Guyatt Parks, Contemporary. President, R.C.S., England, Surgeon, The London Hospital, London.

pile mass to shrink considerably in three or four days when ligation and excision of the piles can be carried out. Some surgeons consider that the operation at this stage increases the risk of post-operative stenosis and delay surgery for a month or so. They then review the situation and only carry out hæmorrhoidectomy if necessary.

In cases where the patient has been admitted because of severe hæmorrhage the cause usually lies in a bleeding diathesis or the use of anti-coagulants (p. 154). If such are excluded, a local compress containing adrenaline solution, with an injection of morphine and blood transfusion if necessary, will usually control the hæmorrhage. After blood replacement is adequate, ligation and excision of the piles may be required.

EXTERNAL HÆMORRHOIDS

Unlike internal hæmorrhoids, external hæmorrhoids comprise a conglomerate group of distinct clinical entities.

1. A thrombosed external hæmorrhoid is commonly termed a perianal hæmatoma. It is a small clot occurring in the perianal subcutaneous connective tissue, usually superficial to the corrugator cutis ani muscle. The condition is due to back pressure on an anal venule consequent upon straining at stool, coughing, or lifting a heavy weight.

The condition appears suddenly and is very painful, and on examination a tense, tender swelling which resembles a semi-ripe blackcurrant is seen (fig. 1214). The hæmatoma is usually situated in a lateral region of the anal margin. Untreated it may resolve, suppurate, fibrose, and give rise to a cutaneous tag or burst and extrude the clot or continue bleeding.

In the majority of cases resolution or fibrosis occurs. Indeed, this condition has been called ' a five-day, painful, self-curing lesion ' (Milligan).

FIG. 1214.—' Thrombotic ' pile.

Provided it is seen within thirty-six hours of the onset, a perianal hæmatoma is best treated as an emergency. Under local anæsthesia the hæmorrhoid is bisected and the two halves are excised together with 1·25 cm. of adjacent skin. This leaves a pear-shaped wound which is allowed to granulate. The relief of pain is immediate and a permanent cure is certain. On the rare occasions in which a perianal hæmatoma is situated anteriorly or posteriorly, it should be treated conservatively because of the liability of a skin wound in these regions to become an anal fissure.

2. Associated with internal hæmorrhoids = intero-external hæmorrhoids. These have been discussed (p. 1089).

3. Dilatation of the veins of the anal verge becomes evident only if the patient strains, when a bluish cushion-like ring appears. This

Edward Thomas Campbell Milligan, 1886–1972. Surgeon, St. Mark's Hospital, London.

variety of external hæmorrhoid is almost a perquisite of those who lead a sedentary life. The only treatment required is an adjustment in habits of the patient.

4. A 'sentinel' pile is associated with an anal fissure, and has been discussed on p. 1086.

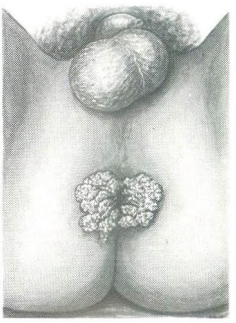


FIG. 1215.—Anal condylomas.

SOME CONDITIONS THAT SIMULATE EXTERNAL HÆMORRHOIDS

Anal warts are usually multiple and present the same characteristics as warts elsewhere. Occasionally they accompany gonococcal proctitis. Simple warts respond well to treatment by precise applications at weekly intervals of 25 per cent podophyllin in liquid paraffin. Large or residual warts require diathermy.

Condylomas (fig. 1215) have a smoother surface and are more pedunculated than the foregoing. Also they are usually moist, with a glairy discharge. Immediate examination of the discharge by 'dark ground' illumination establishes the diagnosis.

Hypertrophic tuberculide of the anus is rare. These are multiple yellowish-brown papillomatous excrescences. The diagnosis is seldom established until microscopic examination has been undertaken.

PRURITUS ANI

This is intractable itching around the anus. Usually the skin is reddened hyperkeratotic and may become cracked and moist. The causes are numerous.

1. *Lack of cleanliness, excessive sweating, and wearing rough or woollen under-clothing* are common causes.

2. *An anal or perianal discharge* which renders the anus moist. The causative lesions include an anal fissure, fistula-in-ano, prolapsed internal or external hæmorrhoids, and excessive ingestion of liquid paraffin. A mucous discharge is an intense pruritic agent.

3. *A vaginal discharge*, especially due to the trichomonas vaginalis.

4. *Parasitic causes.* Threadworms should be excluded, especially in young subjects.[1] Scabies and pediculosis pubis may infest the anal region.

5. *Epidermophytosis* is a common cause especially if the skin between the toes is infected also. Microscopic and cultural examinations are essential. Half-strength Whitfield's ointment quickly gives relief and is the sheet anchor of treatment.

6. *Allergy* is sometimes the cause, in which case there is likely to be a history of other allergic manifestations, such as urticaria, asthma, or hay-fever. Antibiotic therapy may be the precipitating factor.

7. *Skin diseases localised to the perianal skin*—psoriasis, lichen planus and contact dermatitis.

8. *Bacterial infection.*—Intertrigo due to a mixed bacterial infection. Erythrasma due to *Corynebacterium minutissimum* is responsible for some cases and its presence is detected by ultra-violet light which induces a pink fluorescence.

9. *A psychoneurosis.*—It is alleged that in a few instances neurotic individuals become so immersed in their complaint that a pain-pleasure complex develops, the pleasure being the scratching. Possibly this is true, but such a syndrome should not be assumed without firm grounds for coming to this conclusion.

Treatment.—The cause is treated. Other methods include :

Hygienic Measures.—Cotton-wool should be substituted for toilet paper. Soap is avoided, and replaced by a detergent. These measures alone, combined with wearing cotton cellular underwear and applications of calamine lotion, are all that is necessary to cure some cases. If there is much anal hair trapping the moisture and discharge, shaving can be very helpful.

[1] Children suffering from threadworms should wear gloves at night, lest they scratch the perianal region and are reinfested with ova by nail biting—'Parasites lost, Parasites regained'.

Arthur Whitfield, 1868–1947. Professor of Dermatology, King's College Hospital, London.

The following measures are directed mainly to those frequent instances where no obvious reason for the itching can be demonstrated. The best sedative appears to be the alkaloid reserpine, 0·25 mg. daily. This has a tranquillising effect on many anxious patients, but is without direct effect on the pruritus itself.

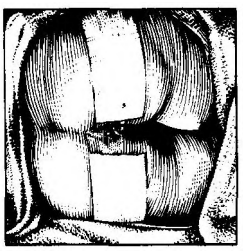


FIG. 1216.—Strapping the buttocks apart.

Hydrocortisone.—In cases with dermatitis, *and only in cases with dermatitis*, prednisolone, applied topically in a cream of 1 per cent. is often beneficial; sometimes after discontinuation of the therapy the pruritus is liable to return, in which event 5 per cent. Xylocaine ointment can be substituted for a time.

Strapping the buttocks apart (fig. 1216) is a most useful procedure, especially when the pruritus is acute, and in chronic cases when the opposing surfaces are moist. The strapping is worn so long as the patient finds it beneficial.

Operative Treatment.—This may be necessary for a concomitant lesion of the ano-rectum which is thought to initiate or contribute to the pruritus. Otherwise, surgery is not indicated and the older operations described for this condition are no longer performed.

PROLAPSE OF THE RECTUM < Partial / Complete

Partial Prolapse.—The mucous membrane and submucosa of the rectum protrude outside the anus for not more than between 1·25 and 3·75 cm. When the prolapsed mucosa is palpated between the finger and thumb, it is evident that it is composed of no more than a double layer of mucous membrane (cf. complete prolapse). The condition occurs most often at the extremes of life—in children between one and three years of age, and in elderly people.

In Infants.—The direct downward course of the rectum, due to the as yet undeveloped sacral curve (fig. 1217) predisposes to this condition, as does the reduced resting anal tone which offers diminished support to the mucosal lining of the anal canal (Mann).

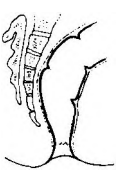


FIG. 1217.—The absence of the normal sacral curve predisposes to rectal prolapse in an infant (cf. fig. 1204).

In children, partial prolapse often commences after an attack of diarrhœa, as a result of severe whooping cough, or from loss of weight and consequent diminution in the amount of fat in the ischio-rectal fossæ.

In adults the condition is usually associated with third-degree hæmorrhoids or a complete prolapse. In the female a torn perineum predisposes to prolapse, and in the male straining from urethral obstruction. In old age, both partial and complete prolapse are due to atony of the sphincter mechanism.

Partial prolapse may follow an operation for fistula-in-ano where a large portion of muscle has been divided. Here the prolapse is usually localised to the damaged quadrant and is seldom progressive.

Charles Victor Mann, Contemporary. Surgeon, St. Mark's Hospital for Diseases of the Rectum and Colon, London.

Prolapsed mucous membrane is pink (fig. 1219); prolapsed internal hæmorrhoids are plum coloured, and more pedunculated.

Treatment :

In Infants and Young Children : (1) *Digital Reposition.*—The mother must be taught to replace the protrusion. The distal two-thirds of the index finger is wrapped in Kleenex tissue. The finger is inserted into the protrusion, and the mass is eased into place. Gently the finger is withdrawn, leaving the Kleenex tissue to disintegrate. In cases of malnutrition, dietetic adjustments are necessary.

(2) *Submucous Injections.*—If digital reposition fails after six weeks' trial, injections of 5 per cent. phenol in almond oil are carried out under general anæsthesia. As a result of the aseptic inflammation following these injections, the mucous membrane becomes tethered to the muscle coat.

Technique.—The submucosa at the apex of the prolapse is injected circularly, so as to form a raised ring, up to 10 ml. of the solution being injected. A similar injection is made at the base of the prolapse. Alternatively, if the prolapse cannot be brought down, the injections are given through a proctoscope.

(3) *Thiersch's Operation.*—When the prolapse persists in spite of these measures, Thiersch's operation (p. 1100) is almost certain to succeed. In infants, insertion of the little finger into the anus before the stitch is tied is recommended. In infants and young children, strong chromic catgut should be used for the stitch instead of silver wire: if wire were employed (or any other retained unabsorbable material) as growth proceeded the stitch would have to be removed or anal stenosis would result. Since the procedure is designed only as a *temporary* measure in the young, chromic catgut is adequate for the purpose.

In Adults : (1) *Submucous injections* of phenol in almond oil occasionally are successful in cases of early partial prolapse.

(2) *Excision of the Prolapsed Mucosa.*—When the prolapse is unilateral the redundant mucosa can be excised after inserting and tying Goodsall's ligature (fig. 1218) which, after the needles have been cut off, permits the base of the prolapsed mucous membrane to be ligated in three portions lying in juxtaposition. When necessary, the operation is combined with hæmorrhoidectomy, and if the pedicle of one or more of the hæmorrhoids is broad, Goodsall's ligature is applied.

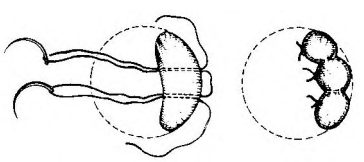


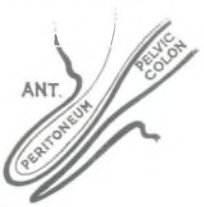
FIG. 1218.—Goodsall's ligature.

Complete prolapse (*syn.* procidentia) is less common than the partial variety. The protrusion consists of all layers of the rectal wall and is a descending hernia-en-glissade of the rectum downward through the levator ani. As the rectum descends it intussuscepts upon itself. The process starts with the anterior wall of the rectum where the supporting tissues are weakest, especially in women. It is more than 3·75 cm. and commonly as much as

Karl Thiersch, 1822–1895. Professor of Surgery, Leipzig, Germany.
David Henry Goodsall, 1843–1906. Surgeon, St. Mark's Hospital for Diseases of the Rectum and Colon, London.

10 to 15 cm. in length. On palpation between the finger and the thumb the prolapse feels much thicker than a partial prolapse, and obviously consists of a double thickness of the entire wall of the rectum. Any prolapse over 5 cm in length contains anteriorly between its layers a pouch of peritoneum, viz. ——→ When large, the peritoneal pouch contains small intestine which returns to the general peritoneal cavity with a characteristic gurgle when the prolapse is reduced. The prolapsed mucous membrane (fig. 1219) is often arranged in a series of circular folds. The anal sphincter is characteristically patulous and gapes widely on straining to allow the rectum to prolapse. Complete prolapse is uncommon in children. In adults it can occur at any age, but is more common in the elderly. Women are six times more often affected than men. In women, prolapse of the rectum is commonly associated with prolapse of the uterus, or a past history of a gynæcological operation, e.g. hysterectomy.

FIG. 1219.—Complete rectal prolapse. (*G. D. Adhia, F.R.C.S., Bombay.*)



Differential Diagnosis.—In the case of a child with abdominal pain, prolapse of the rectum must be distinguished from **ileo-cæcal intussusception** protruding from the anus. Figs. 1220 and 1221 make the differential diagnosis clear. In **recto-sigmoid intussusception** in the adult there is a deep groove (5 cm or more) between the emerging protruding mass and the margin of the anus.

FIG. 1220.—Partial prolapse of the rectum.

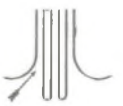


FIG. 1221.—Intussusception protruding from the anus.

Treatment.—The Thiersch operation can be recommended in elderly patients, in those suffering from injury or disease of the spinal cord, and in the feeble-minded, in whom the condition is relatively common, as well as in very early life (p. 1099).

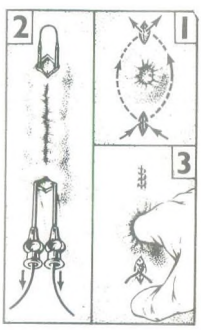


FIG. 1222.—The Thiersch operation (Dodd's modification).

The Thiersch Operation.—A short incision is made in the midline anteriorly and posteriorly about 1·25 cm. from the anal verge. Large-bore hollow needles are inserted one at a time through the posterior wound, in such a way as to encircle the anus 1 cm. from the orifice (fig. 1222 (1)), until their points emerge from the anterior wound (fig. 1222 (2)). A malleable silver wire, gauge 19 or 20, is introduced through the points of the needles. The needles are withdrawn. The assistant introduces his index finger into the anal canal (fig. 1222 (3)), and the surgeon tightens the wire around the finger by twisting the ends of the wire. The finger is withdrawn and the ends of the wire are clipped short, and bent back. Monofilament O nylon can be used instead of wire, and passed round the rectum two or three times to produce a 'watch-spring' effect when the stitch is tightened. The wounds are then closed.

Harold Dodd, Contemporary. (*See footnote p. 184.*)

A glycerol suppository or a low-pressure enema is given as required post-operatively. Confinement to bed is unnecessary.

By and large, the results of Thiersch's operation are remarkably successful, but in about 50 per cent of cases the wire breaks; it is, however, easily removed and the operation can be carried out several times, at intervals, on the same patient. Its main disadvantage is that spurious incontinence of fæces caused by fæcal impaction (p. 1054) is wont to occur in all age groups. Constipation must, therefore, be guarded against vigilantly.

Operative Suspension of the Prolapsed Bowel.—Of many operations designed to cure complete prolapse of the rectum, the following procedures are relatively simple. They are recommended in patients with complete prolapse who are otherwise in good health. All are abdominal procedures designed to replace and hold the rectum in its proper position. Many other procedures have been described incorporating the same principles illustrated by these operations, some of which are carried out through a perineal approach.

Wells' Operation.—In this operation the rectum is fixed firmly to the sacrum by inserting a sheet of polyvinyl alcohol sponge between them. The rectum is separated from the sacrum in the usual way. The sponge is fixed by a series of sutures to the periosteum over the midline of the sacrum and is then wrapped loosely about the rectum covering all except the anterior wall. The free margins of the polyvinyl sponge are sutured to the lateral margins of the anterior wall of the rectum. The peritoneal floor is resutured so that the sponge is excluded from the peritoneal cavity. Polyvinyl sponge does not give rise to a foreign body reaction but it does produce very marked fibrous tissue formation. Many proctologists regard this as the method of choice. The results are excellent.

Ripstein's Operation.—In this operation, the recto-sigmoid junction is hitched up by a Teflon sling to the front of the sacrum just below the sacral promontory. The operation is very safe and simple and the results are good.

Lahaut's Operation.—This operation depends entirely upon mobilising the rectum and lower sigmoid colon and holding it up by extraperitonealising it through the rectus sheath. The results are moderate.

ANAL INCONTINENCE

Congenital.—(*a*) In cases of imperforate anus, partial or complete lack of the sphincter mechanism is the exception rather than the rule; (*b*) patulous anus associated with mental deficiency.

Traumatic.—By far the most frequent cause is a complication of a complete perineal tear during parturition. Other injuries resulting in a torn anal sphincter are extremely rare.

Post-operative.—(*a*) After the ano-rectal ring has been severed during an operation for a high fistula-in-ano; (*b*) following conservative perineal resection of the rectum for carcinoma, an operation that cannot be recommended.

Associated with advanced complete prolapse due to atony of the sphincter.

Interruption of the reflex arc responsible for sphincter control in diseases of the nervous system, notably neuro-syphilis (e.g. tabes dorsalis).

Carcinoma of the Anus involving the Sphincter (p. 1123).

Treatment of Anal Incontinence:

Partial incontinence often can be remedied by the Thiersch operation (p. 1100). This also is the operation of choice in cases of complete incontinence associated with mental deficiency.

Partial and often complete incontinence due to parturition is particularly amenable to operative treatment. Posterior perineorrhaphy with repair of the torn edges of the sphincter often cures the condition.

Charles Alexander Wells, Contemporary. Professor Emeritus of Surgery, Liverpool, England.
Charles Benjamin Ripstein, Contemporary. Surgeon, Brookdale Hospital Center, New York, U.S.A.
Jules Lahaut, Contemporary. Surgeon-in-Chief, Hôpital du Pont Canal, Jemappes (Hainaut), Belgium.

PROCTITIS

Inflammation is sometimes limited to the rectal mucosa; in others it is associated with a similar condition in the colon (procto-colitis). The inflammation can be acute or chronic. The symptoms are tenesmus, the passage of blood and mucus and, in severe cases, of pus also. Although the patient has a frequent intense desire to defæcate, the amount of fæces passed at a time is small. Acute proctitis is usually accompanied by malaise and pyrexia. On rectal examination the mucosa feels swollen and is often exceedingly tender. Proctoscopy is seldom sufficient and sigmoidoscopy is the more valuable method of examination. Skilled pathological assistance is required to establish or exclude the diagnosis of specific infection by bacteriological examination and culture of the stools, examination of scrapings or swabs from ulcers, and serological tests. When early carcinoma cannot be excluded, biopsy is necessary.

Non-specific proctitis is an inflammatory condition affecting the mucosa, and to a lesser extent the submucosa, confined to the terminal rectum and anal canal. It is the most common variety.

Ætiology is unknown. The concept that the condition is a mild and limited form of ulcerative colitis (although actual ulceration is not present) is the most acceptable hypothesis. Approximately 10% of cases of limited procto-sigmoiditis eventually proceed to total ulcerative colitis.

Clinical Features.—The patient is usually middle-aged, and complains of slight loss of blood in the motions. Often the complaint is one of diarrhœa, but on closer questioning it transpires that usually one relatively normal action of the bowels occurs each day, although it is accompanied by some blood. During the day the patient attempts to defæcate, with the passage of flatus and a little blood-stained fæcal matter; which is mistakenly interpreted as diarrhœa. On rectal examination the mucosa feels warm and smooth. Often there is some blood on the examining finger. Proctoscopic and sigmoidoscopic examination shows inflamed mucous membrane of the rectum, but no ulceration. The inflammation extends for only 12·5 or 15 cm from the anus, the mucosa above this level being quite normal.

Treatment.—Although, fortunately, the condition is self-limiting, much relief may be obtained from the use of Salazopyrin, Acetarsol suppositories or Prednisolone retention enemas. Milk should be rigidly excluded from the diet and the motions kept soft with Isogel or Celevac.

Ulcerative Procto-colitis.—Proctitis is present in a high percentage of cases of ulcerative colitis, and the degree of severity of the rectal involvement may influence the type of operative procedure (p. 1007).

Proctitis Due to Specific Infections:

Bacillary Dysentery.—The appearance is that of an acute purulent proctitis with multiple small shallow ulcers. The examination of a swab taken from the ulcerated mucous membrane is more certainly diagnostic than is a microscopical examination of the stools. Proctological examination is painful; agglutination tests may render it unnecessary.

Amœbic Dysentery.—The infection is more liable to be chronic, and exacerbations after a long period of freedom from symptoms often occur. Proctoscopy and sigmoidoscopy are not painful. The appearance of an amœbic ulcer is described on p. 1009. Scrapings from the ulcer should be immersed in warm normal saline solution and sent to the laboratory for immediate microscopical examination.

Amœbic granuloma presents as a soft mass, usually in the recto-sigmoid region. This lesion is frequently mistaken for a carcinoma. Sigmoidoscopy shows an ulcerated surface, but the mass is less friable than a carcinoma. A scraping should be taken, preferably with a small sharp spoon on a long handle, and the material sent for

immediate microscopical examination, as detailed above. If doubt exists, a provocative dose of emetine may cause cysts of the amœbæ to appear in the stools. A biopsy is also required. Treatment is as described on p. 1010.

Amœbic granuloma of the rectum is from time to time encountered in a patient who has never visited a country in which the disease in endemic. Persons living in old people's institutions are liable to harbour this deceptive lesion.

Tuberculous proctitis is nearly always associated with active pulmonary tuberculosis, and is often complicated by a tuberculous fistula-in-ano or tuberculous ulceration of the anus. Submucous rectal abscesses burst and leave ulcers with an undermined edge. A hypertrophic type of tuberculous proctitis occurs in association with tuberculous peritonitis or tuberculous salpingitis. This type of tuberculous proctitis requires biopsy for confirmation of the diagnosis.

Gonococcal proctitis occurs in both sexes as the result of rectal coitus, and in the female from direct spread from the vulva. In the acute stage the mucous membrane is hyperæmic and thick pus can be expressed as the proctoscope is withdrawn. In the early stages the diagnosis can be readily established by bacteriological examination, but later, when the infection is mixed, it is more difficult to recognise. Specific treatment is so effective that local treatment is unnecessary.

Lymphogranuloma Inguinale.—The modes of infection are similar to those of gonococcal proctitis, but in the female infection spreading from the cervix uteri via lymphatics to the pararectal lymph nodes is common. The proctological findings are similar to those of gonococcal proctitis. The diagnosis of lymphogranuloma inguinale should be strongly suspected when the inguinal lymph nodes are greatly enlarged, although the enlargement may be subsiding by the time proctitis commences. Frei's intradermal test is positive in 95 per cent. of cases, and the complement-fixation test is even more accurate (p. 46).

Primary Syphilis.—A primary chancre may occur inside the anus (p. 1087).

'Strawberry' lesion of the recto-sigmoid is due to an infection by *Spirochæta vincenti* and *Bacillus fusiformis*. The leading symptom is diarrhœa, often scantily blood-stained. Occasionally the diagnosis can be made by the demonstration of the specific organisms in the stools. More often sigmoidoscopy is required. The characteristic lesion is thickened, somewhat raised mucosa with superficial ulceration in the region of the recto-sigmoid. The inflamed mucous membrane oozes blood at numerous pin-points, giving the appearance of an over-ripe strawberry. A swab should be taken from the lesion and examined for Vincent's and fusiform organisms. Swabs from the gums and the throat are also advisable.

Treatment.—Acetarsol suppositories together with vitamin C is almost specific.

Rectal bilharziasis is caused by the *Schistosoma mansoni*, which is endemic in many tropical and subtropical countries, and particularly in the delta of the Nile.

Stage 1.—A cutaneous lesion develops at the site of entrance of the cercariæ.[1]

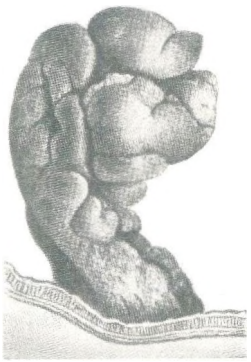


FIG. 1223.—Bilharzial papilloma. (*The late Dr. H. P. Keatinge, Cairo.*)

Stage 2 is characterised by pyrexia, urticaria, and a high cosinophilia.

Both these stages are frequently overlooked.

Stage 3 is due to deposition of the ova in the rectum (much more rarely in the bladder) and is manifested by bilharzial dysentery. On examination in the later stages papillomas are frequently present. The papillomas, which are sessile or pedunculated (fig. 1223), contain the ova of the trematode, the life-cycle of which resembles that of *Schistosoma hæmatobium* (fig. 1224).

Untreated, the rectum becomes festooned, and prolapse of the diseased mucous membrane is usual. Multiple fistulæ-in-ano are prone to develop.

General Treatment of Bilharziasis Mansoni.—(*a*) *Compounds*

FIG. 1224.—Ovum of *Schistosoma hæmatobium* (terminal spike[2]). (*Dr. M. Kinaw, Cairo.*)

[1] Cercariæ = a parasite of freshwater snails.
[2] The ovum of *S. mansoni* has a lateral spike.

Wilhelm Siegmund Frei, 1885–1943. Professor of Dermatology, Berlin, who later settled in New York, U.S.A.
Jean Hyacinthe Vincent, 1862–1950. (*See footnote p. 560.*)
Theodor Maximilian Bilharz, 1825–1862. Professor of Zoology, Cairo.
Sir Patrick Manson, 1844–1922. (*See footnote p. 873.*)

not containing antimony include niridazole (Ambilhar) in cases of infestation with *S. hæmatobium* or *S. mansoni* (**not** *S. japonicum* or in those with heart, mental or liver disease). Dose 25 mg/kg body wt. daily in two divided doses for 5–7 days. Hycanthone, lucanthone, oxamniquine are other compounds with weight related single doses given by deep i.m. injection, and all with toxic side effects. Metriphonate is an effective organophosphorus compound, also to be handled with great care.

(*b*) *Compounds containing antimony,* either as the salts, *tartar emetic* (antimony potassium tartrate) and the sodium salt given i.v., or antimony lithium thiomalate, sodium antimonylgluconate, stibogluconate and stibocaptate (Astiban) may still be required.

Local Treatment.—When the papillomas persist in spite of general treatment, they must be treated in the same manner as other papillomas (p. 1113).

Proctitis due to herbal enemas is a well-known clinical entity to those practising in tropical Africa. Following an enema consisting of a concoction of ginger, pepper, and tree-bark administered by a witch doctor, a most virulent proctitis sets in. Pelvic peritonitis frequently supervenes. Not infrequently a complete gelatinous cast of the mucous membrane of the rectum is extruded. Very large doses of morphine, together with streptomycin, often prevent a fatal issue if commenced early (Bowesman). Temporary colostomy is often advisable.

Treatment of severe proctitis necessitates confinement to bed. It will be appreciated that in most instances specific treatment can be given once the cause has been elucidated. The stools should be kept soft with Isogel. Local instillations of 150 ml. of olive oil are soothing. Suppositories of succinyl-sulphathiazole are often beneficial. The specific treatment for the dysenteries, tuberculosis, gonorrhœa, lymphogranuloma inguinale, and syphilis are described in the appropriate sections of this book.

SOLITARY ULCER

This is a rare condition that takes the form of an ulcer on the anterior wall of the rectum. It is thought to be traumatic in origin, either by sexual malpractises from below, or by intussusception of the anterior rectal wall into the anal sphincter from above. It is a benign condition that is clinically very easily confused with a carcinoma until repeated biopsies are performed. The clinico-pathological features have been well documented by Morson. The condition, although benign, is difficult to cure. The symptoms of bleeding and discharge can sometimes be ameliorated by topical hydrocortisone applications or by diathermy destruction of the ulcer (which usually returns). Some cases respond to perineal operations designed to prevent the downward intussusception, e.g. post-anal repair.

ANO-RECTAL ABSCESSES

In 60 per cent. of cases the pus from the abscess yields a pure culture of *Esch. coli*; in 23 per cent. a pure culture of *Staphylococcus aureus* is obtained. In diminishing frequency, pure cultures of *Bacteroides*, a streptococcus, or *B. proteus* are found. In many cases the infection is mixed. In a high percentage of cases—some estimate it as high as 90 per cent.—the abscess commences as an infection of an anal gland (figs. 1225 and 1226). Other causes are penetration of the rectal wall, e.g. by a fish bone, a blood-borne infection, or an extension of a cutaneous boil.

A large percentage of ano-rectal abscesses coincide with a fistula-in-ano. For this reason, ano-rectal abscess becomes a highly important subject. Moreover, as antibiotics cannot reach the contents of an abscess in adequate concentration, no reliance can be placed on antibiotic therapy alone.

Charles Bowesman, Contemporary. Formerly Surgical Specialist, Colonial Medical Service, Kumasi, Ghana.
Basil Clifford Morson, Contemporary. Pathologist, St. Mark's Hospital, London.

Differential Diagnosis.—The only conditions with which an ano-rectal abscess is likely to be confused are an abscess connected with a pilonidal sinus, Bartholin's gland, or Cowper's gland (p. 1284).

Classification of Ano-rectal Abscesses.—A clear understanding of suppuration in this area is dependent on a concise knowledge of the anatomy (figs. 1225 and 1226). There are four main varieties — perianal, ischio-rectal, submucous, and pelvi-rectal.

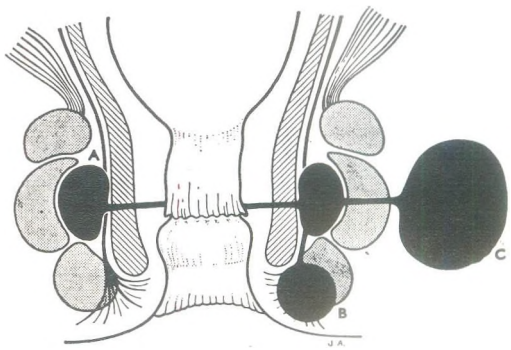


FIG. 1225.—To illustrate the spread of infection from the primary anal gland abscess (A) to the perianal region (B) and the ischio-rectal fossa (C). (*After Sir Alan Parks, F.R.C.S., London.*)

1. **Perianal** (60 per cent.).—This usually occurs as the result of suppuration in an anal gland, which spreads superficially to lie in the region of the subcutaneous portion of the external sphincter (fig. 1226A). It may also occur as a result of a thrombosed external pile. If the hæmatoma is not evacuated, it may become infected and a perianal abscess results. This is the most common abscess of the region. Persons of all ages are affected, and the condition is not uncommon, even in infancy and childhood. The constitutional symptoms and the pain are less pronounced than in the ischio-rectal abscess because the pus can expand the walls of this part of the intermuscular space comparatively easily. Early diagnosis is made by inspecting the anal margin when an acutely tender rounded cystic lump about the size of a cherry is seen and felt at the anal verge below the dentate line (fig. 1227).

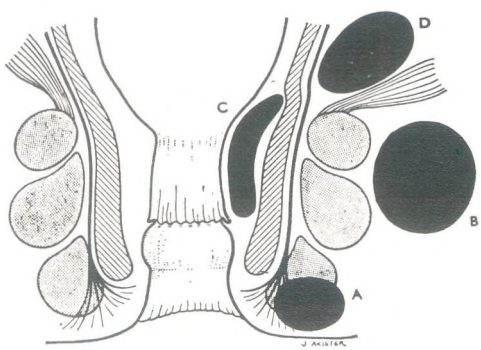


FIG. 1226.—The four types of ano-rectal abscess —(A) Perianal. (B) Ischio-rectal. (C) Submucous. (D) Pelvi-rectal. (*After W. F. W. Southwood, F.R.C.S., Bath.*)

FIG. 1227. — Perianal abscess. (*W. B. Gabriel, F.R.C.S., London.*)

Treatment.—No time should be lost in evacuating the pus.

Operation.—Thorough drainage is achieved by making a cruciate incision over the abscess and excising the skin edges—this completely removes the 'roof' of the abscess. Healing commonly occurs within a few days.

Caspar Bartholin, 1655–1738. Professor of Medicine, Anatomy and Physics, Copenhagen.

2. **Ischio-rectal abscess** (30 per cent.).—Commonly, this is due to an extension laterally through the external sphincter of a low intermuscular anal abscess (fig. 1226B). Rarely, the infection is either lymphatic or blood-borne.

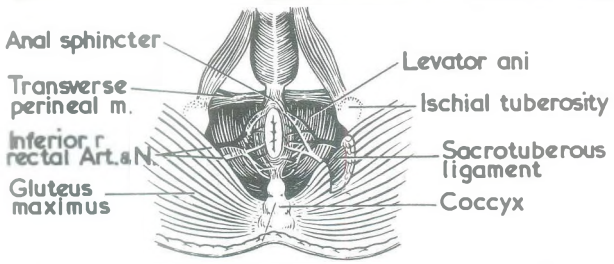


FIG. 1228.—The ischio-rectal fossa. (*After V. C. Shah and Y. V. Shah.*)

The fat, which fills the ischio-rectal fossa (fig. 1228), is particularly vulnerable because it is poorly vascularised; consequently it is not long before the whole space becomes involved. The ischio-rectal fossa communicates with that of the opposite side via the post-sphincteric space, and if an ischio-rectal abscess is not evacuated early, involvement of the contralateral fossa is not uncommon.

An ischio-rectal abscess gives rise to a tender, brawny induration palpable on the corresponding side of the anal canal and the floor of the fossa. Constitutional symptoms are severe, the temperature often rising to 38·9° C. Men are affected more often than women.

Treatment.—Operation should be undertaken early—as soon as it is certain that an abscess is present in this area—remembering that antibiotic therapy often masks the general signs.

Operation.—*Stage* 1.—A +-shaped incision (fig. 1229 inset) is made into the abscess. An adequate portion of skin that includes most, if not all, of the roof of the abscess is excised (fig. 1229). The cavity is explored and, if septa exist, they should be broken down gently with a finger and the necrotic tissue lining the walls of the abscess is removed by the finger wrapped in gauze. Nothing further is done at this stage.

Stage 2.—As soon as the acute infection has subsided, the wound should be re-examined, preferably under general anæsthesia. A careful search is made for a fistulous opening communicating with the anal canal. If such is found, the treatment should be as for fistula (p. 1110). If no fistula is found, the cavity should be lightly packed with gauze wrung out in any weak antiseptic favoured by the operator. A T-bandage is applied. When the cavity has become covered with granulation tissue skin grafting may help to expedite final epithelialisation.

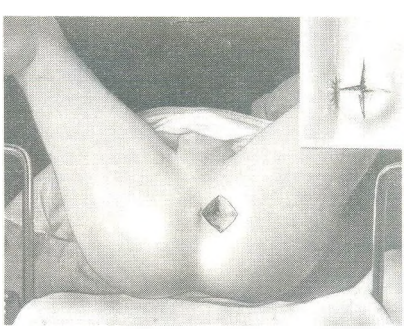


FIG. 1229.—Excision of the skin forming the floor of the ischio-rectal fossa is an essential step in the efficient treatment of an ischio-rectal abscess.

3. **Submucous abscess** (5 per cent.) occurs above the dentate line (fig. 1226C). When it occurs after the injection of hæmorrhoids it always resolves. Otherwise, it can be opened with sinus forceps when adequately displayed by a proctoscope.

4. **Pelvi-rectal abscess** is situated between the upper surface of the

levator ani and the pelvic peritoneum. It is nothing more or less than a pelvic abscess and as such is usually secondary to appendicitis, salpingitis, diverticulitis, or parametritis. An important point to remember is that rarely it may be due to over-enthusiastic attempts to drain an ischio-rectal abscess or to display the site of a fistula resulting therefrom, when a probe is pushed through the levator ani from below (p. 1111). Crohn's disease is an important cause of pelvic disease that can present as peri-anal sepsis (cf. fistula-in-ano).

5. **Fissure Abscess.**—This is the name given to a subcutaneous abscess lying in immediate association with an anal fissure (p. 1086). Drainage is achieved at the same time as the fissure is treated by sphincterotomy.

FISTULA-IN-ANO

A fistula-in-ano is a track, lined by granulation tissue which opens deeply in the anal canal or rectum and superficially on the skin around the anus. It usually results from an ano-rectal abscess which burst spontaneously or was opened inadequately (fig. 1225). The fistula continues to discharge and, because of constant reinfection from the anal canal or rectum, seldom, if ever, closes permanently without surgical aid. An ano-rectal abscess may produce a sinus the orifice of which has the appearance of a fistula, but it does not communicate with the anal canal or the rectum. By definition this is *not* a fistula, but a sinus.

TYPES OF ANAL FISTULÆ

These are divided into two groups, according to whether their internal opening is below or above the ano-rectal ring.

Low Level.—These open into the anal canal below the ano-rectal ring.

High Level.—These open into the anal canal at or above the ano-rectal ring.

As an alternative to the common anatomical classification illustrated in fig. 1230, Parks has produced another based on the origin of the fistulæ from an abscess in an anal gland situated on the plane between the internal and external sphincters (the ' anal intersphincteric plane ') fig. 1231.

The importance of deciding whether a fistulæ is a low- or a high-level type

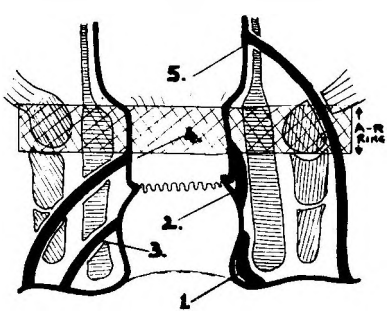


FIG. 1230.—Types of anal fistulæ (standard classification). 1. Subcutaneous, 2. submucous, 3. low anal, 4. high anal, 5. pelvi-rectal.

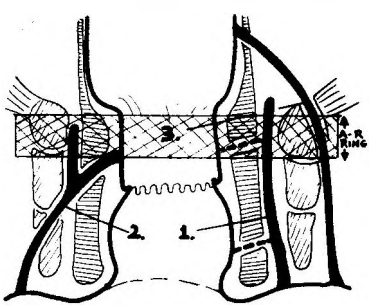


FIG. 1231.—Types of Anal Fistulæ. 1. Intersphincteric, 2. transphincteric (which may be *high* or *low*), 3. supra-levator. *(After A. G. Parks.)*

Sir Alan Guyatt Parks, Contemporary. Surgeon, The London Hospital, London.
For treating successfully Louis XIV's fistula-in-ano, Charles Félix, barber-surgeon to the Court, received a gift of a farm, 300,000 livres, and a title. 300,000 livres today would be worth about £25,000.

is that a low-level fistula can be laid open without fear of permanent incontinence (from damage to the ano-rectal bundle), while a high-level fistula can be treated only by 'staged' operations, often with the use of a protective colostomy to prevent septic complications and to shorten healing time between the stages.

In probing a high fistulous track, great care must be taken not to create an internal opening into the rectum where none existed previously. Such a disaster could convert a relatively straightforward 'intersphincteric' track into a high 'pelvi-rectal' fistula that might prove very difficult to cure.

By the standard classification, a *high* fistula refers to both a high anal (No. 4, fig. 1230) and a pelvi-rectal fistula (No. 5, fig. 1230). By the Parks classification, both a high transphincteric or a supralevator fistula would qualify as high, with the intersphincteric falling into either category depending on whether an internal opening was present at all, and at what level it entered the anal canal (see No. 1, fig. 1231).

Low-level Fistulæ

Clinical Features.—Commonly, the principal symptom is a persistent sero-purulent discharge that irritates the skin in the neighbourhood and causes discomfort. Often the history dates back for years. So long as the opening is large enough for the pus to escape, pain is not a symptom, but if the orifice is occluded pain increases until the discharge recurs. Frequently there is a solitary external opening, usually situated within 3·75 cm. of the anus, presenting as a small elevation with granulation tissue pouting from the mouth of the opening. Sometimes superficial healing occurs, pus accumulates and an abscess reforms and discharges through the same opening, or a new opening. Thus there may be two or more external openings, usually grouped together on the right or left of the middle line, but occasionally, when both ischio-rectal fossæ are involved, an opening is seen on each side, in which case there is often intercommunication between them (fig. 1232). As a rule there is much induration of the skin and subcutaneous tissues around the fistula.

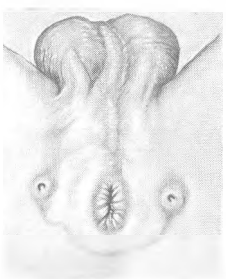


FIG. 1232.—Horseshoe fistula-in-ano. Both ischio-rectal fossæ involved. Usually there is only one internal orifice.

Goodsall's Rule.—Fistulæ with an external opening in relation to the anterior half of the anus tend to be of the direct type (fig. 1233). Those with an external opening or openings in relation to the posterior half of the anus, which are much more common, usually have curving tracks, and may be of the horseshoe-variety.

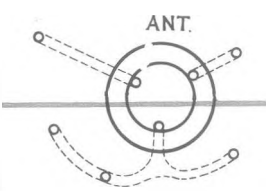


FIG. 1233.—Illustrating Goodsall's rule.

Digital Examination.—Not infrequently an internal opening can be felt as a nodule on the wall of the anal canal. Irrespective of the number of external openings, there is almost invariably but one internal opening.

David Henry Goodsall, 1843–1906. (*See footnote p. 1099.*)

Proctoscopy sometimes will reveal an internal opening of the fistula. A hypertrophied papilla is suggestive that the internal orifice lies within the crypt related to the papilla (fig. 1235).

Probing.—In the past it was the universal practice to probe a fistula in the ward or the out-patient department. Such manœuvres accomplish nothing, are painful, and are liable to reawaken dormant infection. Furthermore, if probing is performed without the utmost gentleness, or if the patient, experiencing pain, makes a sudden jerk, a false passage may result which complicates the condition still further. Probing should be postponed until the patient is under an anæsthetic in the operating theatre.

The injection of lipiodol, or other opaque medium, along the sinus, prior to radiography, has little to recommend it. The radiographs thus obtained are seldom illuminating, and the procedure is likely to cause a recrudescence of inflammation.

Radiography of the thorax should be undertaken and the possibility of pulmonary tuberculosis considered, despite the fact that today it will be found in only a very small proportion of patients with fistula-in-ano.

Special Clinical Types of Fistulæ-in-ano :

1. **Fistula connected with an Anal Fissure.**—Unlike the usual fistula-in-ano, pain (due to the fissure) is a leading symptom. The fistula is very near the anal orifice, usually posterior, and the external opening is often hidden by the sentinel pile.

2. **Fistula with an internal opening above the ano-rectal ring** is due, almost invariably, to penetration by a foreign body or simple intraluminar incision of a high intermuscular abscess erroneously thought to be submucous.

3. **Granulomatous infections.**—If induration around a fistula is lacking, if the opening is ragged and flush with the surface, if the surrounding skin is discoloured and the discharge is watery, it strongly suggests that the fistula is due to a tuberculous infection. In more than 30% of patients suffering from pulmonary tuberculosis, virulent tubercle bacilli are present in the rectum. About 2–3% of fistulæ-in-ano are tuberculous, but in sanatoria and settlements for tuberculous patients the incidence is higher. Histo-pathological examination supplies the only criterion of importance as to whether the tissue removed is tuberculous. The fistula will usually respond to anti-tuberculous drugs alone (p. 37). The differential diagnosis is from Crohn's disease. In Crohn's disease the rectal mucosa is often involved.

4. **Fistulæ with many external openings** may arise from tuberculous proctitis, ulcerative proctocolitis, Crohn's disease of colon or ileum, bilharziasis, and lymphogranuloma inguinale with a fibrous rectal stricture. Colloid carcinoma may complicate fistulæ-in-ano. Crohn's disease is the most frequent cause seen in this country from this group.

5. **Colloid Carcinoma arising within Perianal Fistulæ.**—Colloid carcinoma of the rectum is notoriously liable to be complicated by perianal fistulæ. In some instances the fistulous condition, with its discharge of colloid material, overshadows the primary carcinoma, and not a few unfortunate patients have had their condition diagnosed for a time as an inflammatory fistula-in-ano. If a primary tumour is present in the

FIG. 1234.—Perianal fistulæ due to colloid carcinoma. No primary in the rectum. *(C. E. Dukes, F.R.C.S., London, and C. Galvin, F.R.C.S.)*

Burrill Bernard Crohn, Contemporary. Gastro-enterologist, Mount Sinai Hospital, New York, U.S.A.

rectum, usually it can be detected and its nature established by biopsy. Dukes has established conclusively that colloid carcinomatous fistulæ can develop without a primary neoplasm in the rectum (fig. 1234). He regards such cases as examples of colloid carcinoma developing in a reduplicated portion of the intestinal tract.

Hidradenitis Suppurativa.—This is a chronic infection of the apocrine glands around the anal margin giving rise to numerous sinuses The Mons Pubis and groin also can be affected. After excision of the area, granulation and healing is accelerated by using Silastic foam dressings (L. Hughes).

Treatment.—That the fistulous track must be laid open from its termination to its source was a rule promulgated by John of Arderne more than 500 years ago. Today, pre-operative treatment by cleansing enemas is a necessity.

The operation can best be described in stages :

Step 1.—When the patient has been anæsthetised, he is placed in the lithotomy position or in the prone jack-knife position, according to the preference of the operator. Using bidigital palpation under anæsthesia, it is often possible to obtain more information concerning a fistula than can be learned from probing; it is surprisingly easy to insert a probe through the wall of the track. Unfortunately, many inexperienced operators find it more reassuring to create a false passage than to risk criticism for not being able to demonstrate the internal opening. Careful bidigital palpation of the peri-anal tissue will often reveal a cord-like induration, representing the track, which will lead the intra-anal finger towards the proximal opening. Rather than insert a probe through the distal orifice at this stage, it is better to endeavour to find the internal opening via a proctoscope. If the internal opening still cannot be seen, the insertion of a probe retrogradely into an anal crypt, especially one with a nearby hypertrophied papilla, often reveals the internal portion of the track (fig. 1235). The injection of methylene blue or other dye into the external mouth of the fistula to establish the site of the internal opening is occasionally necessary, but is not recommended as a routine.

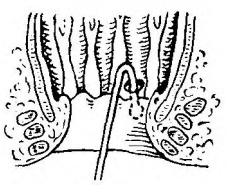


FIG. 1235.—Retrograde probing of an anal crypt sometimes reveals the internal orifice of the fistula.

Step 2.—A probe-pointed director (fig. 1236) is inserted into the distal orifice, and it is advanced delicately until it reaches a point where it does not pass readily. The track is opened along the director, and bleeding is controlled.

FIG. 1236.—A director with a probe-pointed malleable extremity is a useful instrument.

Step 3.—If it is not at once evident in which direction the track passes, granulations are wiped away with gauze (it is seldom necessary to use a curette). Often this will leave a granulation-filled spot at one site only. Gentle probing at this spot frequently will give the clue to the continuation of the fistula. The director is reinserted, and again followed with the knife for a short distance. This procedure is repeated until the entire track, and any side channels, are laid open. As far as possible, all muscle is divided at right angles to its fibres. In the rare event of the track passing above the ano-rectal ring, cutting should cease at the level of the dentate line, and from thenceforth the operation is conducted as suggested below. In most instances probing and laying open the track can be repeated until the entire track is laid open. Pursuing this course, if there is no internal opening the track will become bereft of granulations on wiping it. As a rule the internal opening can be demonstrated either by direct inspection through a proctoscope, or by a bent probe inserted into an anal crypt. In the latter circumstance, the internal portion of the track is excised in continuity.

Step 4.—The edges of the track are trimmed, 1 to 3 mm of tissue being removed —a step that makes post-operative packing unnecessary after the first twenty-four to thirty-six hours. E. Hughes advocates primary split skin grafting of the wound resulting from fistulotomy. The grafts are taken from the inner aspect of the thigh and applied to the anal wound, being stitched to the skin edges and to each other in the

Cuthbert Esquire Dukes, 1890–1977. Pathologist, St. Mark's Hospital, London.
Leslie Ernest Hughes, Contemporary. Professor of Surgery, Welsh National School of Medicine, Cardiff, Wales.
Edward Stuart Reginald Hughes, Contemporary. Surgeon, Melbourne, Victoria, Australia.

depths of the wound. Tulle gras is then superimposed, and a firm pack of cotton-wool applied. The first dressing is done on the fifth post-operative day.

When skin grafting is not employed, digital dilatation of the anus, or the passage of a St. Mark's Hospital dilator, every other day prevents pocketing or bridging of the granulating wound.

High-level Fistulæ

The treatment of these cases is difficult. If the track is laid open as for low-level fistulæ, incontinence will follow. There are three types (Parks):

(1) *Supralevator fistula—secondary* to local disease (type 3, fig. 1231).—It occurs as a result of Crohn's disease, ulcerative colitis, carcinoma, foreign body perforating the rectal ampulla from above, or trauma. This fistula is quite unrelated to the ordinary type and the treatment is that of the cause. A traumatic fistula usually needs a colostomy. None of these fistulæ requires to be laid open, which would in any case cause incontinence.

(2) *Transphincteric fistula* (type 2, fig. 1231) with *perforating secondary track*.—The condition starts as an *intersphincteric* fistula (type 1, fig. 1231), often with a high secondary track in the ischio-rectal fossa up to the levator ani. Here lies the danger. Although the anal opening may be low, during exploration of the high secondary track, unless great care is taken, the probe can be pushed through the levator ani into the rectal ampulla, thus converting a low fistula into a high-level type. Treatment should first of all be directed to the low transphincteric fistula and healing of the upper track may follow. If it fails to do so, or if the opening into the rectum is of any size or near the ano-rectal bundle a colostomy must be done before complete healing will take place. A seton (i.e., a ligature of silk or linen) is rarely used, but is helpful when the internal opening is near the ano-rectal ring. Insertion of a seton and subsequent re-examination of the patient without anæsthesia will establish whether the internal opening is situated so near to the ano-rectal ring that incontinence would result if the track was laid open. Under these conditions, a staged operation and a covering colostomy would be the proper treatment.

(3) *High Intersphincteric Fistula.*—The track starts as a primary anal gland abscess (type A, fig. 1225), and it runs between the internal and external sphincter along the plane of the longitudinal muscle fibres. It may have an opening into the rectum above the ano-rectal ring and below at the site of a perianal abscess (type B, fig. 1225). Providing it is recognised it is easy to treat. The internal sphincter is divided and the whole track is laid open without fear of incontinence.

NON-MALIGNANT STRICTURES

1. **Congenital:**

(*a*) A stricture at the level of the anal valves, due to incomplete obliteration of the proctodeal membrane, sometimes does not give rise to symptoms until early childhood.

(*b*) Patients who have had an operation for imperforate anus in infancy may require periodic ano-rectal dilatation.

2. **Spasmodic :**

(*a*) An anal fissure causes spasm of the internal sphincter, which in time becomes fibrotic.

(*b*) Rarely, a spasmodic stricture accompanies Secondary Megacolon (p. 994).

3. **Organic :**

(*a*) *Post-operative stricture* sometimes follows hæmorrhoidectomy performed incorrectly, and also occurs after sleeve resection of a portion of the rectum for a neoplasm.

(*b*) *Irradiation stricture* is an aftermath of irradiation proctitis.

(*c*) *Senile Anal Stenosis.*—A condition of chronic internal sphincter contraction is sometimes seen in the aged. Increasing constipation is present with pronounced straining at stool. Fæcal impaction is liable to occur. The muscle is rigid and feels like a tight umbrella-ring. There is no evidence of a fissure-in-ano. The treatment is internal sphincterotomy or dilation at frequent intervals.

(*d*) *Lymphogranuloma Inguinale* (p. 1310).—This is by far the most frequent cause of a *tubular* inflammatory stricture of the rectum, and 80% of the sufferers are women. Frei's reaction is usually positive. This variety of rectal stricture is particularly common in Negro races, and may be accompanied by elephantiasis of the labia majora. In the early stages, antibiotic treatment may lead to cure. In advanced cases excision of the rectum is required.

(*e*) *Ulcerative Colitis.*—Stricture of the rectum also complicates ulcerative proctocolitis; in this instance the stricture is *annular*, and often more than one are present. A carcinoma should be suspected if a stricture is found.

(*f*) *Endometriosis* of the recto-vaginal septum may present as a stricture. There is usually a history of frequent menstrual periods with the appearance of severe pain during the first two days of the menstrual flow (p. 1115).

(*g*) *Neoplastic.*—When free bleeding occurs after dilatation of a supposed inflammatory stricture, carcinoma should be suspected (Grey Turner), and a portion of the stricture should be removed for biopsy. Sometimes, in these cases, repeated biopsies show inflammatory tissue only. If, however, the symptoms show a marked progression, malignancy should be strongly suspected.

Clinical Features.—Increasing difficulty in defæcation is the leading symptom. The patient finds that increasingly large doses of aperients are required, and if the stools are formed, they are ' pipe-stem ' in shape. In cases of inflammatory stricture, tenesmus, bleeding, and the passage of mucopus are superadded. Sometimes the patient comes under observation only when subacute or acute intestinal obstruction has supervened.

Rectal Examination.—The finger encounters a sharply defined shelf-like interruption of the lumen at a varying distance from the anus. If the calibre is large enough to admit the finger, it should be noted whether the stricture is annular (fig. 1237) or tubular. Sometimes this point can be determined only after dilatation. A biopsy of the stricture should be taken.

George Grey Turner, 1877–1951. Professor of Surgery, Postgraduate Medical School, London.

Treatment:

Prophylactic.—The passage of an anal dilator during convalescence after hæmorrhoidectomy greatly reduces the incidence of post-operative stricture. Efficient treatment of lymphogranuloma inguinale in its early stages should lessen the frequency of rectal stricture from that cause.

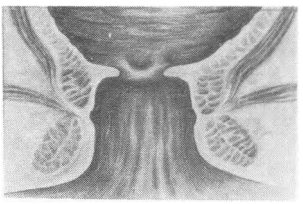


FIG. 1237.—Simple annular stricture of the rectum.

Dilatation by Bougies.—For anal and many rectal strictures dilatation by bougies at regular intervals, combined at first with lavage, is occasionally curative. When the stricture is in the lower two-thirds of the rectum, Hegar's dilators are suitable for this purpose. *Stricture of the upper third of the rectum should never be treated by dilatation* because of the risk of extra- or intra-peritoneal perforation of the bowel.

Incision and Primary Free Skin Graft.—For the post-operative and senile strictures this operation gives by far the best results.

The stricture is exposed and divided posteriorly so as to remove about 1 cm of the fibrosed ring. A ' back cut ' is made and a triangular piece of skin removed with its apex above in such a way as to ensure complete and adequate enlargement of the strictured region. A split skin graft is then taken from the inner side of the thigh, laid firmly into the defect, and sutured into position with fine catgut sutures. Greasy dressings are applied to keep it firmly in position and renewed on about the fourth day when the bowels are allowed to act. The percentage ' take ' is high and the results surprisingly good.

Colostomy must be undertaken when a stricture is situated in the upper third of the rectum, when it is causing intestinal obstruction, and in advanced cases of stricture complicated by fistulæ-in-ano. In selected cases this can be followed by restorative resection of the stricture-bearing area. If this step is anticipated, the colostomy is placed in the transverse colon.

Excision of the rectum is required in some resistant cases due to lymphogranuloma inguinale, especially when the associated proctitis persists in spite of general treatment.

BENIGN TUMOURS OF THE RECTUM

The rectum shares the same types of polyp as the colon, and with the sigmoid colon is the most frequent site of polyps (and cancers) in the gastro-intestinal tract. All neoplastic polyps of the colon and rectum (with rare exceptions) have a tendency to become malignant. This tendency is greatly enhanced if the polyp is more than 1 cm. in diameter, shows obvious signs of increasing size and has a sessile rather than a pedunculated shape. For these reasons, removal of all polyps is recommended, and *total* removal is mandatory. Only total removal will give complete histological examination and exclude (or confirm) localized carcinoma-in-situ, and also prevent local recurrence. For these reasons, destruction of ano-rectal tumours by fulguration is not the best treatment, and should be used for only the tiniest polyps. If one or more rectal polyps are discovered on sigmoidoscopic

examination, an air contrast barium enema should be performed as further polyps are frequently found in the colon and treatment may be influenced. *No rectal tumour should be removed until the possibility of a proximal carcinoma has been ruled out, otherwise local implantation of cancer cells may occur in the distally situated rectal wound.*

The rectum shares substantially the same spectrum of polyps as the colon. Polyps are described chiefly in terms of their tissue organisation. For further clinical details the reader is referred to Chapter 49. Certain polyps that are unique to the ano-rectum, or which have certain features relevant to the rectum, are now described:

1. **Juvenile polyp.**—This is a bright red glistening pedunculated sphere ('cherry tumour') which is found in infants and children. Occasionally it persists into adult life. It can cause bleeding, or pain if it prolapses during defæcation. It often separates itself, but can be removed easily with forceps or a snare. It has no tendency to malignant change but should be treated if it is causing symptoms. It has a unique histological structure of large mucous-filled spaces covered by a smooth surface of thin rectal cuboidal epithelium.

2. **Metaplastic polyps.**—There are small pinkish sessile polyps 2–4 mm. in diameter and frequently multiple. They are harmless.

3. **Pseudo-polyps.**—These are actually œdematous bosses of mucous membrane. They are usually associated with colitis in the United Kingdom but most inflammatory diseases (including tropical diseases) can cause them. They are more likely to cause radiological difficulty as the sigmoidoscopic appearances are usually associated with obvious signs of the inflammatory cause.

4. **Villous adenomas.**—These have a characteristic frond-like appearance. They are often of very large size, and occasionally fill the entire rectum. The large tumours have an enhanced tendency to become malignant—a change that can be detected most easily by palpation with the finger; any hard area should be assumed to be malignant and should be biopsied.

Providing cancerous change has been excluded, these tumours can be removed either by submucous dissection per anum, or by sleeve resection from above. Only very unusually is rectal excision required, and then only when malignant change has occurred.

Rarely, the profuse mucous discharge from these tumours which is high in potassium content causes dangerous electrolyte and fluid losses.

All neoplastic polyps can be solitary or multiple. Small colonic polyps (<5 cm in size) can now be snared through the colonoscope. This new instrument has revolutionised the treatment of multiple polyps.

5. **Familial polyposis coli.**—This disease usually manifests itself by the development of multiple rectal polyps around puberty (p. 1017). An air-contrast barium enema will confirm the presence of multiple colonic polyps. As this condition is pre-malignant, a total colectomy must be performed, but sometimes the rectum can be preserved by regular fulguration of polyps before they develop carcinoma.

Differential Diagnosis.—In patients who have lived in Egypt or any country where bilharzial infestation is rife, bilharzial papilloma must be excluded (p. 1103).

Treatment.—Diathermy coagulation is satisfactory in the case of a small papilloma, but the patient must be examined at regular intervals, for recurrence is common, as in the case of the bladder. For large papillomas, especially the sessile variety, excision of the rectum is the only curative treatment. Some cases (not, as a rule, those invading the anal canal) are suitable for conservative resection of the rectum.

Benign lymphoma, which occurs as a circumscribed movable nodule, firm but not hard, and greyish-white to pink in colour, is essentially submucosal. This neoplasm, which occurs at all ages and in both sexes, has no definite capsule. Notwithstanding, complete local excision is curative.

Endometrioma is not exceedingly rare, and as a rule it is diagnosed as a carcinoma. This neoplasm produces either a constricting lesion of the recto-sigmoid, or a tumour invading the rectum from the recto-vaginal septum. The latter variety gives rise to a very tender submucous elevation of the rectal wall. Endometrioma occurs usually between twenty and forty years of age; less often at the menopause. Dysmenorrhœa with rectal bleeding are the main symptoms. On sigmoidoscopy endometriosis involving the recto-sigmoid junction usually presents as a stricture with the mucous membrane intact. Bilateral öophorectomy may be followed by regression of the tumour, rendering resection unnecessary. The contraceptive 'Pill' is also effective as it inhibits ovulation.

Hæmangioma of the rectum, which is an uncommon tumour, is a cause of serious and, if the neoplasm is large, sometimes fatal hæmorrhage. When localised in the lower part of the rectum or anal canal, a hæmangioma can be excised after applying Goodsall's ligature (p. 1099). When the neoplasm is diffuse, or lying in the upper part of the rectum, the symptoms simulate ulcerative colitis, and often the diagnosis is missed for a long period. At other times the neoplasm is mistaken for a vascular carcinoma, an error which, fortunately, is not often a cause for serious regret, because the correct treatment of an extensive hæmangioma is excision of that portion of the ano-rectum bearing the neoplasm. Lesser procedures are followed nearly always by recurrence and renewed loss of blood (p. 995).

Fibroma (*syn.* fibrous polypus) is not uncommon. *It is not a neoplasm*, but is due to fibrosis of a thrombosed hæmorrhoid.

CARCINOMA OF THE RECTUM

This is the fourth most common variety of malignant tumour found in women, and its frequency in men is surpassed only by carcinoma of the bronchus and stomach.

Origin.—The carcinoma commences as a nodule of atypical columnar epithelium, the rapidly proliferating cells of which extend on the surface by exuberant growth at its edges. Similar changes occur on the deep (submucosal) surface of the nodule. The less malignant varieties continue to extend towards the lumen; the more malignant varieties soon become necrotic in their centre, and give place to an ulcer with indurated, everted edges. In about 30 per cent. of cases, operation specimens show that in some part of the bowel that has been removed, in addition to the carcinoma, there are one or more adenomas or papillomas, proof indeed that adenoma and papilloma of the rectum are pre-carcinomatous conditions. In approximately 3 per cent. of cases there is more than one carcinoma present.

Pathological Histology.—Three types are recognised:

(1) Well differentiated adenocarcinoma; (2) Averagely differentiated adenocarcinoma; (3) Anaplastic, highly undifferentiated adenocarcinoma.

The more malignant varieties frequently contain large numbers of mucin producing cells. The prognosis after treatment is greatly influenced by the histological grading of the tumour (p. 1117)

Usually these carcinomas present as an ulcer, but papilliferous and infiltrating types are not uncommon.

Local spread occurs circumferentially rather than in a longitudinal direction. Usually a period of six months is required for involvement of one-quarter of the circumference, and eighteen months to two years for complete encirclement, the annular variety being common at the recto-sigmoid junction. After the muscular coat has been penetrated the growth spreads into the underlying fat, but is still limited by the fascia propria (peri-rectal fascia). Eventually the fascia propria is penetrated but this occurrence is rare before eighteen months from the commencement of the disease. If penetration occurs anteriorly, the prostate, seminal vesicles, or the bladder become involved in the male; in the female the vagina or the uterus are invaded. In either sex, if the penetration is lateral, a ureter may become implicated, while posterior penetration involves the sacrum and the sacral plexus. Downward spread for more than a few centimetres is rare except in anaplastic tumours.

Lymphatic Spread.—Enlargement of lymph nodes from bacterial infection is more frequent than enlargement from metastasis, and microscopical examination is required to detect carcinomatous involvement of the nodes.

Lymphatic spread from a carcinoma of the rectum above the peritoneal reflexion occurs almost exclusively in an *upward* direction; below that level to within 1 to 2 cm of the anal orifice the lymphatic spread is still *upwards*, but the first halting place is in the para-rectal lymph nodes of Gerota. The exception to this rule is when the neoplasm lies within the field of the middle rectal artery, i.e. between 4 and 8 cm from the anus, in which case primary *lateral* spread along the lymphatics that accompany the middle rectal vein is not infrequent.

Downward spread is exceptional, drainage along the subcutaneous lymphatics to the groins being confined, for practical purposes, to the lymph nodes draining the perianal rosette and the epithelium lining the distal 1 to 2 cm of the anal canal.

Metastasis at a higher level than the main trunk of the superior rectal artery occurs only late in the disease. A radical operation should ensure that the high-lying lymph nodes are removed by ligating the inferior mesenteric artery and vein at the highest possible level.

Atypical and widespread lymphatic permeation can occur in highly undifferentiated neoplasms.

Venous Spread.—As a rule, spread via the venous system occurs late, except in that portion of the anal canal where the anoderm is firmly adherent to deeper structures (fig. 1187). Anaplastic and rapidly growing tumours in younger patients are much more liable to spread in this way than tumours of relatively low malignancy. The principal sites for blood-borne metastases are: liver (34 per cent.), lungs (22 per cent.), adrenals (11 per cent.). The remaining 33 per cent. is divided among the many other locations where secondary carcinomatous deposits are wont to lodge.

Peritoneal dissemination may follow penetration of the peritoneal coat by a high-lying rectal carcinoma.

Stages of Progression.—As a rule carcinoma of the rectum does not metastasise early. Dukes classifies carcinoma of the rectum into three stages (fig. 1238).

(A) The growth is limited to the rectal wall (15 per cent.).

(B) The growth is extended to the extra-rectal tissues, but no metastasis to the regional lymph nodes (35 per cent.).

(C) There are secondary deposits in the regional lymph nodes (50 per cent.). These are subdivided into C^1 where the local para-rectal lymph nodes alone are involved, and C^2 where the nodes accompanying the supplying blood-vessels are implicated. This does not take into account cases that have metastasised beyond the regional lymph nodes or by way of the venous system.

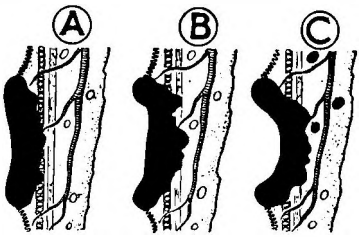


FIG. 1238.—The three cardinal stages of progression of the neoplasm. (*After Cuthbert Dukes, F.R.C.S.*)

Histological Grading.—In the great majority of cases carcinoma of the rectum is a columnar-celled adenocarcinoma. The more nearly the tumour cells approach normal shape and arrangement, the less malignant is the tumour. Conversely, the greater the percentage of cells of an embryonic or undifferentiated type, the more malignant is the tumour:—

Low grade = well-differentiated tumours	11 per cent.	Prognosis good.
Average grade	64 per cent.	,, fair.
High grade = anaplastic tumours . .	25 per cent.	,, poor.

Colloid carcinoma is present in 12 per cent. of cases. There are two forms—primary and secondary; much the more frequent is secondary mucoid degeneration of an adenocarcinoma. Histologically the glandular arrangement is preserved and mucus fills the acini. This type is of average malignancy. In a small number of cases the tumour is a primary mucoid carcinoma. The mucus lies within the cells, displacing the nucleus to the periphery, like the seal of a signet ring. Primary mucoid carcinoma gives rise to a rapidly growing bulky growth which metastasises very early and the prognosis of which is very bad. See also Colloid Carcinoma arising in a fistula-in-ano (p. 1109).

Clinical Features.—Carcinoma of the rectum is not uncommon early in life, and when the disease commences in youth, in spite of radical treatment death usually results within a year. Usually the early symptoms are so slight that the patient does not seek advice for six months or more.

Bleeding is the earliest and most constant symptom. There is nothing characteristic about the time at which it occurs, neither is the colour nor the amount of blood distinctive: often the bleeding is slight in amount, and occurs at the end of defæcation, or is noticed because it has stained underclothing. Indeed, more often than not, the bleeding in every respect simulates that of internal hæmorrhoids[1], and it is lamentable that, in spite of oft-repeated exhortations, the patient's doctor sometimes fails to examine the rectum but prescribes a salve while the growth advances to inoperability (footnote, p. 1060).

[1] Hæmorrhoids and carcinoma sometimes co-exist (fig. 1207).

Cuthbert Esquire Dukes, 1890–1977. (*See footnote p. 1110.*)

Sense of Incomplete Defæcation.—The patient has his bowels open but feels that there are more fæces to be passed. This is a very important early symptom[1] and is almost invariably present in tumours of the lower half of the rectum. The patient may endeavour to empty the rectum several times a day (spurious diarrhœa), often with the passage of flatus and a little blood-stained mucus (' bloody slime ').

Alteration in bowel habit is the next most frequent symptom, and the commonest deviation from normality is increasing constipation. The patient finds it necessary to start taking an aperient, or to supplement his usual dose, and as a result a tendency towards diarrhœa ensues. A patient who has to get up before the accustomed hour in order to defæcate, and one who passes blood and mucus in addition to fæces[2], is usually found to be suffering from carcinoma of the rectum. Usually it is the patient with an annular carcinoma at the pelvi-rectal junction who suffers with increasing constipation, and the one with a cauliflower growth in the ampulla of the rectum with early morning diarrhœa (Bruce).

Pain is a late symptom, but pain of a colicky character accompanies advanced growths of the recto-sigmoid and is due to some degree of intestinal obstruction. When a deep carcinomatous ulcer of the rectum erodes the prostate or bladder, there is severe pain. Pain in the back, or sciatica, occurs when the growth invades the sacral plexus. Weight loss is suggestive of hepatic metastases.

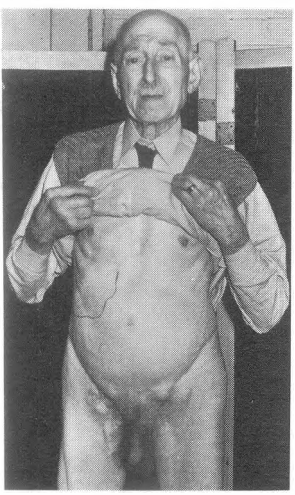


FIG. 1239.—Massive irregular enlargement of the liver. Rectal examination revealed a carcinomatous ulcer on the right side of the ano-rectal junction. Hard lymph node in right groin. The patient also has a left inguinal hernia and a right saphenous varix.

Abdominal examination is negative in early cases. Occasionally when an advanced annular growth is situated at the recto-sigmoid junction, signs of obstruction to the large intestine are likely to be present. By the time the patient seeks advice metastases in the liver may be palpable (fig. 1239). When the peritoneum has become studded with secondary deposits, ascites results.

Rectal Examination.—In approximately 90 per cent. of cases the neoplasm can be felt digitally: in early cases as a plateau or as a nodule with an indurated base. When the centre ulcerates, a shallow depression will be found, the edges of which are raised and everted; this, combined with induration of the base of the ulcer, is a frequent and unmistakable finding. On bimanual examination it may be possible to feel the lower extremity of a carcinoma situated in the recto-sigmoid junction. After the finger has been withdrawn, if it has been in direct contact with a carcinoma, it is smeared with blood, or mucopurulent material tinged with blood. When a car-

[1] *Tenesmus* is painful straining to empty the bowels without resultant evacuation.
[2] ' Early morning bloody diarrhœa.'

Sir John Bruce, 1905–1975. Regius Professor of Clinical Surgery, University of Edinburgh.

cinomatous ulcer is situated in the lower third of the rectum, involved lymph nodes can sometimes be felt as one or more hard oval swellings in the extra-rectal tissues posteriorly or posterolaterally above the tumour. In females a vaginal examination should be performed, and when the neoplasm is situated on the anterior wall of the rectum, with one finger in the vagina and another in the rectum, very accurate palpation can be carried out.

Procto-sigmoidoscopy in the complete knee-elbow position (fig. 1194) will always show a carcinoma, if present.

Biopsy.—Employing biopsy forceps (fig. 1240) by way of a proctoscope, a portion of the edge of the tumour is removed. If possible another specimen from the more central part of the growth is obtained also. Expert histological examination will not only enable the diagnosis of carcinoma to be confirmed, but the tumour can be graded as to its relative malignancy.

Barium enema is required (*a*) in rare instances when sigmoidoscopy is unsatisfactory because of spasm, (*b*) in cases of suspected carcinoma of the pelvi-rectal junction when sigmoidoscopy fails to reveal the growth because of spasm of the bowel below it, (*c*) when multiple adenomatous polyposis of the colon must be excluded and (*d*) when practical, to exclude other synchronous carcinomas (5% of cases).

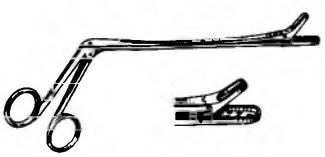


FIG. 1240.—Yeomans' biopsy forceps.

Differential Diagnosis.—When a seemingly benign **papilloma** or **adenoma** shows evidence of induration or friability, it is certain that malignancy has occurred, even in spite of biopsy findings to the contrary. On the other hand, biopsy is invaluable in distinguishing carcinoma from an **inflammatory stricture** or an **amœbic granuloma,** which simulates a carcinoma very closely. The possibility of a neoplasm being an **endometrioma** should always be entertained in patients with dysmenorrhœa. Finally, the importance of bearing in mind the possibility of a **carcinoid tumour** in atypical cases has already been emphasised. In the last four instances biopsy will frequently establish the correct diagnosis. The **solitary ulcer** syndrome has already been alluded to (p. 1104).

Treatment

Some form of excision of the rectum is essential, if at all possible, because of the extreme suffering entailed if the neoplasm remains.

Apart from co-existent disease or senile enfeeblement, the only prohibitions to excision of the rectum are widespread distant metastases and extensive peritoneal deposits. Many instances have been reported where a presumed solitary metastasis in the liver has been resected, either at the time of excision of the rectum, or subsequently. Even when metastases in the liver are irremovable, resection of the rectum is often justifiable, and the patient may survive in comfort for a year or two.

A combined (abdominal and perineal) excision offers the best prospect of eradicating the disease for tumours in the lower half of the rectum. For those in the upper half of the rectum a restorative type of operation (anterior resection) is usually possible. Because of the much wider degree of local spread by anaplastic tumours and the high risk of local recurrence, restorative operations are not usually done for this group of lesions.

The combined operation can be carried out as an abdomino-perineal procedure (Miles), by which is meant that the abdominal part of the operation

William Ernest Miles, 1869–1947. Surgeon, Royal Cancer Hospital (now the Royal Marsden Hospital), London.

is undertaken first, or a perineo-abdominal (Gabriel), where the perineal stage is performed before the abdominal stage, or thirdly, as a synchronised procedure, where two operating teams work simultaneously.

Five days pre-operative sterilisation of the alimentary tract by sulphasuccidine is required. The colon is cleansed by enemas or a colon wash as necessary. Blood and electrolyte deficiencies are corrected. Before commencing the operation, an indwelling catheter is inserted into the bladder. In male patients a full sized catheter is preferred as damage to the urethra is thereby made less likely during dissection anterior to the rectum.

Combined Synchronised Excision of the Rectum.—This is the operation which is now commonly performed. It combines the advantages of both the abdomino-perineal and the perineo-abdominal operations. With the patient in Trendelenburg-lithotomy position, the legs being supported in special crutches designed by Lloyd-Davies access is afforded to the abdomen and the perineum at the same time. Two surgeons operate simultaneously, one performing the perineal dissection and the other the abdominal portion of the operation. This considerably reduces the time expended in performing the operation, and obviates turning the patient. The resectability rate is increased by this operation.

The *Abdominal Surgeon* makes a left paramedian incision, extending it well above the umbilicus. The liver and the peritoneum are examined for metastases and the degree of fixity of the growth established. The small intestine is packed away from the pelvis. A self-retaining retractor is placed in the wound and the pelvic colon freed by dividing any congenital adhesions on the left side. The peritoneum and the pelvic floor are divided with a knife by an incision which runs from the colon at the proposed site of division over the mesocolon and across the base of the bladder or near the cervix on the peritoneal floor and then upwards on the right side of the mesocolon. The peritoneum is now raised, using the points of the scissors to expose the ureters and testicular or ovarian artery. The mesocolon is now divided at the site of the proposed division on the colon and the trunk of the inferior mesenteric artery (fig. 1241) ligated and divided distal to the first branch. (Some surgeons emphasise ' flush ligation ' of the artery at its origin from the aorta.) The recto-sigmoid mesentery is further divided and separated from the sacrum by blunt dissection with the fingers. In this way the sacrum is cleared almost down to the coccyx. The peritoneal incision anterior to the rectum is now deepened and the seminal vesicles or the vaginal wall is identified so that the fascia behind them is cleared by a dissection leading down to the prostate or perineal body. The middle rectal vessels usually lying anterior to the lateral ligaments on each side are now seized with clamps, divided and ligated. The site of division of the pelvic colon is cleared of fat and the colon divided between clamps with diathermy.

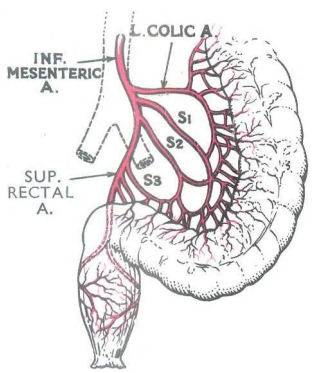


FIG. 1241.—The inferior mesenteric artery. S_1 S_2 S_3 = sigmoid branches.

By this time the perineal surgeon working from below has mobilised the anus and the lower rectum so that the whole of the bowel below together with a clamp can be passed through the perineal wound by the abdominal surgeon. Hæmostasis over the sacrum may be difficult but it is achieved by diathermy and a hot saline pack left in position for a few minutes. The pelvic peritoneum is now united by continuous catgut stitches from the bladder right back over the promontory of the sacrum (fig. 1242).

The site in the L.I.F. for the colostomy is then selected. This should be equidistant from the umbilicus and the left anterior superior iliac spine and is

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig, Germany.
Oswald Vaughan Lloyd-Davies, Contemporary. Surgeon, Middlesex Hospital, London.

usually sited at the linea semilunaris about 2·5 cm above the spino-umbilical line. A circular piece of skin and fascia, about 3 cm in diameter, is incised and this hole deepened to excise similar layers of fascia and peritoneum. The protected end of the colon with the clamp is now passed through this incision and the colostomy performed by suturing the colon to the peritoneum and the mucosa directly to the skin. The paracolic gutter is closed with sutures—this will close the 'lateral space'. The abdomen is closed and the layers of the incision protected from the colostomy. An adherent plastic colostomy bag is then fitted in position and the dressings are placed on the abdominal wound.

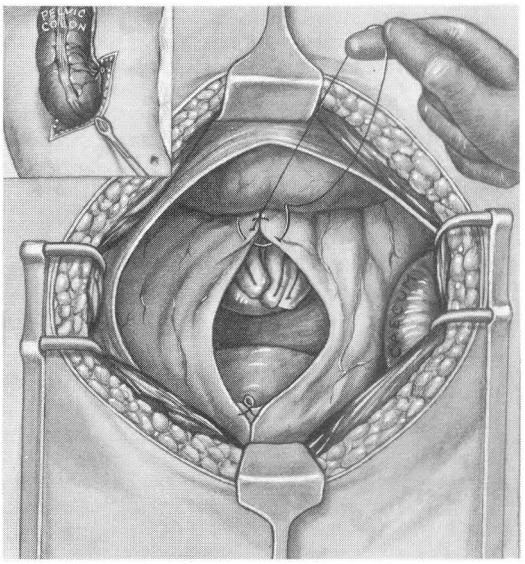


FIG. 1242.—The cut edges of the pelvic peritoneum are being united by the abdominal surgeon over the space (filled by packing) where the perineal surgeon is working. *Inset:* The rectum and pelvic colon drawn through the left iliac incision to form a terminal colostomy.

Perineal Surgeon.—When the abdominal surgeon has made certain that the condition is operable, the perineal surgeon closes the anus with purse-string sutures of stout silk. An elliptical incision between the tip of the coccyx and the central perineal point is made around the anus and deepened. The left forefinger is insinuated into the levator ani which is divided lateral to the finger first on one side and then on the other. The dissection is deepened posteriorly by incising Waldeyer's fascia which is a thick condensation of pelvic fascia lying between the rectum and the sacrum. Contact is made with the abdominal surgeon. The apex of skin anterior to the anus is grasped in a hæmostat, which serves as a retractor, and by scissors and gauze dissection the wound is deepened, when the catheter within the membranous urethra will be felt. Both in the male and the female a plane of cleavage will be found between the rectum and the prostate or the rectum and the vagina, respectively. This plane having been carefully determined, the strong median raphé of the perineum is divided, after which the rectum can be stripped from the prostate or the vagina. The posterior wall of the vagina is frequently excised with the rectum. When the abdominal surgeon has cleared the rectum laterally, the whole of the anus and rectum can be drawn downwards and removed. Hæmostasis must be secured and the perineal wound closed anteriorly and posteriorly in layers around a large drainage tube. Large dressings of gauze and wool are applied over the area and a triangular bandage is used to keep the dressing in place. It is becoming a more common practice in some centres to employ *primary closure* of the perineal wound, and use laterally-situated suction drains brought out through each ischio-rectal fossa to keep the large perineal cavity from filling up with blood and serous exudate. These drains can be removed after 5 days.

After Treatment.—The patient is returned to bed, blood transfusion being continued as necessary. The catheter is connected to a closed catheter drainage system (fig. 1383) and left in for five days. It may have to be re-inserted if voluntary micturition is not re-established; carbachol may be necessary to establish this.

Reactionary hæmorrhage from the perineal wound may demand return to the theatre to open and pack the wound with gauze. The colour of the colostomy must be watched to make sure the blood supply is adequate. Small bowel obstruction may occur by herniation in the lateral space of the colostomy or through the pelvic peritoneal closure line. Discharge of urine from the perineal wound demands immediate investigation for bladder or ureteric damage.

Heinrich Wilhelm Gottfried Waldeyer-Hartz, 1836–1921. Professor of Pathological Anatomy, Berlin.

Care of the Colostomy.—This is much the same as the care of an ileostomy (p. 1007). Within a very short time the colostomy acts once or twice a day. The patient soon learns which foods cause diarrhœa and he therefore avoids them.

Stenosis of the colostomy is usually avoided by the removal of the circle of skin and subcutaneous tissues at the colostomy site. Anal dilators (fig. 1206) may be necessary if there is any tendency for stenosis to occur (see also pp. 1026–1028).

Less Extensive Operations :

Abdominal Radical Restorative Resection (anterior resection).—In cases of carcinoma of the rectum situated above the peritoneal reflection, lymphatic spread is virtually confined to the upward paths, and wide resection of the bowel with its lymphatic field, followed by end-to-end anastomosis and preservation of the sphincter mechanism is both justifiable and highly desirable.

This apparently ideal treatment was, and still is, unpopular in many centres because it has been found that local recurrence occurred in a formidable percentage of cases. It has now been substantiated that the so-called recurrences are due, for the most part, to local implantation of free malignant cells into the distal segment that is to be preserved, the survival and rooting of these cells being favoured by disinfection of the intestine by sulphasuccidine and antibiotics. Naunton Morgan has shown that when a suitable clamp is placed at least 5 cm below the neoplasm, and the rectum is irrigated with buffered Dakin's solution (Chlorinated soda–0·5 per cent. available chlorine), and the lumen of the proximal end and the edges of both segments are swabbed with the same solution before anastomosis, the incidence of local recurrence is reduced considerably.

Restorative resection should be performed only when the tumour is high in the rectum, i.e. at least 10 cm above the anal orifice, and even then it should not be attempted if the build of the patient, and/or the anatomy of the colon and its vascular supply, makes restoration of the continuity of the alimentary canal difficult, nor in a young subject in whom these neoplasms grow rapidly and recur.

The perineal operation (Lockhart-Mummery) does not remove the lymphatic field in relation to the inferior mesenteric artery, and it is reserved mainly as a palliative for poor-risk patients when the neoplasm is situated in the lower third of the rectum.

Hartmann's Operation.—This is an excellent procedure in an old and feeble patient who would not stand an abdomino-perineal procedure. Through an abdominal incision the rectum is excised down to within an inch of the anus, a colostomy performed and the peritoneum oversewn to cover the pelvic defect in the usual way. The cavity below can be drained through the anus, which is divided posteriorly. In the old patient, where the neoplasm is usually slow growing and spread is late, this is a most useful operation.

Palliative colostomy is indicated only in cases giving rise to intestinal obstruction, or where there is gross infection of the neoplasm. It is often possible to resect the growth later.

Local Operations.—For small, low-grade mobile lesions, which are often Grade A tumours, local removal should be curative. For these tumours, especially in the unfit or patients who will not accept a colostomy, local removal has been used. Such operations are only suitable for lesions within 10 cm of the anal verge. Turnbull has advocated local diathermy removal while York-Mason has developed a trans-anal (trans-sphincteric) approach.

More Extensive Operations.—When the carcinoma of the rectum has spread to contiguous organs, the radical operation can often be extended to remove these structures. Thus in the male, where the spread is usually to the bladder, a total cystectomy and resection of the rectum can be affected. In the female the uterus acts as a barrier preventing spread from the rectum to the bladder. Accordingly, a total hysterectomy should be undertaken in addition to excision of the rectum. Should the bladder be involved, then pelvic evisceration must include that structure. Pelvic evisceration for carcinoma of the rectum is justifiable only when the surgeon is reasonably confident that the growth can be removed *in toto*.

John Percy Lockhart-Mummery, 1875–1957. Surgeon, St. Mark's Hospital, London.
Henri Albert Charles Antoine Hartmann, 1860–1952. Professor of Surgery, Paris.
R. Turnbull, Contemporary. Surgeon, Cleveland Clinic, Cleveland, Ohio, U.S.A.
Aubrey York-Mason, Contemporary. Surgeon, St. Helier Hospital, Carshalton, Surrey.

Pelvic Evisceration (Brunschwig's operation).—The aim is to remove all the pelvic organs, together with the internal iliac and the obturator groups of lymph nodes (fig. 1243). The lithotomy-Trendelenburg position facilitates the procedure, and ligation of both internal iliac arteries diminishes the blood loss. The small intestine fills the empty pelvis. Especial care must therefore be taken to suture accurately the perineal skin, and to avoid pressure necrosis of the perineal incision by nursing the patient on alternate sides. Some form of urinary diversion is necessary (p. 1258).

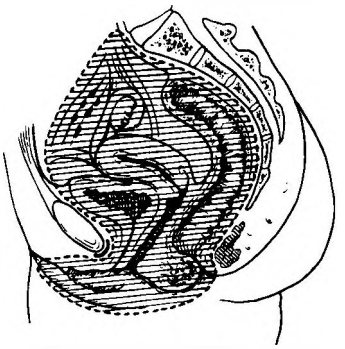


FIG. 1243.—Radical pelvic evisceration, indicating the extent of the dissection and the viscera removed.

Carcinoid Tumour.—Carcinoid tumour of the rectum, as far as its lethal properties are concerned, can be looked upon as a gradation between a benign tumour and a carcinoma. A number of carcinoid tumours of the rectum have been reported during recent years so that a latter-day aphorism is ' keep carcinoid in mind when an atypical neoplasm of the rectum is encountered '. Like benign lymphoma, carcinoid tumour originates in the submucosa, the mucous membrane over it being intact. Consequently it seldom produces evidence of its presence in the early stages, when it appears as a small plaque-like elevation. The incidence of clinical malignancy, i.e. the occurrence of metastases, is 10 per cent. This is much less than that for carcinoid tumour of the small intestine (p. 1016) but it is greater than that of carcinoid tumour of the vermiform appendix (p. 1074). Multiple primary carcinoid tumours of the rectum are not infrequent. The neoplasm is slow of progression, and usually metastasises late.

Treatment.—Resection of the rectum is advisable if the growth is more than 2·5 cm. in diameter, if recurrence follows local excision, or if the growth is fixed to the peri-rectal tissues. Even when metastases are present, resection may prolong life.

Radiotherapy.—With modern techniques (MV cobalt therapy or neutron beam irradiation) some adenocarcinomas now respond to radiotherapy. Some large tumours can be shrunk as a pre-operative measure to make removal easier, and palliative irradiation can be given for inoperable primary tumours or local recurrence. Papillon claims cures of early low rectal cancer by direct intracavitary irradiation.

Cytotoxic and Chemotherapeutic Drugs.—These are still under evaluation, but have proved to be of little effect so far.

MALIGNANT TUMOURS OF THE ANUS

Carcinoma of the anus differs from carcinoma of the rectum in histological structure, behaviour, and the requisites for treatment. This is mainly because of its abundant lymph drainage, both superficial and deep (fig. 1192).

Squamous-celled Carcinoma.—Because of its superficial situation the presence of the lesion is recognised by the patient, who often presents early. The exception is when radiation carcinoma develops in the anal and perianal skin of a patient unwisely treated with lightly filtered X-rays for pruritus ani. The chronic radiation dermatitis becomes so familiar to the patient that too often he does not perceive the superimposition of carcinoma. Simple papillomas (anal warts) sometimes take on a carcinomatous change (fig. 1244). Anal cancer may also masquerade as an anal fissure (p. 1087). Occasionally a squamous cell carcinoma develops in the track of a long-standing fistula-in-ano.

Basal-celled Carcinoma.—Of twenty-eight cases occurring at the Mayo Clinic, seven were *basaloid* small-celled carcinomas that occurred near the dentate line, and showed a high degree of malignancy. With these exceptions, basal-celled carcinomas are found predominantly at the anal margin and are of relatively low malignancy.

Melanoma.—Melanoma of the anus presents as a bluish-black soft mass that is

Alexander Brunschwig. Formerly Surgeon, Memorial Hospital for the Treatment of Cancer, New York.
J. Papillon, Contemporary. Professor of Radiotherapy, Lyons, France.
The Mayo Clinic, Rochester, Minnesota (footnote, p. 658).

apt to be confused with a thrombotic pile, and therefore unfortunately incised. Such trauma, followed by the trauma of defæcation, incites the tumour to rapid metastasis. Left undisturbed, it ulcerates, and the colour of the tumour changes from blue to black. The inguinal lymph nodes are soon involved. Unless a melanoma is excised at an early stage, it disseminates by the blood-stream.

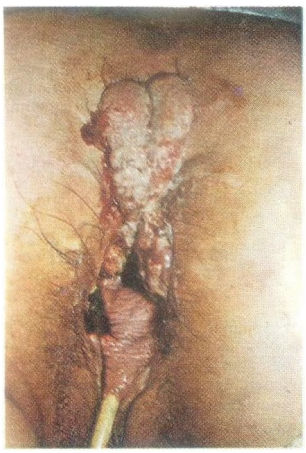


FIG. 1244.—Neglected papillomas of the anus which have become malignant. The patient was a woman of forty-three years of age and symptoms had been present for eight years.

Treatment:

Operation.—For small squamous cell lesions at the anal verge, wide local excision leaving a margin of at least 2·5 cm of tissue all round is occasionally sufficient to effect a cure: in such cases, lymphatic dissemination will be to the inguinal nodes, which should be watched carefully. If they become involved, block dissection removal of the glands of both groins will be necessary.

With the above exception (and for those suitable for treatment by radiotherapy) all other cases, including the basaloid carcinomas, should be treated by:—

An abdomino-perineal excision, removing the growth and perianal area widely. If and when the inguinal lymph nodes become involved, a radical dissection of the groin is carried out. This formidable operation, which is founded on sound premises based on pathology, gives more favourable results than has been generally believed. It should be noted that dissection of the lymph nodes of the groin or groins, is carried out only when involvement of the nodes can be recognised clinically, or when a doubtful lymph node, submitted to frozen section at the time of the operation, proves to be involved. As has been emphasised already, patients with carcinoma of the anus often present early, and in many patients the inguinal lymph nodes are not involved clinically. Should they become so later, dissection of the groin is carried out.

A Super-radical operation comprises removal of the area of skin shown in fig. 1245, together with a block dissection of the inguinal lymph nodes and a radical abdominoperineal excision of the rectum. This operation is very formidable and causes considerable morbidity.

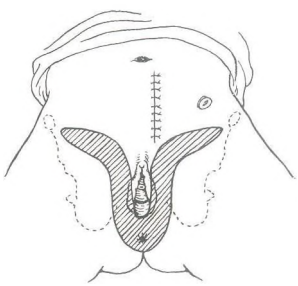


FIG. 1245.—Super-radical operation for anal cancer, showing the area of skin removed. In the male the lower half of the scrotum is included. (*After G. T. Pack and J. C. Baldwin.*)

Radiotherapy.—Either low-voltage contact X-rays or interstitial gold radon seeds, 2 millicuries each, placed 1·5 cm. apart at the base of a small tumour, should be reserved for the following types of cases (Pack):

1. Early papillary or basal-celled carcinoma of low-grade malignancy.
2. Advanced lesions that have recurred after operation.
3. In an inoperable case, with the hope of converting an inoperable carcinoma into one that is resectable.

George Thomas Pack. Formerly Clinical Professor of Surgery, New York Medical College, New York.

CHAPTER 53

THE UMBILICUS AND THE ABDOMINAL WALL (EXCLUDING HERNIA)

THE UMBILICUS

The umbilical cord, formed in the second month of fœtal life from the body stalk, is composed of Wharton's jelly, covered by ectoderm, and contains:

1. **The allantois,** a diverticulum from the yolk sac, is a narrow tube passing from the cloaca through the umbilicus and umbilical cord to end blindly at the placenta. The part within the body forms the urachus and the bladder, except the trigone. The remainder disappears.

2. **The vitello-intestinal duct** is a tubular outgrowth of yolk sac from which the gut is derived (fig. 1246). In 98 per cent. of cases by the time the embryo is six weeks old the vitello-intestinal duct disappears (p. 1127).

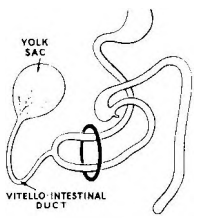


FIG. 1246.—Embryonic canal, showing the vitello-intestinal duct attached to the yolk sac.

3. **The Umbilical Vein.**—Originally two, early in fœtal life the right one atrophies and disappears. The vein carries oxygenated blood from the placenta to the fœtal liver where, having given off branches, it bifurcates (*a*) to join the left branch of the portal vein, and (*b*) to pass on as the ductus venosus to empty its contents into the inferior vena cava.

4. **The umbilical arteries** pass from the internal iliac arteries to, and through, the umbilicus, and, in a spiral fashion, traverse the umbilical cord to join the placental plexus.

The fibrous ring of the umbilicus is situated in the blended aponeurosis of the linea alba at, or below, the centre of that line. In the early days of infancy, while the umbilicus becomes a consolidated scar, the structures just referred to shrink into impervious cords (except that section of the allantois which forms part of the bladder). These cords diverge from the abdominal aspect of the umbilicus into the extraperitoneal fatty tissue, and produce peritoneal folds. Upwards, in the free edge of the falciform ligament, runs the ligamentum teres (the obliterated umbilical vein) to the longitudinal fissure of the liver. Directly downwards to the apex of the bladder runs the urachus (obliterated allantois). Obliquely downwards and outwards pass the obliterated hypogastric (umbilical) arteries to join the internal iliac arteries.

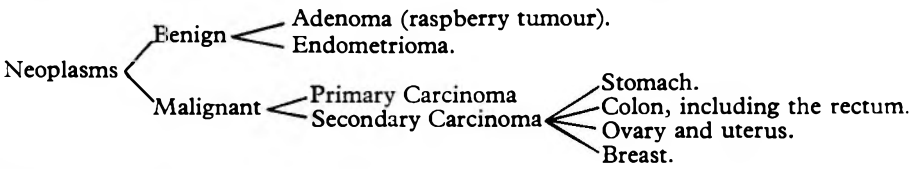
Lymphatics.—The lymphatic vessels drain into both groins and both axillæ.[1]

DISEASES OF THE UMBILICUS

- Inflammations
  - Infection of the stump of the umbilical cord (omphalitis).
  - Umbilical granuloma.
  - Umbilical dermatitis.
  - Pilonidal sinus.
- Fistulæ
  - Fæcal
    - Patent vitello-intestinal duct.
    - Neoplastic ulceration from the transverse colon.
    - Tuberculous peritonitis.
  - Urinary = Patent urachus.
  - Biliary.

[1] A society lady wished to be re-vaccinated ' where the scar wouldn't show '. She selected an area just beneath the umbilicus. Four days later she was confined to bed with painful adenitis of both groins and both axillæ.

Thomas Wharton, 1614–1673. Physician, St. Thomas's Hospital, London.



Hernia (p. 1151).
Umbilical calculus.
Eversion (in ascites).

INFLAMMATIONS

Infection of the Umbilical Cord.—By the third or fourth day of life the stump of the umbilical cord is found to be carrying staphylococci in over 50 per cent. of babies born in maternity hospitals. Less commonly the stump of the cord harbours streptococci, and epidemics of puerperal sepsis in maternity hospitals have been traced to the umbilical cord of one infant in the nursery thus infected. *Esch. coli* and *Cl. Tetani* (causing neonatal tetanus) are other possible invaders. The chief prophylaxis is strict asepsis during severance of the cord, and the use of 0·1% chlorhexidine locally, for a few days.

Omphalitis.—The incidence of an infected umbilicus is much higher in communities that do not practise aseptic severance of the umbilical cord. When the stump of the umbilical cord becomes inflamed, antibiotic therapy usually localises the inflammation. By employing warm moist dressings the crusts separate, giving exit to pus. Exuberant granulation tissue requires a touch of silver nitrate. In more serious cases infection is liable to spread along the defunct hypogastric arteries or umbilical vein, when, in all probability, one or other of the following complications will supervene:

1. **Abscess of the Abdominal Wall.**—If gentle pressure is exerted below or above the navel, and a bead of pus exudes at the umbilicus, a deep abscess associated with one of the defunct umbilical vessels is present. This must be opened. A probe is passed into the sinus to determine its direction and this is followed by a grooved director on to which the skin and overlying tissues are incised in the middle line.
2. **Extensive ulceration of the abdominal wall,** due to a synergic infection, is treated in the same way as post-operative subcutaneous gangrene (p. 1131).
3. **Septicæmia** can occur from organisms entering the blood-stream via the umbilical vein. Jaundice is often the first sign. An abscess in the abdominal wall above the umbilicus should be sought. In other respects the treatment of this grave complication follows the usual lines (p. 28).
4. **Jaundice in the Newborn.**—Infection reaching the liver via the umbilical vein may cause a stenosing intrahepatic cholangiolitis, appearing some three to six weeks after birth.
5. **Portal vein thrombosis** and subsequent portal hypertension (p. 897).
6. **Peritonitis** carries a particularly bad prognosis.
7. **Umbilical Hernia** (p. 1151).

Umbilical Granuloma.—Chronic infection of the umbilical cicatrix which continues for weeks causes granulation tissue to pout at the umbilicus. There is no certain means of distinguishing this condition from an adenoma (p. 1128). Usually an umbilical granuloma can be destroyed by one application of a silver nitrate stick followed by dry dressings but an adenoma soon recurs in spite of these measures (p. 1128).

Dermatitis of and around the umbilicus is common at all times of life. Fungus and parasitic infections are more difficult to eradicate from the umbilicus than from the skin of the abdomen. Sometimes the dermatitis is

consequent upon a discharge from the umbilicus, as is the case when an umbilical fistula or a sinus is present.

A deep, tender swelling in the midline below the umbilicus signifies an abscess present in the extraperitoneal fat, and is usually due to an *infected urachal remnant*. Exploration and proper drainage is necessary.

Pilonidal sinus (a sinus containing a sheaf of hairs) is sometimes encountered. It should be excised.

Umbilical calculus (umbolith), often black in colour, is composed of desquamated epithelium which becomes inspissated and collects in a deep recess of the umbilicus. Eventually it gives rise to inflammation, and often a blood-stained discharge. The treatment is to dilate the orifice and extract the calculus, but, to prevent recurrence, it may be better to excise the umbilicus.

UMBILICAL FISTULÆ

The umbilicus being a central abdominal scar, it is understandable that a slow leak from any viscus is liable to track to the surface at this point (fig. 1247). Added to this, very occasionally, the vitello-intestinal duct or the urachus remains patent; consequently it has been remarked aptly that the umbilicus is a creek into which many fistulous streams may open.

For instance, an enlarged inflamed gall-bladder perforating at its fundus may discharge gall-stones through the umbilicus. Again, an unremitting flow of pus from a fistula at the umbilicus of a middle-aged woman led to the discovery of a length of gauze overlooked during hysterectomy five years previously.

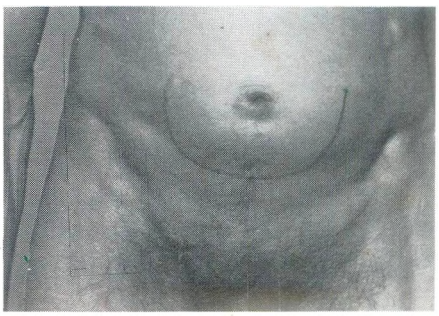


FIG. 1247.—This patient, aged sixty-two, complained only of a purulent discharge from the umbilicus. A lump could be felt in the position outlined. Perforated carcinoma of the transverse colon.

The vitello-intestinal duct occasionally persists, and gives rise to one of the following conditions:

1. It remains patent (figs. 1248 and 1249 (*a*)). The resulting umbilical fistula discharges mucus and, rarely, fæces.

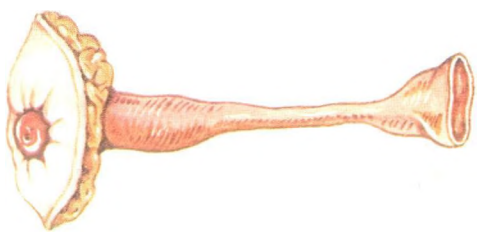


FIG. 1248.—Patent vitello-intestinal duct opening at the umbilicus. (*After A. L. Taylor.*)

(*b*) A small portion only of the duct near the umbilicus remains unobliterated. This gives rise to a sinus that discharges mucus. The epithelial lining of the sinus often becomes everted to form an adenoma (p. 1128).

2. Sometimes both the umbilical and the intestinal ends of the duct close, but the mucous membrane of the intervening portion remains, and an intra-abdominal cyst develops (fig. 1249 (*c*)).

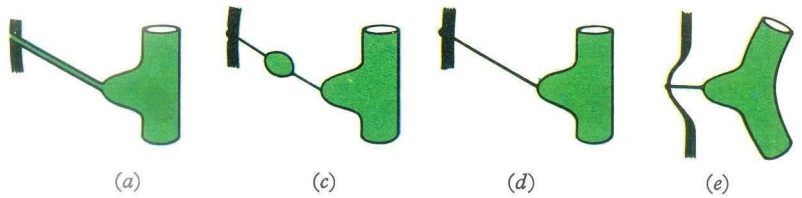


FIG. 1249.—Anomalies connected with the vitello-intestinal duct: (*a*) umbilical fistula; (*b*) umbilical sinus (not illustrated); (*c*) intra-abdominal cyst; (*d*) intraperitoneal band; (*e*) Meckel's diverticulum adherent to the sac of a congenital umbilical hernia.

3. With its lumen obliterated or unobliterated, the vitello-intestinal duct provides an intraperitoneal band (fig. 1249 (*d*)) which is a potential danger, for intestinal obstruction is liable to occur. The obstruction results from a coil of small intestine passing under or over (fig. 1250), or becoming twisted around the band.

FIG. 1250.—Vitello-intestinal duct causing intestinal obstruction.

4. Such a band may contract and pull a Meckel's diverticulum into a congenital umbilical hernia (fig. 1249 (*e*)).

5. A vitello-intestinal cord connected to Meckel's diverticulum, but not attached to the umbilicus, becomes adherent to, or knotted around, another loop of small intestine, and so causes intestinal obstruction (fig. 1250).

6. Sometimes a band extending from the umbilicus is attached to the mesentery near its junction with a distal part of the ileum. In this case the band is probably an obliterated vitelline artery, and is not necessarily associated with a Meckel's diverticulum.

Treatment.—A patent vitello-intestinal duct should be excised, together with a Meckel's diverticulum, if such be present, preferably when the child is about six months old. When a vitello-intestinal band gives rise to acute intestinal obstruction, after removing the obstruction by division of the band, it is expedient, when possible, to excise the band and to bury the cut ends.

A patent urachus seldom reveals itself until maturity, or even old age. This is because the contractions of the bladder commence at the apex of the organ and pass towards the base. A patent urachus, because it opens into the apex of the bladder, is closed temporarily during micturition, and so the potential urinary stream to the umbilicus is cut off. Therefore the fistula remains unobtrusive until a time when the organ is overfull, usually due to some form of obstruction.

Treatment.—Usually treatment is directed to removing the obstruction to the lower urinary tract. If, after this is remedied, the leak continues or a cyst develops in connection with the urachus, umbilectomy and excision of the urachus down to its insertion into the apex of the bladder, with closure of the latter organ, is indicated.

Abscess in a urachal remnant, causing persistent umbilical dermatitis and discharge, is described on p. 1127.

NEOPLASMS OF THE UMBILICUS

Umbilical adenoma or **Raspberry tumour** is commonly seen in infants (fig. 1251), but only occasionally later in life. It is due to a partially (occasionally a completely) unobliterated vitello-intestinal duct. Mucosa

Johann Friedrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle, Germany.

prolapsing through the umbilicus gives rise to a raspberry-like tumour, which is moist with mucus and tends to bleed.

Treatment.—If the tumour is pedunculated, a ligature is tied around it, and in a few days the polypus drops off. Should the tumour reappear after this procedure, umbilectomy is indicated. Sometimes a patent vitello-intestinal duct, or more often a vitello-intestinal band, will be found associated with a Meckel's diverticulum. The Meckel's diverticulum and the attached cord or duct should be excised at the same time as the umbilicus. Histologically, the tumour at the umbilicus consists of columnar epithelium rich in goblet cells.

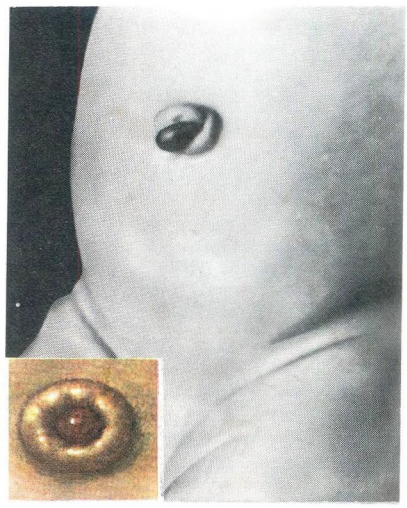


FIG. 1251.—Adenoma (raspberry tumour) of the umbilicus.

Endometrioma occurs in women between the ages of twenty and forty-five. On histological examination it is found to consist of endometrial glands occupying the same plane in the dermis as the sudoriferous glands, and opening on to the surface in the same way. The umbilicus becomes painful and bleeds at each menstruation, when the small fleshy tumour between the folds of the umbilicus becomes more apparent. Occasionally an umbilical endometrioma is accompanied by endometriomas in the uterus or ovary. When, as is usually the case, the tumour is solitary, umbilectomy will cure the condition.

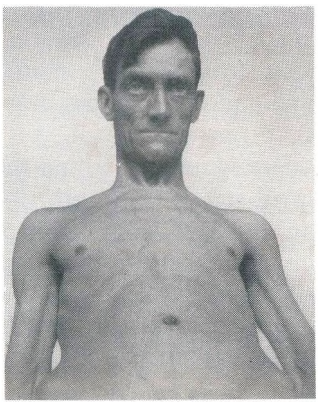


FIG. 1252.—Secondary nodule at the umbilicus in a case of carcinoma of the stomach.

Secondary carcinoma at the umbilicus[1] (fig. 1252) is not very uncommon, but it is always a late manifestation of the disease. The primary neoplasm is often situated in the stomach, colon or ovary, but a metastasis from the breast, probably transmitted along the lymphatics of the round ligament of the liver, is sometimes located here.

THE ABDOMINAL WALL

Tearing of the inferior epigastric artery occurs in three dissimilar types of individual, viz. elderly women, often thin and feeble; athletic, muscular men, usually below middle age; and pregnant women, mainly multiparæ late in pregnancy. The site of the hæmatoma is usually at the level of the arcuate line, where the posterior sheath of the rectus abdominis is lacking.

Clinical Features.—The possibility of tearing of the epigastric vessels

[1] The neoplastic nodule so caused is known as Sister Joseph's nodule. Sister Joseph of the Mayo Clinic imparted this clinical observation to the late Dr. William Mayo.

should always be considered when, following a bout of coughing, or a sudden blow to the abdominal wall, an exquisitely tender lump appears in relation to the rectus abdominis (fig. 1253). Occasionally, a hæmatoma occurs within the muscles lateral to the rectus sheath. Unless there is bruising of the overlying skin the diagnosis may be difficult.

FIG. 1253.—Tearing of the inferior epigastric artery, showing the tell-tale swelling and bruising (see text). (*Dr. C. L. Colton, Bristol.*)

Differential Diagnosis.—The conditions for which the hæmatoma is frequently mistaken are, in the female, a twisted ovarian cyst, and in both sexes, when the lump is on the right side, an appendix abscess. The sign most likely to be of value in differentiating a hæmatoma of the abdominal wall from these conditions, namely tensing the abdominal musculature, is often unsatisfactory because of the pain it causes. Again, the differential diagnosis between the hæmatoma and a strangulated Spigelian hernia (p. 1158) may be difficult. The absence of vomiting suggests a hæmatoma, and the presence of resonance over the swelling favours a Spigelian hernia, while a plain radiograph of the abdomen sometimes gives positive evidence of the latter.

As a Complication of Pregnancy.—Rupture of the inferior epigastric artery occurs occasionally during pregnancy. Surprising to relate, the hæmorrhage into this closed space from this comparatively small artery has proved fatal.

Treatment.—With rest, a comparatively small hæmatoma may resolve, but sometimes renewed hæmorrhage causes the hæmatoma to rupture into the peritoneal cavity. Therefore it is safer to operate early, evacuate the clot, and ligate the artery.

BURST ABDOMEN (*syn.* ABDOMINAL DEHISCENCE)

In 1 to 2 per cent. of cases a laparotomy wound disrupts and viscera are eventrated. The peak incidence of the catastrophe is between the sixth and eighth day after operation. The predisposing causes are infection, persistent cough, abdominal distension, leakage of pancreatic enzymes, hypoproteinæmia, delayed healing in anæmia, jaundice, malignancy, poor suturing technique and the too early removal of deep tension sutures, which should remain in place for fourteen days. Upper abdominal incisions disrupt more frequently than lower abdominal incisions.

In most cases dehiscence of the deeper layers occurs some days before the wound actually bursts asunder; there may be a small serous discharge; the peritoneal stitches snap or become untied within the first three days of operation, though often the peritoneum at the site of the suture line tears. Sometimes the damage is done while the patient is coming round from the anæsthetic; a most potent factor in this respect is the violent coughing reflex set up if an endotracheal tube is withdrawn while the patient is but lightly anæsthetised.

Prophylaxis.—Great care should be taken in suturing the posterior as well as the anterior layer of the rectus sheath. It is all too easy to omit the suture of this layer when the peritoneum is sewn together. It may have retracted laterally and therefore needs to be sought for diligently. The insertion of deep tension sutures of braided nylon is an additional insurance. When infection of the wound has supervened, or in other conditions in which it is thought that the stitches are liable to give way, the abdominal wall should be supported by ' corsets ' of adhesive plaster. In debilitated and

aged subjects wound healing is retarded, so vitamins, particularly vitamin C, and a high protein diet are indicated.

Clinical Features.—A serosanguineous (pink) discharge from the wound is a forerunner of disruption in fully 50 per cent. of cases. It is the most pathognomonic sign of impending wound disruption, and it signifies that intraperitoneal contents are lying extraperitoneally. The patient often volunteers the information that he ' felt something give way '. If skin sutures have been removed, omentum or coils of intestine may be forced through the wound and will be found lying on the skin. Pain and shock are often absent. It is important to note that there may be symptoms and signs of intestinal obstruction.

Treatment.—An emergency operation is required to replace the bowel, relieve any obstruction, and to resuture the wound. While awaiting operation, reassure the patient and cover the wound with a sterile towel. The stomach is emptied by a gastric tube and intravenous fluid therapy commenced.

Operation.—Each protruding coil of intestine is washed gently with saline solution, and returned to the abdominal cavity. Then protruding greater omentum is treated similarly and spread over the intestine. The abdominal wall having been cleansed, all layers are approximated by through-and-through sutures of braided nylon each passed through a soft rubber or plastic tube collar. The abdominal wall may be supported by strips of adhesive plaster encircling the anterior two-thirds of the circumference of the trunk. Antibiotic therapy is started.

Contrary to what might be thought, peritonitis rarely supervenes and, though the skin wound becomes infected, healing is satisfactory. Also a second dehiscence rarely occurs. There is biochemical evidence that healing after disruption produces a stronger wound. This is due to the improvement in collagen metabolism under these circumstances.

INFECTIONS

Cellulitis can occur in any of the planes of the abdominal wall.

Superficial cellulitis is usually discovered when an abdominal wound is inspected following pyrexia. The earliest sign is when the stitches become embedded in the œdematous skin. Later there is a blush extending for a variable distance from the incision or the stitch holes. On palpation with the gloved hand usually one area is found to be more indurated and tender than the remainder. A stitch should be removed from the immediate vicinity, and if pus or sero-pus escapes it is sent for bacteriological examination, meanwhile one of the tetracyclines is administered.

Deep cellulitis is characterised by brawny œdema towards one or both flanks, and not infrequently of the scrotum or vulva as well. Antibiotic therapy is the mainstay of treatment. When tenderness persists, an anatomical incision dividing the muscles carefully, layer by layer, until pus or purulent fluid is encountered, is often advisable.

Progressive post-operative bacterial synergistic gangrene is fortunately a rare complication after laparotomy, usually for a perforated viscus (notably perforated appendicitis). It has also occurred after gall-bladder operations, colectomy for ulcerative colitis, and even after drainage of an empyema thoracis. The condition is

due to the synergic action of microærophilic non-hæmolytic streptococci and, usually, a staphylococcus. The skin in the immediate vicinity of the wound exhibits signs of cellulitis. Within a few days a central purplish zone with an outer brilliant red zone can be distinguished, and the whole region is extremely tender. Soon the purplish area becomes gangrenous and looks like suède leather. Slowly the condition advances (fig. 1254). The gangrenous skin liquefies, exposing underlying granulation tissue. If the condition persists, the general condition deteriorates.

FIG. 1254.—Bacterial synergistic gangrene of the chest and abdominal wall.

Treatment.—Identification of the organisms and a report on their sensitivity to antibiotics is essential. The usual antibiotics may be of little avail. Bacitracin zinc can be effective (10,000 to 20,000 units I.M., every six hours with local application of 500 units per ml, 4 times daily). Without vigorous and effective treatment the gangrene spreads to the flanks and the patient may die of toxæmia. Hyperbaric oxygen (p. 85), if available, can be life-saving. Cellulitis due to bacteriodes may give no bacteriological growth by conventional techniques and may be missed. Metronidazole is required (p. 967).

Subcutaneous gas infection.—This is described on page 34.

NEOPLASMS OF THE ABDOMINAL MUSCULATURE

Desmoid tumour is a tumour arising in the musculo-aponeurotic structures of the abdominal wall, especially below the level of the umbilicus. It is a completely unencapsulated fibroma, and is so hard that it creaks when it is cut. Some cases recur repeatedly in spite of apparently adequate excision.

Ætiology.—Eighty per cent. of cases occur in women, many of whom have borne children, and the neoplasm occurs occasionally in scars of old hernial or other abdominal operation wounds. Consequently, trauma—e.g. the stretching of the muscle fibres during pregnancy or possibly a small hæmatoma of the abdominal wall—appears to be an ætiological factor.

Pathology.—The tumour is composed of fibrous tissue containing multinucleated plasmodial masses resembling foreign-body giant-cells. Usually of very slow growth, it tends to infiltrate muscle in the immediate neighbourhood. Eventually it undergoes a myxomatous change: it then increases in size more rapidly. Metastasis does not occur. Unlike fibromata elsewhere, no sarcomatous change occurs.

Treatment.—Unless the tumour is excised widely, with a surrounding margin of at least 2·5 cm of healthy tissue, recurrence commonly takes place. After removal of a large tumour, repair of the defect in the abdominal wall by tantalum gauze or nylon mesh (p. 1158) is required. These tumours are moderately radio-sensitive.

Fibrosarcoma of the abdominal wall is rare. It is resistant to radiotherapy and only in some cases can a wide excision, with tantalum or nylon net repair, offer hope of a cure.

Adenocarcinoma of the colon (fig. 1247) or of other viscera may invade the abdominal wall. In such cases the resection of this extension, along with the primary growth, may require special repair of the resulting defect.

Secondary implantation in the wound may follow any abdominal operation for carcinoma.

CHAPTER 54

HERNIA

A HERNIA is the protrusion of a viscus or part of a viscus through an abnormal opening in the walls of its containing cavity. The external abdominal hernia is the commonest form and mostly of the inguinal, femoral and umbilical

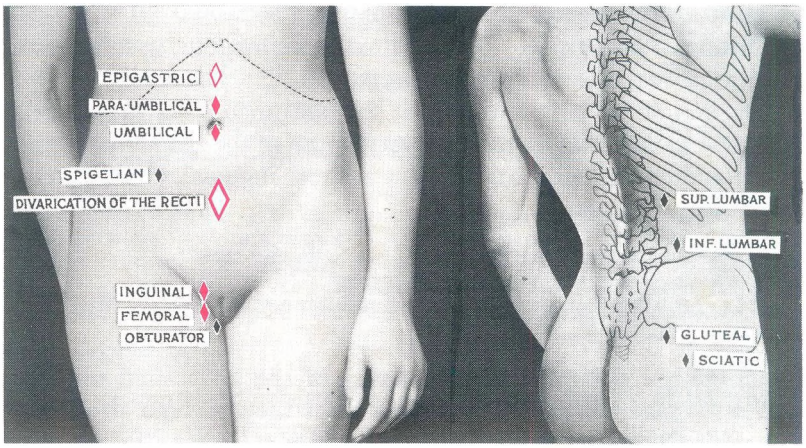


FIG. 1255.—External herniæ. Red = common. White = not unusual. Black = rare.

varieties, respectively 73, 17 and 8·5 per cent (fig. 1255). This leaves 1·5 per cent for the rarer forms. Post-operative incisional hernia has not been included in these figures by Keith.

Ætiology.—An indirect inguinal hernia may occur in a congenital preformed sac —the remains of the processus vaginalis (Hamilton Russell, p. 1138). Sometimes a powerful muscular effort or strain occasioned by lifting a heavy weight, indeed any condition which raises intra-abdominal pressure is liable to be followed by a hernia. Whooping cough is a predisposing cause in childhood, whilst a chronic cough, straining on micturition because of urethral obstruction and straining on defæcation[1] may precipitate a hernia in an adult.

Stretching of the abdominal musculature because of an increase in contents as in obesity and in pregnancy, can be another factor. Fat acts as a kind of ' pile-driver ' for it separates muscle bundles and layers, weakens aponeuroses, and favours the appearance of paraumbilical, direct inguinal, and hiatus herniæ.

Pathological Anatomy.—As a rule, a hernia consists of three parts—the sac, the contents of the sac, and the coverings of the sac.

[1] If a patient over forty presents with recent hernia one should enquire particularly as to bowel habits, for rectal and colonic carcinoma may be brought to light in this way.

Sir Arthur Keith, 1866–1933. Surgeon, Alfred Hospital, Melbourne, Victoria, Australia.
Robert Hamilton Russell, 1860–1933. Surgeon, Alfred Hospital, Melbourne, Victoria, Australia.

The sac consists of a diverticulum of peritoneum which is divided into a mouth, neck, body, and fundus (fig. 1256). Usually the neck is well-defined, but in certain direct inguinal herniæ and in many incisional herniæ there is no actual neck. The body of the sac varies greatly in size and is not necessarily occupied. In cases occurring in infancy and childhood the sac is more delicate than the parietal peritoneum with which it is continuous. In old-standing cases, especially after years of pressure by a truss, the wall of the sac is comparatively thick and even (in places) of cartilaginous consistency.

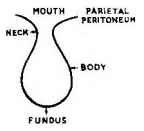


FIG. 1256.—The various parts of a hernial sac.

Contents.—The most common contents of a hernial sac are one or more of the following:

1. Omentum = omentocele (*syn.* epiplocele).
2. Intestine = enterocele. Usually small intestine, but, in some instances, large intestine or the vermiform appendix.
3. A portion of the circumference of the intestine = Richter's hernia.
4. A portion of the bladder, or a diverticulum of the bladder, is sometimes present in addition to other contents in a direct inguinal, a sliding inguinal, and in a femoral hernia.
5. Ovary with or without the corresponding Fallopian tube.
6. A Meckel's diverticulum = Littre's hernia.
7. Fluid. As a part of ascites, or as a residuum thereof. Blood-stained fluid accompanies strangulation.

Coverings are derived from the layers of the abdominal wall through which the sac passes. In long-standing cases they become atrophied from stretching and so amalgamated that they are indistinguishable one from another.

Classification.—A fundamental classification of herniæ, irrespective of their site, is as follows:

1. *Reducible.*
2. *Irreducible.* (*Complication of* 1.)
3. *Obstructed*[1].
4. *Strangulated.* } (*Complications of* 2.)
5. *Inflamed.*

Reducible Hernia

The hernia either reduces itself when the patient lies down, or can be reduced by the patient or by the surgeon. The physical signs of reduction vary somewhat with the nature of the contents of the sac. *Intestine* gurgles on reduction, and the first portion is more difficult to reduce than the last. *Omentum* is doughy, and the last portion is more difficult to reduce than the first. A reducible hernia imparts an expansile impulse on coughing.

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[1] The term 'incarcerated' should be reserved for impaction of fæces within large intestine.

August Gottlieb Richter, 1742–1812. Lecturer on Surgery, Göttingen, Germany.
Gabriele Falloppio, (Fallopius), 1523–1563. Professor of Anatomy, Surgery and Botany, Padua, Italy.
Johann Freidrich Meckel (The Younger), 1781–1833. Professor of Anatomy and Surgery, Halle, Germany.
Alexis Littre, 1658–1726. Surgeon and Anatomist, Paris. Littre described 'Meckel's' diverticulum in a hernial sac in 1700, 81 years before Meckel was born.

Irreducible Hernia

A hernia is said to be irreducible when its contents cannot be returned to the abdomen, and there is no evidence of other complications. Usually such a condition is brought about by adhesions between the sac and its contents or from overcrowding within the sac. Irreducibility without other symptoms is almost diagnostic of an omentocele. Femoral and umbilical herniæ are most often complicated in this manner. Any degree of irreducibility predisposes to strangulation.

Obstructed Hernia

An obstructed hernia is an irreducible hernia containing intestine the lumen of which is obstructed from without or from within; but there is no interference to the blood-supply to the bowel. The symptoms are less severe and the onset more gradual than is the case in strangulation, but more often than not the obstruction culminates in strangulation. Usually no clear distinction can be made between obstruction and strangulation in herniæ, so the safe course is to assume that strangulation is imminent and treat the case accordingly.

Incarcerated Hernia.—The term ' incarceration ' is often used loosely as an alternative to obstruction or strangulation. As emphasised already, the term ' incarceration ' should be employed only when it is considered that the lumen of that portion of the *colon* occupying a hernial sac is blocked with fæces. In that event the scybalous contents of the bowel should be capable of being indented with the finger, like putty.

Strangulated Hernia

A hernia becomes strangulated when the blood-supply of its contents is seriously impaired, rendering gangrene imminent. Gangrene may occur as early as five or six hours after the onset of the first symptoms of strangulation.

Although inguinal hernia is four times more common than femoral hernia, a femoral hernia is more likely to strangulate.

Pathology.—The intestine is obstructed, and in addition its blood supply is constricted. At first only the venous return is impeded. The wall of the intestine becomes congested and bright red, and serous fluid is poured out into the sac. As the congestion increases, the intestine becomes purple in colour. As a result of increased intestinal pressure the strangulated loop becomes distended, often to twice its normal diameter. As venous stasis increases, the arterial supply becomes more and more impaired. Ecchymoses appear under the serosa. Blood is effused into the lumen of the loop, and also through the serosa, causing the fluid in the sac to become blood-stained. The shining serosa becomes dull and covered by a fibrinous, sticky exudate. By this time the walls of the intestine have lost their tone; they feel flabby, and are very friable. The lowered vitality of the intestine favours migration of bacteria through the intestinal wall, and the fluid in the sac teems with bacteria. Gangrene appears at the rings of constriction (fig. 1257), which become deeply furrowed and grey in colour. Also from the anti-mesenteric border gangrene spreads upwards, and the colour changes, which vary from black to grey or green, are due to decomposition of blood in

the subserosa. If the strangulation is unrelieved, perforation of the wall of the intestine occurs, either on the convexity of the loop or at the seat of constriction. The mesentery involved in the strangulation becomes congested and hæmorrhagic, and thrombosis of its vessels occurs. Finally it, too, becomes gangrenous. Peritonitis spreading from the sac to the peritoneal cavity is the usual terminal event.

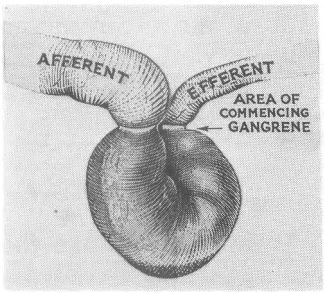

FIG. 1257.—Gangrene commences at the areas of constriction and then at the antimesenteric border.

Clinical Features.—Pain comes on suddenly, and is at first situated over the hernia. Generalised abdominal pain soon supervenes; it is paroxysmal in character and is often located mainly at the umbilicus. Vomiting is forcible and usually oft-repeated. The patient often says that the hernia has recently become larger. On examination, the hernia is *tense*, extremely *tender*, and there is *no expansile impulse on coughing*.

The patient is seriously ill and treatment is vitally urgent. Unless the strangulation is relieved, the paroxysms of pain continue until peristaltic contractions cease with the onset of gangrene when paralytic ileus (often the result of peritonitis) and endotoxin shock (p. 83) develops. Spontaneous cessation of pain is therefore of grave significance.

Strangulated Richter's Hernia (*syn.* Strangulated Partial Enterocele).—A portion of the circumference of the intestine is affected (fig. 1258). The condition, which usually complicates femoral hernia (p. 1146), and, rarely, an obturator hernia, is particularly dangerous as operation is frequently delayed because the clinical features mimic gastro-enteritis. The local signs of strangulation are often not obvious, the patient may not vomit, or vomits only once or twice. Intestinal colic occurs, but the bowels are often opened normally or there may be diarrhœa; absolute constipation (p. 1032) is delayed until paralytic ileus supervenes. For these reasons gangrene of the knuckle of bowel and peritonitis often have occurred before operation is undertaken.

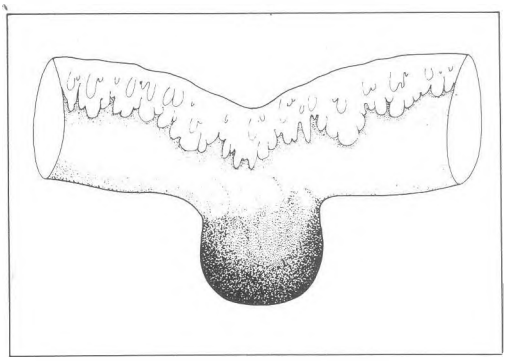

FIG. 1258.—Gangrenous Richter's hernia from a case of strangulated femoral hernia.

Strangulated Omentocele.—The initial symptoms are in general similar to those of strangulated bowel, but the recurring attacks of generalised abdominal pain are not maintained. Vomiting and constipation may be absent. Unlike intestine, omentum can exist on a very meagre blood-supply. The onset of gangrene is therefore correspondingly delayed, and it occurs

first in the centre of the fatty mass. Unrelieved, a bacterial invasion of the dying contents of the sac will almost certainly occur. Infection is limited to the sac for days, and sometimes for weeks. In an inguinal hernia infection usually terminates as a scrotal abscess, but extension of peritonitis from the sac to the general peritoneal cavity is always a possibility.

Inflamed Hernia

Inflammation can occur from external causes, such as pressure from an ill-fitting truss, or from inflammation of contents within the sac, e.g. acute appendicitis or salpingitis. The hernia is tender but *not* tense, and the overlying skin becomes red and œdematous. If it is considered the condition is due to inflammation of an organ within the sac, herniotomy is necessary in order to deal with the cause.

INGUINAL HERNIA

Surgical Anatomy

The superficial inguinal ring is a triangular aperture in the aponeurosis of the external oblique, and lies 1·25 cm above the pubic tubercle. The ring is bounded by a superomedial and an inferolateral crus joined by criss-cross intercrural fibres. Normally the ring will not admit the tip of the little finger (fig. 1259A).

The deep inguinal ring lies 1·25 cm above the inguinal (Poupart's) ligament, midway between the symphysis pubis and the anterior superior iliac spine. It is a U-shaped condensation of the transversalis fascia, incomplete above (fig. 1259C). The competency of the deep inguinal ring depends upon the integrity of this fascia.[1]

The Inguinal Canal.—In infants the superficial and deep inguinal rings are almost superimposed, and the obliquity of this canal is slight. In adults the inguinal canal, which is about 3·75 cm long, is directed downwards and medially from the deep to the superficial inguinal ring. In the male the inguinal canal transmits the spermatic cord, the ilio-inguinal nerve, and the genital branch of the genito-femoral nerve. In the female the round ligament replaces the spermatic cord.

Boundaries of the Inguinal Canal.—The best way to understand these is to study these diagrams (viewing the canal from the superficial to the deep layers as is seen at operation):

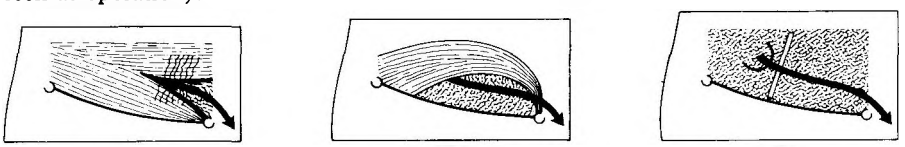

FIG. 1259.—The boundaries of the right inguinal canal. The inguinal ligament passes between the anterior superior iliac spine laterally, and the pubic tubercle medially. A. The superficial layer, the external oblique aponeurosis, showing the crura of the external ring and the intercrural fibres. B. The conjoined muscle (internal oblique and transversus) arches over the cord. Laterally it lies superficial to the cord and the internal ring, then it is above the cord and medially, as the conjoined tendon, behind the cord. C. The deepest layer, the transversalis fascia. The internal inguinal ring, with the inferior epigastric artery lying medially.

Thus, the boundaries of the inguinal canal are as follows:

Anteriorly.—(fig. 1259A and B). External oblique aponeurosis. The conjoined muscle (mainly internal oblique) laterally.

Posteriorly.—Inferior epigastric artery; fascia transversalis; conjoined tendon (internal oblique and transversus) (fig. 1259C and B).

Superiorly.—Conjoined muscles (internal oblique and transversus), (fig. 1259B).

Inferiorly.—Inguinal ligament (fig. 1259A, B and C).

An indirect inguinal hernia travels down the canal on the outer (lateral and anterior) side of the spermatic cord. A direct hernia comes out directly forwards through the

[1] The transversalis fascia is the 'fascial envelope' of the abdomen.

François Poupart, 1661–1708. Surgeon, Hôtel Dieu, Paris.

posterior wall of the inguinal canal. While the neck of the indirect hernia is lateral to the inferior epigastric vessels, the direct hernia usually emerges medial to this except in the saddle-bag or pantaloon type, which has both a lateral and a medial component (p. 1142). An inguinal hernia can be differentiated from a femoral hernia by ascertaining the relation of the neck of the sac to the medial end of the inguinal ligament and the pubic tubercle, i.e. in the case of an inguinal hernia the neck is above and medial, while that of a femoral hernia is below and lateral (fig. 1274).

Indirect (*syn.* Oblique) Inguinal Hernia

An indirect inguinal hernia is the most common of all forms of hernia. Many subscribe to the belief that, at whatever age it appears, it occurs into a preformed sac which is a partially or completely patent processus vaginalis. Normally, shortly before birth, the processus vaginalis becomes obliterated, at first at the deep inguinal ring, and a little later immediately above the upper pole of the epididymis; the tunnel of peritoneum between these two points becomes a fibrous cord.

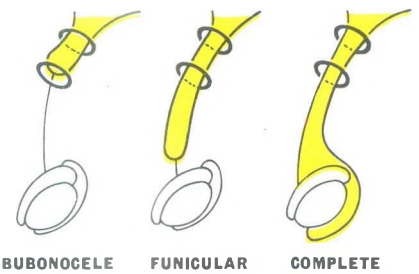

FIG. 1260.—Types of oblique inguinal hernia.

In the first decade of life inguinal hernia is more common on the right side in the male. This is no doubt associated with the later descent of the right testis (p. 1317). After the second decade left inguinal herniæ are as frequent as right. The hernia is bilateral in nearly 30 per cent. of cases.

There are three types of oblique inguinal hernia (fig. 1260):

1. **Bubonocele.**[1]—The hernia is limited to the inguinal canal, the processus vaginalis having been obliterated at the superficial inguinal ring. This type of hernia is seen commonly in young adults; the history is usually a short one.

2. **Funicular.**[2]—The processus vaginalis is closed only at its lower end, just above the epididymis. When the sac is occupied, the contents of the sac can be felt separately from the testis, which lies below the hernia. The funicular variety is common in adults who give a history of inguinal hernia of some standing.

3. **Complete** (*syn.* Scrotal).—There is a persistence of the prenatal condition before the processus vaginalis becomes obliterated; nevertheless a complete inguinal hernia is rarely present at birth. Commonly encountered in infancy, it may not appear until adolescent or adult life. The testis appears to lie within the lower part of the hernia.

Clinical Features.—An oblique inguinal hernia can appear at any age. Males are twenty times more commonly affected than females.

In the early stages when the sac is still limited to the inguinal canal, the diagnosis can present some difficulty. The patient complains of pain in the groin or pain referred to the testicle when he is performing heavy work, or taking strenuous exercise. He is asked to cough and a small transient bulging may be seen and felt together with an expansile impulse. The bulge may be better seen by observing the inguinal region from the side or even looking down the abdominal wall while standing slightly behind the respective shoulder of the patient.

[1] Boubon. Gr. = the groin. Bubo = an enlarged lymph node in the groin or axilla.
[2] Funiculus. L. = a small cord.

As an oblique inguinal hernia increases in size it becomes apparent when the patient coughs, and persists (fig. 1261) until reduced, usually by the act of lying down.[1]

As time goes on the hernia comes down as soon as the patient assumes the upright position. In large herniæ (fig. 1262) there is a sensation of weight, and dragging on the mesentery may produce epigastric pain. If the contents of the sac are reducible, the inguinal canal will be found to be commodious.

FIG. 1261.—Oblique left inguinal hernia which became apparent when the patient coughed, and persisted until it was reduced.

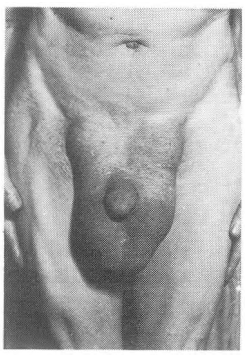

FIG. 1262.—Bilateral oblique inguinal herniæ which have descended into the scrotum.

In infants the swelling appears when the child cries. An inguinal hernia may be translucent in infancy and early childhood, but never in an adult. In girls an ovary may prolapse into the sac.

Differential Diagnosis in the Male.—(*a*) *A vaginal hydrocele.* (*b*) *An encysted hydrocele of the cord.* (*c*) *Spermatocele.* (*d*) *A femoral hernia* (p. 1146). (*e*) *An incompletely descended testis in the inguinal canal.* An inguinal hernia is often associated with this condition. (*f*) *A lipoma of the cord.* This is often a difficult, but unimportant, diagnosis. It is usually not settled until the parts are displayed by operation.

Differential Diagnosis in the Female.—(*a*) *A hydrocele of the canal of Nuck* is the commonest differential diagnostic problem. (*b*) *A femoral hernia.*

Treatment of Indirect Inguinal Hernia

Operative Treatment.—*Operation is undoubtedly the treatment of choice.* The use of a truss and contraindications to it is mentioned on pages 1141 and 1148. It must be remembered that patients who have a bad cough from chronic bronchitis should not necessarily be denied operation, for these are the very people who are in danger of getting a strangulated hernia. Operation can, if necessary, be undertaken in adults, using spinal, epidural or local anæsthesia.

The types of operation are usually classified as (1) *Herniotomy*, (2) *Herniorrhaphy* and *Hernioplasty.*

Inguinal Herniotomy.—This is the essential basic operation and it entails dissecting out and opening the hernial sac, reducing any contents and

[1] *Notes on the clinical examination.*—The clinician is seated in front of the patient who stands with his legs apart. He is instructed to look at the ceiling and to cough at the ceiling. If the hernia will come down it usually does. The examiner looks for the impulse and feels for the impulse and then satisfies himself on the following points: (1) Is the hernia right, or left, or bilateral? (2) Is it an inguinal or a femoral hernia? (see p. 1146). (3) Is it a direct or an indirect inguinal hernia? (4) Is it reducible or irreducible? (patient has to lie down for this to be ascertained). (5) Is the inguinal hernia incomplete (bubonocele) or complete (scrotal)? (6) What are the contents — bowel (enterocele), or omentum (omentocele or epiplocele)?

Anton Nuck, 1650–1692. Professor of Anatomy and Medicine, Leiden, Netherlands.

then transfixing the neck of the sac and removing the remainder. It is employed either by itself or as the first step in herniorrhaphy or hernioplasty. By itself it is sufficient for the treatment of hernia in infants, adolescents and young or fit adults who have good inguinal musculature. In fact, any attempts at repair (herniorrhaphy or hernioplasty) in such cases are meddlesome, and do more harm than good.

In infants it is not necessary to open the canal, as the internal and external rings are superimposed. Excellent results are obtained. The operation should be done in the morning and the child allowed home in the evening. Usually there is no need for the child to stay in hospital. 'The best nurse is the mother.'

Inguinal Herniorrhaphy (and Hernioplasty).—This operation consists of (1) excision of the hernial sac (herniotomy) plus (2) repair of the stretched internal inguinal ring (transversalis fascia) and (3) strengthening or reconstruction of the posterior wall of the inguinal canal. This is achieved by suturing without tension, or by darning with monofilament materials, such as nylon or polyethylene. Fascial flaps, or Dacron mesh implants are employed when the deficiency of the wall is extensive.

OPERATIVE PROCEDURES

(1) Excision of the Hernial Sac (Adult Herniotomy)

An incision[1] is made in the skin and subcutaneous tissues 1·25 cm above and parallel to the medial two-thirds of the inguinal ligament. In large irreducible herniæ the incision is extended into the upper part of the scrotum. After dividing the superficial fascia, the external oblique aponeurosis and the superficial inguinal ring are identified. The external oblique aponeurosis is incised in the line of its fibres, so as to open the inguinal canal. The structures beneath the external oblique aponeurosis are separated from its deep surface before completing the incision into the superficial inguinal ring, which is divided. In this way the ilio-inguinal nerve is safeguarded. By blunt dissection the upper leaf of the external oblique aponeurosis is separated from the internal oblique. The lower leaf is likewise dissected until the inner aspect of the inguinal ligament is seen. The cremasteric muscle fibres are divided longitudinally to open up the subcremasteric space and display the spermatic cord, which is then hooked up on the forefinger.

Excision of the sac. The sac is easily distinguished as a pearly white structure lying on the outer side of the cord and, when the internal spermatic fascia has been incised longitudinally, it can be dissected out and then opened between hæmostats.

Variations in Dissection.—If the sac is small (e.g. bubonocele) it can be freed *in toto*. If it is of the long funicular or scrotal type, or is extremely thickened and adherent (the result of wearing a truss), the fundus need not be sought. The sac is cut across in the inguinal canal. Care must be taken to avoid damage to the vas and the spermatic artery, including the blood supply to the epididymis.

An adherent sac can be separated from the cord by first injecting saline through its posterior wall. A similar tactic is used when dissecting the gossamer sac of infants and children (p. 1142).

Reduction of Contents.—Intestine or omentum is returned to the peritoneal cavity. Omentum is often adherent to neck of fundus of the sac; if to the neck, it is freed, and if to the fundus of a large sac, it may be transfixed, ligated and cut across at a suitable site. The distal part of omentum, like the distal part of a large scrotal sac, can be left *in situ* (the fundus should not, however, be ligated).

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[1] Prior to the skin incision in large inguino-scrotal hernias, the usual antiseptic preparation of the skin should not be extended to the perineal aspect of the scrotum, for, by so doing, severe bacterial contamination of the operation site is likely.

Isolation and Ligation of the Neck of the Sac.—Whatever type of sac is encountered, it is necessary to free its neck by blunt and gauze dissection until the parietal peritoneum can be seen on all sides. Only when the extraperitoneal fat is encountered and the inferior epigastric vessels are seen on the medial side has the dissection reached the required limit. If it has not been done already, the sac is opened. The finger is passed through the mouth of the sac in order to make sure that no bowel or omentum is adherent. The neck of the sac is transfixed and ligated as high as possible, and the sac is excised 1·25 cm below the ligature.

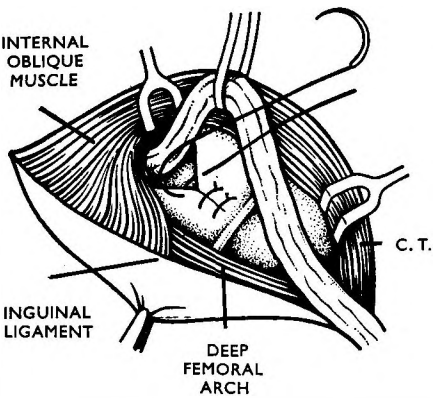


FIG. 1263.—Repair of the internal inguinal ring with lateral displacement of the cord. C.T. = Conjoined Tendon. *(After F. S. A. Doran, F.R.C.S., Bromsgrove.)*

(2) Repair of the Stretched Internal Ring (Lytle) (fig. 1263).

This is an important stage in the operation and must never be omitted. The transversalis fascia is plicated with unabsorbable sutures, the most lateral of these displacing the cord laterally as much as possible and narrowing the diameter of the internal ring to the size of the tip of the little finger.

Lateral displacement of the stump of the sac. Frequently, the surgeon displaces the stump of the sac laterally beneath the arching fibres of the internal oblique. A large curved round-bodied needle, threaded with each of the ends of the ligature used for the transfixion and closure of the neck of the sac, is passed through the lateral fibres of the muscle from within outwards, and tied without tension.

Completion of Operation.—If possible the cremasteric muscle should be reconstituted; the external oblique is directly sutured or overlapped leaving a new external ring which will accommodate the tip of the little finger.

(3) Reconstruction of a weak Posterior Wall of the Inguinal Canal

What to do with a large indirect hernia with a neck of over 10 cm., or a direct inguinal hernia, is often a problem. All are agreed that the first step is the careful repair of the transversalis fascia with lateral displacement of the cord (above), and that the second step, reinforcement of the posterior wall, must be accomplished without tension.

The First Step.—Repair the stretched transversalis fascia as well as circumstances permit, not forgetting to take the repair as far laterally as possible, and narrowing the remnants of the internal ring. Even in a direct hernia the internal ring needs attention.

The Second Step.—Three main types of herniorrhaphy are possible:

(*a*) *Darning.*—Using two layers of nylon or similar material (Moloney, Patey, fig. 1265).

(*b*) *Bassini's operation and rectus relaxing incision* (fig. 1266).

(*c*) *Fascial flap.*—A flap of the anterior rectus sheath, hinged on the linea semilunaris is lifted, turned down, and sewn to the inguinal ligament (Bloodgood).

(*d*) *Dacron mesh* (fig. 1267).

Should further reinforcement be required, the external oblique aponeurosis may be overlapped behind rather than in front of the spermatic cord, thus directing this structure into the subcutaneous fat at the site of the repaired internal ring (Halsted and Keynes modifications). Great care must be exercised to tailor the opening in the aponeurosis through which the cord emerges, neither too small nor too large.

A Truss: A truss is used when operation is contraindicated because of cardiac, pulmonary, or other systemic disease, or when operation is refused. The hernia must be reducible. A rat-tailed spring truss (fig. 1264) with a perineal band to

William James Lytle, Contemporary. Consulting Surgeon, Royal Infirmary, Sheffield, England.
George Edward Moloney, Contemporary. Professor of Surgery, University of Riyadh, Saudi Arabia.
David Howard Patey, 1899-1977. Surgeon, The Middlesex Hospital, London.
Edoardo Bassini, 1844–1924. Professor of Surgery of Padua, Italy.
Joseph Colt Bloodgood, 1867–1935. Surgeon, Johns Hopkins Hospital, Baltimore, Maryland, U.S.A.
William Stewart Halsted, 1852–1922. Surgeon, Johns Hopkins Hospital, Baltimore, Maryland, U.S.A.
Sir Geoffrey Langdon Keynes, Contemporary. Consulting Surgeon, St. Bartholomew's Hospital, London.

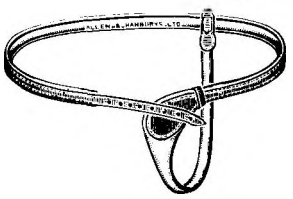


FIG. 1264.—Rat-tailed truss.

prevent the truss slipping will, with due care and attention, control a small or moderate-sized inguinal hernia. A truss must be worn continuously during waking hours, kept clean, and in proper repair, and renewed when it shows signs of wear. It must be applied before the patient gets up and while the hernia is reduced. A properly-fitting truss controls the hernia when the patient stands with his legs wide apart, stoops, and coughs violently. However, as Farquharson stressed, often the pad of a truss is seen to lie to the side of the swelling, where the pressure it exerts is both useless and harmful. In these circumstances the truss is a menace, for it increases the risk of strangulation.

For a large irreducible hernia a bag truss is sometimes employed, but as a rule operation should be urged in cases of irreducibility, because of the danger of strangulation.

Infants and Trusses.—Special inflatable rubber trusses, formerly popular for the control of an infant's hernia are rarely used. Provided urgent admission is not required for sudden irreducibility, the parents are advised to wait until operation is performed when the infant is at least three months old.

Direct Inguinal Hernia

Between 10 and 15 per cent. of inguinal herniæ are direct. Over half of the herniæ are bilateral.

A direct inguinal hernia is always acquired. The sac passes through a weakness or defect of the transversalis fascia in the posterior wall of the inguinal canal. In some cases the defect is small and closely related to the insertion of the conjoint tendon, while in others there is a generalised bulge. Often the patient has poor lower abdominal musculature, as shown by the presence of elongated bulgings (Malgaigne's bulges). Women practically never develop a direct inguinal hernia (Brown). Predisposing factors are a chronic cough, straining, and heavy work.

Direct herniæ rarely attain a large size or descend into the scrotum. In contradistinction to an oblique inguinal hernia, a direct inguinal hernia lies behind the spermatic cord. The sac is often smaller than the hernial mass would indicate, the protruding mass mainly consisting of extraperitoneal fat. As the neck of the sac is wide, direct inguinal herniæ rarely strangulate.

Injury to the ilio-hypogastric or ilio-inguinal nerves during the gridiron operation for appendicitis appears to be associated with the development of a right inguinal hernia, usually, but not necessarily, of the direct variety.

Funicular direct inguinal hernia (*syn.* prevesical hernia) is a narrow-necked hernia with prevesical fat and a portion of the bladder that occurs through a small oval defect in the medial part of the conjoined tendon just above the pubic tubercle. It occurs principally in elderly males, and occasionally it becomes strangulated. Unless there are definite contraindications, operation should always be advised.

Dual (*syn.* **Saddle-bag; Pantaloon**) **Hernia.**—Here there are two sacs which straddle the inferior epigastric artery, one sac being medial and the other lateral to this vessel. The condition is not a rarity, and is a cause of recurrence, one of the sacs having been overlooked at the time of operation.

Operation for Direct Hernia.—The principles of repair of direct hernia are the same as those of an indirect hernia with the exception that the hernial sac need not be removed. The sac, after it has been dissected free from surrounding structures, is inverted into the abdomen and the transversalis fascia repaired in front of it. Some form of reconstruction of the posterior wall of the inguinal canal is necessary. Nylon darns, and Dacron mesh implantations are popular (figs. 1265 and 1267).

Eric Leslie Farquharson, 1905–1970. Surgeon, Royal Infirmary, Edinburgh.
Joseph François Malgaigne, 1806–1865. Professor of Surgery, Paris.
Francis Robert Brown, 1889–1967. Surgeon, Royal Infirmary, Dundee, Scotland.

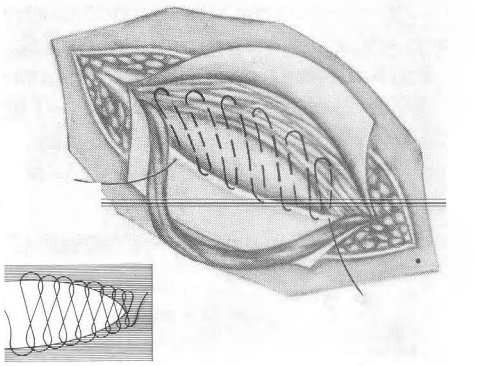


FIG. 1265.—The first layer of the two-layer darn of the posterior wall of the inguinal canal. The darning is conducted from the neighbourhood of the pubic tubercle up to and above the deep inguinal ring and back to the starting point. The darning is kept fairly loose, and it forms a lattice upon which fibrous tissue is laid down. The external oblique aponeurosis is reunited either in front or behind the cord. Meticulous asepsis is essential. Inset: An alternative lattice darn.

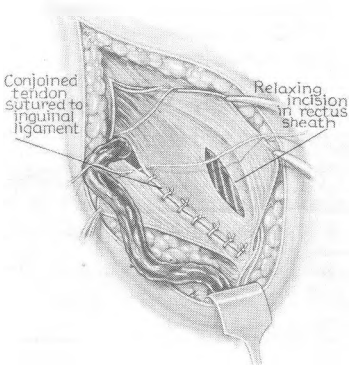


FIG. 1266.—Bassini's operation with relaxing incision (Tanner's slide). Suitable when the conjoint muscle is well developed and the space between it and the inguinal ligament narrow enough to permit approximation without tension. The relaxing incision in the rectus sheath aids this essential requirement. (*After L. W. Ester, Jnr.*)

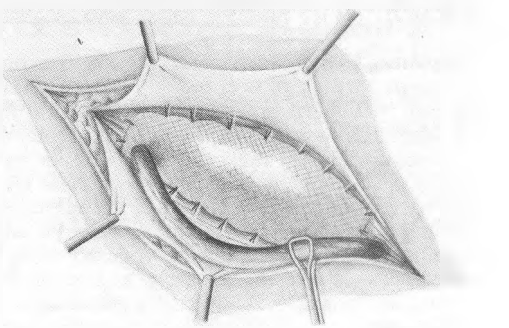


FIG. 1267.—Dacron mesh reinforcement. The mesh is attached to the inguinal ligament, the coverings of the pubis, the rectus sheath and the conjoined muscle. It is also fashioned by a slot laterally above and below the cord at the internal ring.

Strangulated Inguinal Hernia

Strangulation of an inguinal hernia occurs at any time during life, and in both sexes. Oblique inguinal hernias strangulate commonly; the direct variety but rarely owing to the wide neck of the sac. Sometimes a hernia strangulates on the first occasion that it descends; more often strangulation occurs in patients who have worn a truss for a long time, and in those with a partially reducible or irreducible hernia.

In order of frequency, the constricting agent is: (*a*) the neck of the sac; (*b*) the external abdominal ring in children; (*c*) rarely adhesions within the sac.

Contents.—Usually small intestine is involved in the strangulation; the

Norman Cecil Tanner, Contemporary. Consulting Surgeon, Charing Cross Hospital, London.

next most frequent is omentum; sometimes both are implicated. For large intestine to become strangulated in an inguinal hernia is of the utmost rarity, even when the hernia is of the sliding variety (p. 1145).

Pathological and Clinical Features are described on pp. 1135–1137.

Strangulation during Infancy.—The incidence of strangulation is 4% (Gross), and the ratio of females to males is 5:1. But this is much higher than current experience suggests. More frequently the hernia is irreducible but not strangulated. In most cases of strangulated inguinal hernia occurring in female infants the content of the sac is an ovary, or an ovary plus its Fallopian tube.

FIG. 1268.

Maydl's hernia (*syn.* Hernia-in-W) (fig. 1268) is rare. The strangulated loop of the W lies within the abdomen, thus local tenderness over the hernia is not marked. At operation two comparatively normal-looking loops of intestine are present in the sac. After the obstruction has been relieved, the strangulated loop will become apparent if traction is exerted on the middle of the loops occupying the sac.

Treatment of Strangulated Inguinal Hernia.—The treatment of strangulated hernia is by *emergency operation.*[1]

If dehydration and collapse are present, intravenous fluid replacement (p. 94) and gastric aspiration for one to three hours are invaluable. It is *absolutely essential* to make sure that the stomach is emptied just before commencing the anæsthetic. The passing of a large-bore stomach tube is the best way of preventing vomiting, drowning, and cardiac arrest during the induction. The bladder must also be emptied, if necessary by a catheter.

Inguinal Herniotomy for Strangulation.—An incision is made over the most prominent part of the swelling. The external oblique aponeurosis is exposed, and the sac, with its coverings, is seen issuing from the superficial inguinal ring. In all but very large herniæ it is possible to deliver the body and fundus of the sac together with its coverings and (in the male) the testis on to the surface. Each layer covering the anterior surface of the body of the sac near the fundus is incised, and if possible it is stripped off the sac. The sac is then incised, the fluid therein is mopped up or aspirated very thoroughly, for it may be highly infected. The external oblique aponeurosis and the superficial inguinal ring are divided. Returning to the sac, a finger is passed into the opening, and employing the finger as a guide, the sac is slit up along its length. If the constriction lies at the superficial inguinal ring or in the inguinal canal, it is readily divided by this procedure. When the constricting agent is at the deep inguinal ring, by applying hæmostats to the cut edge of the neck of the sac and drawing them downwards, and at the same time retracting the internal oblique upwards, it may be possible to continue slitting up the sac over the finger beyond the point of constriction. When the constriction is too tight to admit a finger, a grooved director is inserted and the neck of the sac is divided with a hernia knife in an upward and inward direction, i.e. parallel to the inferior epigastric artery, under vision. Once the constricting agent has been divided, the strangulated contents can be drawn down. Devitalised omentum is excised after being securely ligated, by interlocking stitches if bulky. Viable intestine is returned to the peritoneal cavity. Doubtfully viable and gangrenous intestine is dealt with as described on p. 1036. If the hernial sac is of moderate size and can be separated easily from its coverings, it is excised and closed by a purse-string suture. When the sac is large and adherent, much time is saved by cutting across the sac as described (p. 1140). Having tied off the neck of the sac, if the condition of the patient permits, a simple form of herniorrhaphy can be carried out.

[1] ' The danger is in the delay, not in the operation '—Astley Cooper.

Robert Edward Gross, Contemporary. Ladd Professor of Children's Surgery, Harvard University Medical School, Boston, Mass., U.S.A.
Karel Maydl, 1853–1903. Professor of Surgery, Prague, Czechoslovakia.
Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London.

Conservative Measures.—These are only indicated in infants. The child is given a sedative and then slung to a Balkan beam[1] or to the bedrail by his feet (the judgment of Solomon[2] position) for no longer than three hours. In 75 per cent. of cases reduction is effected, and there appears to be no danger of gangrenous intestine being reduced (Irvine Smith).

N.B.—Vigorous manipulation (taxis) has no place in modern surgery, and is mentioned only to be condemned. Its dangers include:

1. Contusion or rupture of the intestinal wall.
2. Reduction-en-masse (fig. 1269). 'The sac together with its contents, is pushed forcibly back into the abdomen; and as the bowel will still be strangulated by the neck of the sac, the symptoms are in no way relieved' (Treves). 3. Reduction into a loculus of the sac. 4. The sac may rupture at its neck and its contents are reduced, not into the peritoneal cavity, but extraperitoneally.

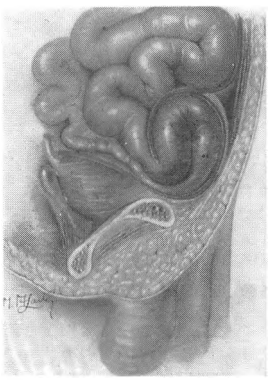


FIG. 1269.—Reduction-en-masse.

Recurrent Inguinal Hernia

Although some individual series show a recurrence-rate of under 1 per cent, the general rate of recurrence is nearer 10 per cent. The recurrence takes place within a year in most cases.

The following summarises the principal causes of recurrence:

1. **Pre-operative.**—Faulty selection of cases (e.g. those with Malgaigne's bulging (p. 1142)) or chronic bronchitis.
2. **Operative.**—Faulty technique.

(*a*) Failure to ligate the sac at the neck. (*b*) Tension and tying stitches too tightly so that intervening tissues are devitalised. (*c*) Imperfect hæmostasis, predisposing to infection. (*d*) The use of absorbable sutures in the repair of the inguinal canal.

3. **Post-operative.**—The common causes of a recurrence are (*a*) infection of the wound; (*b*) persistent cough; (*c*) heavy lifting before three months after herniorrhaphy: (*d*) urethral obstruction; (*e*) straining at defæcation.

Treatment.—Unless there is some reason why further operation is unlikely to be successful, e.g. persistent cough, re-operation performed carefully with special precautions against recurrence, e.g. nylon darn or Dacron mesh.

The Spermatic Cord as a Barrier to Effective Closure of the Inguinal Canal.—In the elderly patient, removal of the testis and cord aids in an effective repair in the case of recurrent inguinal hernia, sliding hernia, and some large direct herniæ. The signed permission of the patient must always be obtained beforehand.

Sliding Hernia (*syn.* **Hernie-en-Glissade**) (fig. 1270)

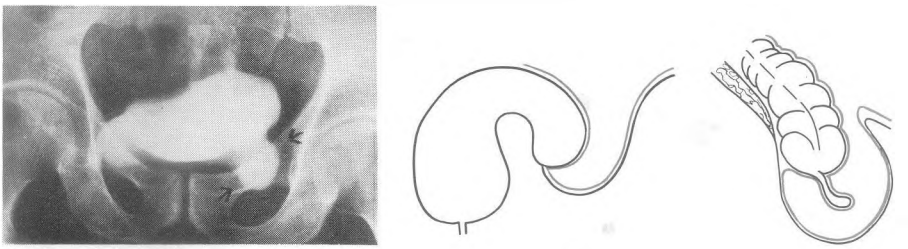
As a result of slipping of the posterior parietal peritoneum on the underlying cellular tissue, the posterior wall of the sac is not formed of peritoneum alone, but by the sigmoid colon and its mesentery on the left, the cæcum on the right and, sometimes, on either side by a portion of the bladder. It should be clearly understood that the cæcum, appendix, or a portion of the colon *wholly within* a hernial sac does not constitute a sliding hernia. A small bowel sliding hernia occurs once in 2,000 cases; a sacless sliding hernia once in 8,000 cases.

Clinical Features.—A sliding hernia occurs almost exclusively in males. Five out of six sliding herniæ are situated on the left side; bilateral sliding

[1] An overhead beam or bar for applying traction to a fractured femur, first used in the Balkan theatre of war, 1914–1918.

[2] Solomon, King of Israel. The story of the judgment of Solomon is told in the Bible 1 Kings III, 16–28.

Irvine Battinson Smith, Contemporary. Surgeon, Burton-on-Trent General Hospital, Staffs., England.
Sir Frederick Treves, 1853–1923. Surgeon, The London Hospital.



(*a*) (*b*) (*c*)

FIG. 1270.—Sliding hernia.—(*a*) Cystogram showing bladder involving a left inguinal hernia. (*b*) Diagram of the same. (*c*) Cæcum and appendix in right sliding hernia.

herniæ are exceedingly rare. The patient is nearly always over forty, the incidence rising with the weight of years. There are no clinical findings that are pathognomonic of a sliding hernia, but it should be suspected in every large globular inguinal hernia descending well into the scrotum.

Occasionally large intestine is strangulated in a sliding hernia; more often non-strangulated large intestine is present behind the sac containing strangulated small intestine.

Treatment.—A sliding hernia is impossible to control with a truss, and as a rule the hernia is a cause of considerable discomfort. Consequently operation is indicated, and the results generally are good.

Operation.—It is unnecessary to remove any of the sliding hernial sac provided it is freed completely from the cord and the abdominal wall, and that it is replaced deep to the repaired fascia transversalis. In most instances it is desirable to perform orchiectomy (p. 1145) in order to effect a secure repair. No attempt should be made to dissect the cæcum or colon free from the peritoneum under the impression that these are adhesions in which case peritonitis or a fæcal fistula resulting from necrosis of a devascularised portion of the bowel may occur. This is specially liable to occur on the left side, as vessels in the meso-colon may be injured.

FEMORAL HERNIA

Femoral hernia is the third most common type of hernia (incisional hernia comes second). It accounts for about 20 per cent. of herniæ in women, and 5 per cent. in men. The overriding importance of femoral hernia lies in the facts that it cannot be controlled by a truss, and that of all herniæ it is the most liable to become strangulated.

Surgical Anatomy.—The femoral canal occupies the most medial compartment of the femoral sheath, and is shaped like a truncated cone. ——→ It extends from the femoral ring above to the saphenous opening below. It is 1·25 cm long, and 1·25 cm wide at its base, which is directed upwards. The femoral canal contains fat, lymphatic vessels, and the lymph node of Cloquet. It is closed above by the septum crurale, a condensation of extraperitoneal tissue pierced by lymphatic vessels, and below by the cribriform fascia.

The femoral ring (fig. 1271) is bounded:

Anteriorly by the inguinal ligament.

Posteriorly by Astley Cooper's (ilio-pectineal) ligament, the pubic bone, and the fascia over the pectineus muscle.

Medially by the concave knife-like edge of Gimbernat's (lacunar) ligament (fig. 1271), which is also prolonged along the ilio-pectineal line as Astley Cooper's ligament.

Laterally by a thin septum separating it from the femoral vein.

Jules Germain Cloquet, 1790–1883. Professor of Anatomy and Surgery, Paris.
Don Antonio de Gimbernat, 1734–1816. Professor of Anatomy, Barcelona, Spain, and later Director of the Royal College of Surgeons in Spain.

Sex Incidence.—The female to male ratio is about 2:1, but it is interesting that whereas the female patients are frequently elderly, the male patients are usually between thirty to forty-five years. The condition is more prevalent in women who have borne children than in nulliparæ.

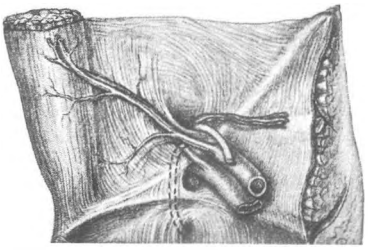


FIG. 1271.—The right femoral ring from within. The dotted structure is an abnormal obturator artery.

Pathology.—A hernia passing down the femoral canal descends vertically as far as the saphenous opening. While it is confined to the inelastic walls of the femoral canal the hernia is necessarily narrow, but once it escapes through the saphenous opening into the loose areolar tissue of the groin, it expands, sometimes considerably. A fully distended femoral hernia assumes the shape of a retort (fig. 1272), and its bulbous extremity may be above the inguinal ligament. By the time the contents have pursued so tortuous a path they are usually irreducible and apt to strangulate.

FIG. 1272.

Clinical Features.—Femoral hernia is very rare before the fifteenth year. Between twenty and forty years of age the prevalence rises, and continues to old age. The right side (fig. 1273) is affected twice as often as the left, and in 20 per cent. of cases the condition is bilateral. The symptoms to which a femoral hernia gives rise are less pronounced than those of an inguinal hernia; indeed, a small femoral hernia may be

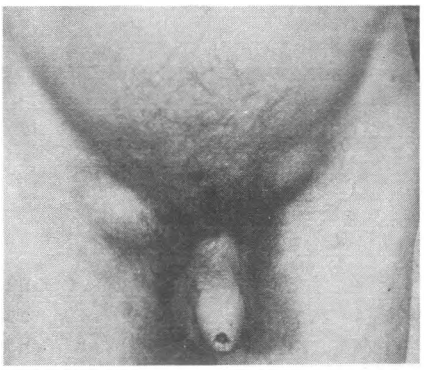


FIG. 1273.—The patient has a left inguinal and a right femoral hernia.

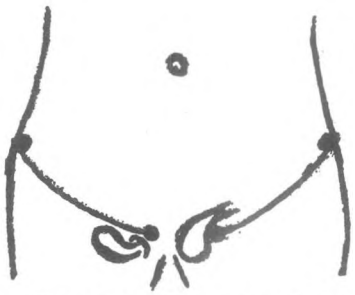


FIG. 1274.—The essentials of differential diagnosis between a femoral and an inguinal hernia (see p. 1137).

unnoticed by the patient or disregarded for years, until perhaps one day it strangulates. Adherence of greater omentum sometimes causes a dragging pain. Occasionally a large sac is present.

Differential Diagnosis

A Femoral Hernia has to be distinguished from:

(1) *An Inguinal Hernia.*—An inguinal hernia lies above and medial to the medial end of the inguinal ligament at its attachment to the pubic tubercle. The femoral hernia lies below this (fig. 1274). Occasionally the fundus of a femoral hernia sac overlies the inguinal ligament.

(2) *A Saphena Varix* (fig. 1275).—A saphena varix is a saccular enlargement of the termination of the long saphenous vein and it is usually accompanied by other signs of varicose veins. The swelling disappears completely when the patient lies down, while a femoral hernia sac usually is still palpable. In both there is an impulse

FIG. 1275.—Sgphena varix.

on coughing. A saphena varix will, however, impart a fluid thrill to the examining fingers when the patient coughs, or when the saphenous vein below the varix is tapped with the fingers of the other hand. Sometimes a venous hum can be heard when a stethoscope is applied over a saphena varix.

(3) *An Enlarged Femoral Lymph Node.*—If there are other enlarged lymph nodes in the region the diagnosis is tolerably simple, but when Cloquet's lymph node alone is affected the diagnosis may be impossible unless there is a clue, such as an infected wound or abrasion on the corresponding limb or on the perineum.

(4) *Lipoma.*

(5) *A Femoral Aneurysm.*—The intrinsic signs of an aneurysm are described on p. 148.

(6) *A Psoas Abscess.*—There is often a fluctuating swelling—an iliac abscess—which communicates with the swelling in question (fig. 1276). Examination of the spine and an X-ray will settle the diagnosis.

(7) *A distended Psoas Bursa.*—The swelling diminishes when the hip is flexed, and osteoarthritis of the hip is present.

(8) *Rupture of the adductor longus* with hæmatoma.—If there is no superficial discoloration, the diagnosis can be very difficult.

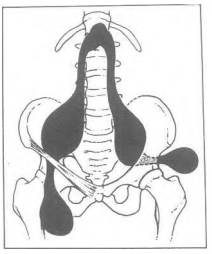


FIG. 1276.—A psoas abscess appearing beneath the inguinal ligament may simulate a reducible femoral hernia. Likewise, when it points in the buttock, a gluteal hernia has been suspected. (*After J. F. Calot.*)

Hydrocele of a Femoral Hernial Sac.—The neck of the sac becomes plugged with omentum or by adhesions, and a hydrocele of the hernial sac results.

Laugier's femoral hernia is a hernia through a gap in the lacunar (Gimbernat's) ligament. The diagnosis is based on the unusual medial position of a small femoral hernial sac. Nearly always the hernia has strangulated.

Narath's femoral hernia occurs only in patients with congenital dislocation of the hip and is due to lateral displacement of the psoas muscle. The hernia lies hidden behind the femoral vessels.

Cloquet's hernia is one in which the sac lies under the fascia covering the pectineus muscle. Strangulation is likely. The sac may coexist with the usual type of femoral sac (see interparietal inguinal hernia, p. 1157).

Strangulated Femoral Hernia.—It cannot be emphasised too strongly that not only does a femoral hernia become strangulated frequently, but often gangrene develops rapidly. This is accounted for by the narrow, unyielding femoral ring. In 40 per cent. of cases the obstructing agent (fig. 1277) is not Gimbernat's (lacunar) ligament but the narrow neck of the femoral sac itself (Henry Souttar). The frequent occurrence of a *Richter's hernia* (p. 1136) also must be stressed.

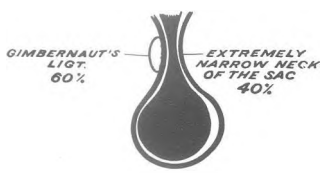


FIG. 1277.

Gaur has reported that, in the elderly with thin elastic skin, the superficial inferior epigastric or the circumflex iliac vein may be seen to be distended if strangulation is present.

Treatment of Femoral Hernia

The constant risk of strangulation is sufficient reason for urging the patient to have herniorrhaphy performed. A *femoral truss is highly unsatisfactory* in that it becomes displaced as the thigh is flexed, and strangulation is thereby encouraged.

Stanislas Laugier, 1799–1872. Surgeon, Hôtel Dieu, Paris.
Albert Narath, 1864–1924. Professor of Surgery, Heidelberg, Germany.
Sir Henry Sessions Souttar, 1875–1964. Surgeon, The London Hospital.
Durga Dutt Gaur, Contemporary. Surgeon, Bombay, India.

Operative Treatment.—The low operation (Lockwood), the high operation (McEvedy), and the inguinal operation (Lotheissen) all have their advocates. In all cases the bladder *must* be emptied immediately before commencing the operation.

The Low Operation (Lockwood).—The sac is dissected out below the inguinal ligament via a groin-crease incision. It is essential to peel off all the anatomical layers which cover the sac. These are often thick and fatty. After dealing with the contents (e.g. freeing adherent omentum) the neck of the sac is pulled down, ligated as high as possible and allowed to retract through the femoral canal. The canal is closed by suturing the inguinal ligament to the ilio-pectineal line using three unabsorbable sutures (e.g. monofilament nylon).

The High (McEvedy) Operation.—A vertical incision is made over the femoral canal and continued upwards above the inguinal ligament. Through the lower part of the incision the sac is dissected out. The upper part of the incision exposes the inguinal ligament and the rectus sheath. The superficial inguinal ring is identified, and an incision 2·5 cm above the ring and parallel to the outer border of the rectus muscle is deepened until the extraperitoneal space is found. By gauze dissection in this space the hernial sac entering the femoral canal can be easily identified. Should the sac be empty and small, it can be drawn upwards; if it is large, the fundus is opened below, and its contents, if any, dealt with appropriately before delivering the sac upwards from its canal. The sac is then freed from the extraperitoneal tissue and its neck is ligated. An excellent view of the iliopectineal ligament is obtained and the conjoined tendon is sutured to it with non-absorbable sutures (fig. 1278).

An advantage of the operation is that if resection of intestine is required, ample room can be obtained by opening the peritoneum. The disadvantage of the operation is that if infection occurs incisional hernia is not very unusual.

Lotheissen's Operation.—The inguinal canal is opened as for *inguinal* herniorrhaphy. The transversalis fascia is incised to the medial side of the epigastric vessels and the opening is enlarged. The peritoneum is now in view; one must be certain that it *is* the peritoneum and not the bladder or a diverticulum thereof. The peritoneum is picked up with dissecting forceps, and incised. It is now possible to ascertain if any intraperitoneal structure is entering the femoral sac. Should the sac be empty, hæmostats are placed upon the edges of the opening into the peritoneum, and by gauze dissection the sac is withdrawn from the femoral canal. An empty sac can be delivered easily; in the event of the sac being occupied, the technique described below for strangulation should be followed.

The Lotheissen Repair.—The conjoined tendon is sutured to the iliopectineal line to form a shutter. While protecting the external iliac/femoral vein with the forefinger, unabsorbable sutures are passed through the periosteum and Cooper's ligament overlying the iliopectineal line (fig. 1279). The retractor having been removed, the long ends of the sutures are passed from within, outwards, through the conjoined tendon, and tied, thus approximating the conjoined tendon to the iliopectineal line. If there is any tension, a Tanner's slide (p. 1143) will facilitate this step. The incised external oblique is sutured.

Strangulated Femoral Hernia.—It is necessary to modify some of the steps of the Lotheissen operation. As soon as the external oblique has been exposed, the inferior margin of the wound is retracted strongly, thereby displaying the swelling. The coverings of the sac are incised and peeled off, until the sac, dark from contained bloodstained fluid, is apparent. The sac is incised, and the fluid that escapes is mopped up with great care. The retractor is removed and the operation is continued above the inguinal ligament in the same way as described already. Once the peritoneum has been opened above the inguinal ligament, one can see exactly what is entering the sac. Should the obstruction lie in a narrow neck of the sac, the beak of a hæmostat is insinuated, and with great care the neck is stretched[1]. The contents of the sac are delivered, and dealt with, p. 1140. Sometimes, in order to facilitate

[1] An abnormal obturator artery is present either on the medial or the lateral side of the neck of the sac in 28 per cent. of cases.

Charles Barret Lockwood, 1856–1914. Surgeon, St. Bartholomew's Hospital, London.
Peter George McEvedy, 1890–1951. Surgeon, Ancoats Hospital, Manchester, England.
Georg Lotheissen, 1868–1941. Surgeon, Kaiser Franz Josef Hospital, Vienna.

reduction of the hernial contents, it becomes necessary to divide or digitally to avulse part of Gimbernat's ligament.

N.B.—It must be repeated that throughout operations for the repair of a femoral hernia, *on the lateral side*, the external iliac/femoral vein must be protected, and *on*

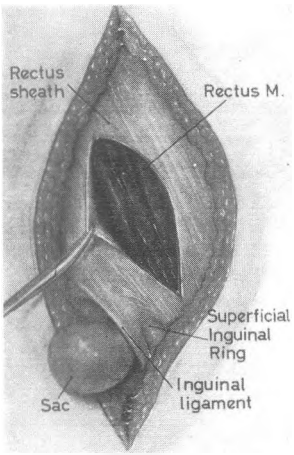


FIG. 1278.—McEvedy's operation. Right side.

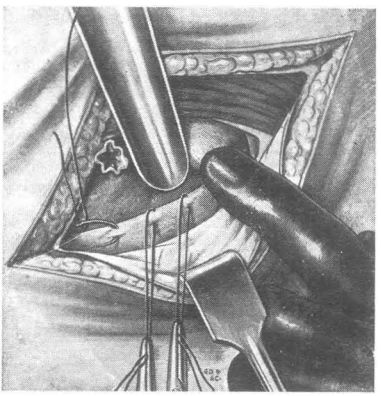


FIG. 1279.—Method of placing the deep sutures (see text). The finger is protecting the left external iliac/femoral vein.

the medial side great care must be taken not to injure the bladder, particularly since a portion of the bladder may form part of the wall of the sac (a sliding femoral hernia).

UMBILICAL HERNIA

Exomphalos (*syn.* omphalocele) occurs once in every 6,000 births; it is due to failure of all or part of the mid-gut to return to the cœlom during early fœtal life. Sometimes a large sac ruptures during birth (fig. 1280). When the sac remains unruptured, it is semi-translucent, and although very thin it consists of three layers—an outer layer of amniotic membrane, a middle layer of Wharton's jelly, and an inner layer of peritoneum. There are two varieties of exomphalos:

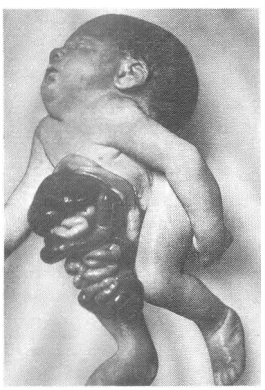


FIG. 1280.—Exomphalos. The delicate sac burst during delivery.

Exomphalos Minor.—The sac is relatively small and to its summit is attached the umbilical cord (fig. 1281). Inadvertently a loop of small intestine or a Meckel's diverticulum has been included in the ligature applied to the base of an umbilical cord containing this protrusion.

Exomphalos Major.—The umbilical cord is attached to the inferior aspect of the swelling (fig. 1282), which contains small and large intestine, and nearly always a portion of the liver. Half the cases belong to this group.

Treatment: (*a*) *Exomphalos Minor.*—It is necessary only to twist the cord,

Thomas Wharton, 1614–1673. Physician, St. Thomas's Hospital, London.

so as to reduce the contents of the sac through the narrow umbilical opening into the peritoneal cavity, and to retain them there by strapping applied firmly. In spite of a sero-purulent discharge from beneath it, on no account must the strapping be removed for fourteen days (Denis Browne).

(*b*) *Exomphalos Major.*—Operation within the first few hours of life is the only hope, otherwise the sac will burst. In order to prevent further distension of the contents of the sac, the infant should not be fed. A few newborn infants with a ruptured sac have survived following immediate operation and antibiotic therapy. A drip blood-transfusion is desirable.

FIG. 1281.—Exomphalos minor.

FIG. 1282.—Exomphalos major.

Operation.—It must be realised that most of the contents of the sac have never been housed within the abdominal cavity; consequently that cavity is unduly small, and to attempt to replace the contents of the sac is like endeavouring to put 2 kg. of sugar into a 1 kg. bag—a feat that so often results in respiratory embarrassment, compromise of venous return, and possibly intestinal obstruction. It is necessary to create flaps of skin by undermining the subcutaneous tissue on either side, so that the flaps can be brought together over the sac. If necessary relaxing incisions must be made in the loins to permit closure. For several days following the operation it is advisable to carry out aspiration through an indwelling gastric tube, in order to relieve or prevent distension. If the patient survives the construction of this protective cutaneous coverage, repair of the hernia can be delayed for months, or even years. At the second operation, it is surprising to find that the peritoneum and the muscles can be drawn together and closed in layers.

Congenital Umbilical Hernia.—On rare occasions a well-developed umbilical hernia is present at birth; the condition is believed to be due to intra-uterine epithelialisation of a small exomphalos.

Umbilical Hernia of Infants and Children.—This is a hernia through a weak umbilical scar usually the result of neonatal sepsis (p. 1126). The ratio of males to females is 2:1. The hernia is often symptomless, but increase in the size of the hernia by crying causes pain, which makes the infant cry the more. Small herniæ are spherical; those that increase in size tend to assume a conical shape (fig. 1283) and are present apart from crying. Obstruction or strangulation below the age of three years is extremely uncommon.

FIG. 1283.—Infantile umbilical hernia.

Treatment.—Conservative treatment by *masterly inactivity* is successful in about 93 per cent. of cases. When the hernia is symptomless, reassurance of the parents is all that is necessary, for in a very high percentage of cases the hernia will be found to disappear spontaneously during the first few months of life. Cure may also be hastened by pulling the skin and abdominal musculature together by adhesive strapping placed across the abdomen.

Sir Denis John Wolko Browne, 1892–1967. Surgeon, Hospital for Sick Children, Great Ormond Street, London.

Herniorrhaphy.—In cases where masterly inactivity fails, operation is required, and it should be carried out, preferably, about the age of two years.

Operation.—In infants a small curved incision is made immediately below the umbilicus. The skin cicatrix is dissected upwards, and the neck of the sac is isolated. After ensuring that the sac is empty of contents, it is either inverted into the abdomen or it is ligated by transfixion and excised. The defect in the linea alba is closed with two unabsorbable sutures.

Para-umbilical Hernia of Adults (syn. supra-, infra-umbilical hernia).—It should be noted that in adults the hernia does not occur through the umbilical scar. It is a protrusion through the linea alba just above the umbilicus or, occasionally, just below that structure (fig. 1284). As it enlarges, it becomes rounded or oval in shape (fig. 1285) with a tendency to sag downwards, viz. Para-umbilical herniæ increase steadily in size and frequently attain very large dimensions (fig. 1286). The neck of the sac is often remarkably narrow as compared with the size of the sac and the volume of its contents, which consist of greater omentum often accompanied by small intestine and, alternatively or in addition, a portion of the transverse colon.

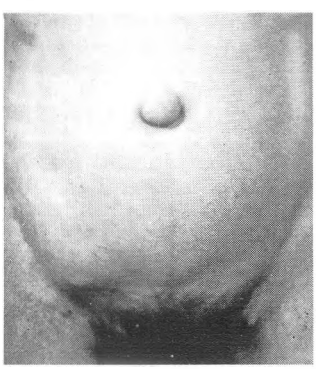


FIG. 1284.—Small.

FIG. 1285.—Large. Para-umbilical hernia.

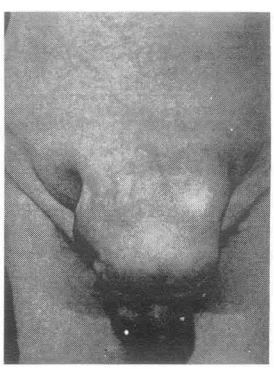


FIG. 1286.—Very large.

In old-standing cases the sac sometimes becomes loculated due to adherence of omentum to its fundus.

Clinical Features.—Women are affected five times more frequently than men. The patient is usually corpulent and between the ages of thirty-five and fifty. Increasing obesity, with flabbiness of the abdominal muscles, and repeated pregnancy are important factors. These herniæ soon become irreducible because of omental adhesions within the sac. A large umbilical hernia causes a local dragging pain by its weight. Gastro-intestinal symptoms are common and are probably due to traction on the stomach or transverse colon. Often there are transient attacks of intestinal colic due to subacute intestinal obstruction. In old-standing cases intertrigo of the adjacent surfaces of the skin is a troublesome complication.

Treatment.—Untreated, the hernia increases in size, and more and more of its contents become irreducible. Eventually, strangulation may occur. Therefore without undue delay operation should be advised in nearly all cases. If the patient is obese and the hernia is symptomless, operation can be postponed with advantage until weight has been reduced.

Mayo's Operation.—A transverse elliptical incision is made around the umbilicus. The subcutaneous tissues are dissected off the rectus sheath to expose the neck of the sac. The neck is incised to expose the contents. Intestine is returned to the abdomen. Any adherent omentum is freed, and ligated by transfixion if it is bleeding. Excess adherent omentum can be removed with the sac if necessary. The sac is then removed and the peritoneum of the neck closed with catgut. The aponeurosis on both sides of the umbilical ring is incised transversely for 2·5 cm or more—sufficiently to allow an overlap of 5 or 7·5 cm. Three to five mattress sutures are then inserted into the aponeurosis as shown in fig. 1287. When this row of mattress sutures has been tied, the over-lapping upper margin is stitched to the sheath of the rectus abdominis and the midline aponeurosis. It is important to denude this area of fat before stitching the flap in position. In fat patients, who ooze blood and liquid fat, a drain is provided at each end of the wound. The subcutaneous fat and skin are then approximated with deep sutures. If the patient has a tendency to bronchitis, it is wise to prescribe antibiotic therapy and breathing exercises.

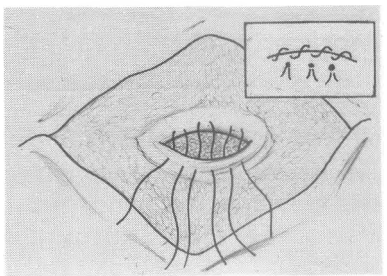


FIG. 1287.—Mayo's operation for umbilical hernia.

A few operators, dissatisfied with the results of Mayo's operation (which is acclaimed generally to be satisfactory), prefer to employ the fascial flap operation depicted in fig. 1288.

Additional Lipectomy.—In patients with a para-umbilical hernia associated with a large pendulous, fat-laden abdominal wall the operation can, with great advantage, be combined with lipectomy by fashioning the incisions to embrace a larger area of the fat-laden superficial layers of the abdominal wall.

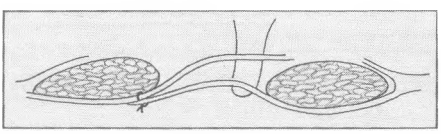


FIG. 1288.—Method of repairing a gap in the linea alba. (*After Charles Wells, F.R.C.S.*)

Strangulation is a frequent complication of a large para-umbilical hernia in adults. Owing to the narrow neck and the fibrous edge of the linea alba, gangrene is liable to supervene unless early operation is carried out. Also it should be remembered that in large hernias, the presence of loculi may result in a strangulated knuckle of bowel in one part of an otherwise soft and non-tender hernia.

Operation.—In early cases the operation does not differ from that for non-strangulated cases. Gangrenous contents are dealt with as in other situations. If a portion of the transverse colon is gangrenous, it should be exteriorised by the Paul-Mikulicz method (fig. 1152) and the gangrenous portion excised. If the ring is large enough to transmit the colon unhampered, it is left alone; otherwise it is enlarged. It is important that the small intestine be thoroughly scrutinised as a small loop may have been trapped and slipped back when the constriction was relieved. If non-viable gut is overlooked, peritonitis quietly supervenes, and the symptoms are ascribed to 'post-operative discomfort'. The condition of the patient steadily deteriorates until she succumbs after a few days (Franklin).

William James Mayo, 1861–1939. Surgeon, The Mayo Clinic, Rochester, Minnesota, U.S.A., described this operation in 1901.
Richard Harrington Franklin, Contemporary. Consulting Surgeon, Royal Postgraduate Medical School, London.

INCISIONAL HERNIA

(*syn.* VENTRAL HERNIA: POST-OPERATIVE HERNIA)

Ætiology.—An incisional hernia usually starts as a symptomless, partial disruption of the deeper layers of a laparotomy wound during the immediate or very early post-operative period. The concept that the hernia occurs during late convalescence is erroneous. A soundly healed laparotomy scar will withstand the buffeting of any walk of life, including repeated pregnancy. Often the event passes unnoticed if the skin wound remains intact after the stitches have been removed. A serosanguinous discharge is often the signal of dehiscence (p. 1130), and calls for systematic palpation of the immediate proximity of the incision, with the examiner duly masked and gloved. Re-suture of the deeper disrupted layers of the incision obviates the more difficult repair of an established hernia later on.

Incisional hernia occurs most often in obese individuals, and a persistent post-operative cough and post-operative abdominal distension are its precursors. There is a high incidence of incisional hernia following operations for peritonitis because, as a rule, the wound becomes infected. The accommodation of a necessary drainage tube in a stab incision, as opposed to placing such a tube through the laparotomy wound, reduces the frequency of its occurrence.

Clinical Features.—There are great variations in the degree of herniation. The hernia may occur through a small portion of the scar, often the lower end. More frequently there is a diffuse bulging of the whole length of the incision, A post-operative hernia, especially one through a lower abdominal scar. usually increases steadily in size, and more and more of its contents become irreducible. Sometimes the skin overlying it is so thin and atrophic that normal peristalsis can be seen in the underlying coils of intestine. Attacks of subacute intestinal obstruction are common, and strangulation is liable to occur at the neck of a small sac or in a loculus of a large one.

Treatment.—*Palliative.*—An abdominal belt is sometimes satisfactory, especially in cases of a hernia through an upper abdominal incision.

Operation.—Many procedures are advocated.

Pre-operative Measures.—In order to obtain a lasting repair, very special preparation is required. If the patient is obese, reduction by dieting should precede the operation. To attempt to return the contents of a very large hernia to the main abdominal cavity if they have not been there for several years is to court danger, unless weight reduction has been effected. In these circumstances, not only is there a risk of failure of the hernioplasty, but there is a greatly increased risk of paralytic ileus from visceral compression, and of pulmonary complications from elevation of the diaphragm.

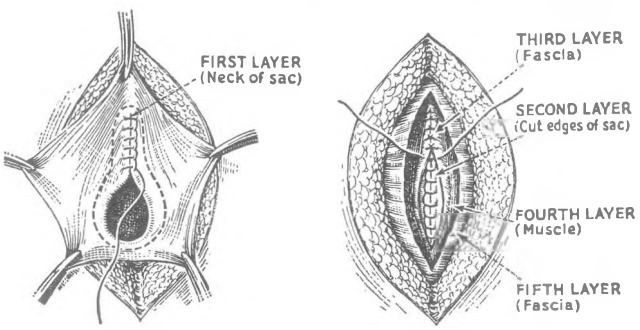
The 'Keel' Operation (Rodney Maingot).—In this operation, the hernial sac is not opened. It is dissected out and pushed back into the abdomen, and by a series of inverting and pleating layers of unabsorbable sutures the abdominal deficiency is closed. This repair, viewed as a cross-section, looks something like the keel of a

ship (fig. 1289). The incidence of post-operative ileus and distension is less than that occurring after operations in which the hernial sac is opened. The technique includes: (*a*) A wide excision of skin and scar tissue overlying the sac. Special care must be taken not to damage any bowel which is often adherent to the underside of the sac, (*b*) an extensive mobilisation of skin-flaps and fat, and exposure of healthy and strong aponeurosis around the neck of the sac, (*c*) inversion of the sac into the abdomen, pleating it with strong interrupted unabsorbable sutures, care being taken not to damage any underlying bowel. A second, third, or fourth layer of sutures are inserted until, finally, the healthy margins of the aponeurosis are brought firmly together. Any degree of tension in this final layer can be averted by a few relaxation incisions in the aponeurosis, parallel to the suture line.

FIG. 1289. — Cross-section of the 'keel' operation.

When the scar is paper-thin and devoid of normal skin, Ibrahim advises that no attempt should be made to separate dermal scar from sac—both should be inverted as one.

Cattell's Operation.—In this operation the hernial sac is dissected out with equal care, but then it is formally opened, the viscera are reduced, and the repair starts from the inside margins of the sac (fig. 1290). During the freeing of the contents of the sac any adherent omentum can be ligated and divided, leaving a portion of it attached to the fundus of the sac and skin. The layers of repair are as follows: The peritoneum and the abdominal wall are approximated at the neck with an interlocking suture of thick chromic catgut, including all layers of the abdominal wall that are attached to the hernial ring (fig. 1290(*a*)). An incision is made through the sac as shown by the dotted line in fig. 1290(*a*). (2) The cut edges of the base of the sac are approximated with unabsorbable sutures. An elliptical incision is made 2 cm. lateral to the previous suture line. (3) The medial borders of the incision are approximated. The lateral edges of the fascia are freed from the overlying muscles for some distance, and this fascial layer is approximated with interrupted sutures at the upper and lower ends of the wound. This is done to relieve tension on the muscles. (4 and 5) The muscles and the remaining fascial layer are approximated by alternating stitches (fig. 1290(*b*)). Tension-relaxing incisions may be required and should be placed well laterally.



(*a*) (*b*)

FIG. 1290.—(*a*). The closure of the hernial defect from inside the sac. Dotted line indicates the excess sac. (*b*). Approximation of the third layer. (*After R. B. Cattell.*)

Plastic Fibre Mesh or Net closures.—These are becoming increasingly popular. The sac is dealt with in either of the methods just described. Any deficiency in the abdominal wall can very easily be bridged without tension by laying and sewing on a sheet of tantalum gauze, or a mesh or net made of Nylon or Dacron, cut to well oversize and tacked down to the aponeurosis.

Careful haemostasis and meticulous asepsis are essential during these operations. Post-operative collections of serum can be removed by drainage, using plastic tubing led, via skin punctures lateral to the wound, into closed suction drainage bottles (e.g. Redi-vac).

Post-operative Treatment.—Gastric decompression and intravenous fluids are employed, and nothing by mouth allowed until the bowels have functioned. Early ambulation is to be discouraged, but exercises, especially of the legs, are to be encouraged. The patient should not resume strenuous exercise for three months.

Hassan Ibrahim, Contemporary. Professor of Surgery, Kasr-el-ain Hospital, Cairo University.
Richard Bartley Cattell, 1900–1964. Surgeon, Lahey Clinic, Boston, Mass., U.S.A.

EPIGASTRIC HERNIA

(*syn.* FATTY HERNIA OF THE LINEA ALBA)

An epigastric hernia occurs through the linea alba anywhere between the xiphoid process and the umbilicus, usually midway between these structures. Such a hernia commences as a protrusion of extra-peritoneal fat through the linea alba, where the latter is pierced by a small blood-vessel. Sometimes more than one hernia is present.

A swelling the size of a pea consists of a protrusion of extraperitoneal fat only (fatty hernia of the linea alba (figs. 1291 and 1292)). If the protrusion

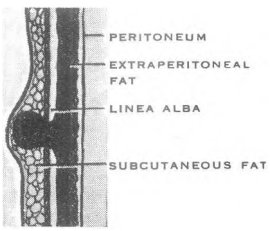


FIG. 1291.—Fatty hernia of the linea alba.

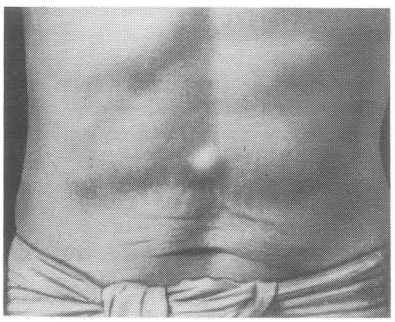


FIG. 1292.—Epigastric hernia.

enlarges, it drags a pouch of peritoneum after it, and so becomes a true epigastric hernia. The mouth of the hernia is rarely large enough to permit a portion of a hollow viscus to enter it; consequently, either the sac is empty or it contains a small portion of greater omentum.

It is probable that an epigastric hernia is the direct result of a sudden strain tearing the interlacing fibres of the linea alba. The patients are often manual workers between thirty and forty-five years of age.

Clinical Features:

(*a*) *Symptomless.*—A small fatty hernia of the linea alba can be felt better than it can be seen, and may be symptomless, being discovered only in the course of routine abdominal palpation.

(*b*) *Painful.*—Sometimes such a hernia gives rise to attacks of local pain, worse on physical exertion, and tenderness to touch and tight clothing, possibly because the fatty contents become nipped sufficiently to produce partial strangulation.

(*c*) *Referred Pain* (Peptic ulcer cases).—It is not uncommon to find that the patient, who may not have noticed the hernia, complains of pain suggestive of a peptic ulcer. On the other hand, a patient suffering from a gastric or duodenal ulcer may attribute all his symptoms to an epigastric hernia. In many cases of epigastric hernia there is coexistent upper abdominal disease.

Treatment.—If the hernia is giving rise to symptoms, operation should be undertaken.

Operation.—An adequate vertical or transverse incision is made over the swelling, exposing the linea alba. The protruding extraperitoneal fat is cleared from the hernial orifice by gauze dissection. If the pedicle passing through the linea alba is slender, it is separated on all sides of the opening by blunt dissection. After ligating the pedicle, the small opening in the linea alba is closed by unabsorbable sutures. When a hernial sac is present it is opened and any contents reduced, after which the sac is excised before repairing the linea alba. Sometimes smaller protrusions of fat are found above and below the hernia. These should also be dealt with.

FIG. 1293. — Interparietal hernia in an adult male dwarf with congenital deformities of the lower limbs. It contained appendix, 60 cm of ileum and jejunum, and the right testis. (*J. Samuel, F.R.C.S., F.A.C.S., Quilon, Kerala, India.*)

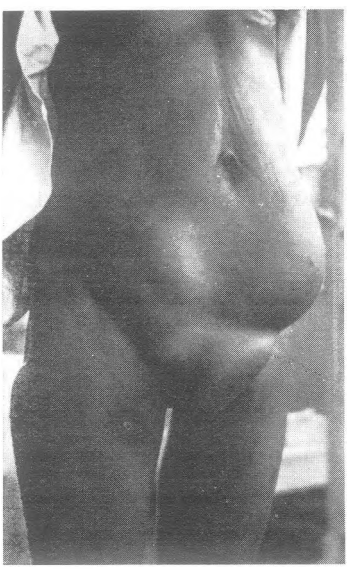


FIG. 1294.—Bilateral interstitial and bilateral inguinal hernias. The interstitial hernias were between the external and internal oblique muscles, the left containing several feet of small bowel. (*V. J. Hartfield, formerly S.E. Nigeria.*)

DIVARICATION OF THE RECTI ABDOMINIS

Divarication of the recti abdominis is seen principally in elderly multiparæ. When the patient strains, a gap can be seen between the recti abdominis through which gap the abdominal contents bulge. When the abdomen is relaxed the fingers can be introduced between the recti.

Treatment.—An abdominal belt is all that is required.

A similar condition is met with in babies, only the divarication exists above the umbilicus. No treatment is necessary; as the child develops a spontaneous cure results.

RARE EXTERNAL HERNIAS

Interparietal Hernia (*syn.* Interstitial Hernia).—An interparietal hernia has a hernial sac that passes between the layers of the anterior abdominal wall. The sac may be associated with, or communicate with, the sac of a concomitant inguinal or femoral hernia. Lack of knowledge of this condition is the cause of misdiagnosis and mismanagement (fig. 1293).

Other Varieties (figs. 1294 and 1295): 1. *Properitoneal* (20 per cent.).—Usually the sac takes the form of a diverticulum from a femoral or inguinal hernia.

2. *Intermuscular* (60 per cent.).—The sac passes between the muscular layers of the anterior abdominal wall, usually between the external oblique and internal oblique muscles. The sac is nearly always bilocular, and is associated with an inguinal hernia.

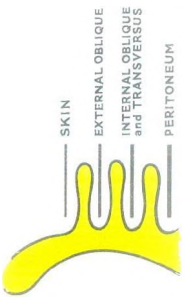


FIG. 1295.—The planes that an interparietal hernia may occupy.

3. *Inguino-superficial* (20%).—The sac expands beneath the superficial fascia of the abdominal wall or the thigh. This type is commonly associated with an incompletely descended testis.

Clinical Features.—The incidence in the male is higher than in the female. Most patients present with intestinal obstruction, due to obstruction or strangulation of the hernia. In these circumstances the cause of the obstruction should be apparent in the intermuscular and inguino-superficial varieties, but in the properitoneal variety, as no swelling is likely to be apparent, delays in diagnosis occur and consequently the mortality in this variety is high.

Treatment.—Operation is imperative because of intestinal obstruction. In all cases of inguinal and femoral hernia a finger should be introduced into the sac with the object of detecting an unsuspected intraparietal loculus.

A Spigelian hernia is a variety of interparietal hernia. This type of hernia is one occurring through the Spigelian fascia, commonly at the level of the arcuate line (fig. 1296). The fundus of the sac, clothed by extraperitoneal fat, may lie beneath the internal oblique muscle, where it is virtually impalpable. More often it advances through that muscle and spreads out like a mushroom between the external and internal obliques and gives rise to a more evident swelling. The patient is often corpulent, and usually over fifty years of age, men and women being affected equally. Typically, a soft, reducible mass will be encountered lateral to the rectus muscle and below the umbilicus. After reduction a small oval defect can often be felt and because of the rigid fascia surrounding its neck, strangulation may occur.

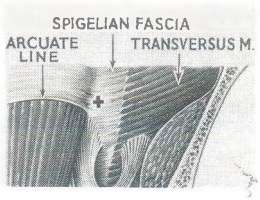


FIG. 1296.—The usual site for a Spigelian hernia.

Treatment.—Operation should be advised and repair is simple. The external oblique aponeuosis is split. After isolating the sac, dealing with any contents, and ligating and excising it, the transversus, internal oblique, and external oblique muscles are approximated in layers.

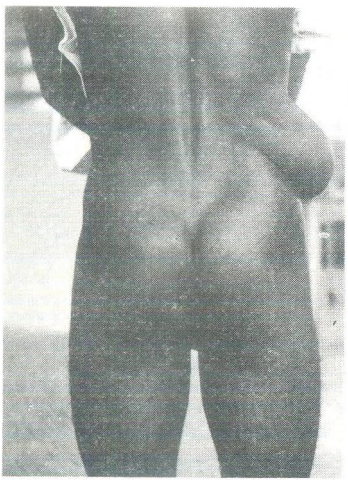


FIG. 1297.—Inferior lumbar hernia. It contained cæcum, appendix and small bowel. Note filiarial skin rash on buttocks. (*V. J. Hartfield, Sussex.*)

Lumbar Hernia. — Most primary lumbar herniæ gain exit through the inferior lumbar triangle of Petit (fig. 1297), bounded below by the crest of the ilium, laterally by the external oblique, and medially by the latissimus dorsi. Some come through the superior lumbar triangle, which is bounded by the twelfth rib above, medially by the sacrospinalis, and laterally by the posterior border of the internal oblique. An extensive incisional lumbar hernia sometimes follows an operation upon an infected kidney, in which the wound suppurated post-operatively.

Differential Diagnosis.—A lumbar hernia must be distinguished from: (*a*) Lipoma. (*b*) A cold abscess pointing to this position. (*c*) Phantom hernia due to local muscular paralysis. Lumbar phantom herniæ can result from any interference with the nerve-supply of the affected muscles (e.g. poliomyelitis).

Treatment.—A primary lumbar hernia, being small, does not entail an extensive operation for its repair. On the other hand, a post-operative lumbar hernia may be large, and the defect is impossible to repair unless fascial flaps are used. When necessary, the area can be reinforced still further by stitching in place a piece of tantalum gauze or Dacron mesh.

Perineal hernia (*syn.* hernia through the pelvic floor) is rare, though not uncom-

Andrien Van der Spieghel (Spigelius), 1578–1625. Professor of Anatomy, Padua, Italy.
Jean Louis Petit, 1664–1750. Director of the Academy of Surgery, Paris.

mon in the ageing dog—a reducible swelling appears immediately lateral to the anus.

(*a*) *Post-operative hernia through a perineal scar* may occur after excision of the rectum.

(*b*) *Median sliding perineal hernia* is a complete prolapse of the rectum (p. 1099).

(*c*) *Antero-lateral perineal hernia* occurs in women and presents as a swelling of the labium majus.

(*d*) *Postero-lateral perineal hernia*, which passes through the levator ani to enter the ischio-rectal fossa.

Treatment.—A combined operation is generally the most satisfactory for (*c*) or (*d*). The hernia is exposed by an incision directly over it. The sac is opened and its contents are reduced. The sac is cleared from surrounding structures and the wound is closed. With the patient in semi-Trendelenburg position, the abdomen is opened and the mouth of the sac is exposed. The sac is inverted, ligated, and excised, and the pelvic floor is repaired as adequately as possible.

Obturator Hernia.—The hernia, which passes through the obturator canal, occurs six times more frequently in women than in men. Most of the patients are over sixty years of age. The swelling is liable to be overlooked because it is covered by the pectineus. It seldom causes a definite swelling in Scarpa's triangle, but if the limb is flexed, abducted, and rotated outwards, sometimes the hernia becomes more apparent. The leg is usually kept in a semi-flexed position and movement increases the pain. In more than 50 per cent. of cases of strangulated obturator hernia pain is referred along the obturator nerve by its geniculate branch to the knee. On vaginal or rectal examination the hernia sometimes can be felt as a tender swelling in the region of the obturator foramen.

Cases of obturator hernia which present themselves have usually undergone strangulation, which is often of the Richter type (p. 1136).

Treatment.—(*a*) Perform lower laparotomy (on the side of the lesion, if known). Confirm the diagnosis and then adopt full Trendelenburg's position; (*b*) the constricting agent is the obturator fascia. Taking every precaution to avoid spilling infected fluid from the hernial sac into the peritoneal cavity, this fascia can be stretched to allow reduction by inserting suitable forceps through the gap in the fascia and opening the blades with care. If incision of the fascia is required, it is made parallel to the obturator vessels and nerve; (*c*) the contents of the sac are dealt with; (*d*) the broad ligament is stitched over the opening to prevent recurrence.

Gluteal and Sciatic Herniæ.—A *gluteal hernia* passes through the greater sciatic foramen, either above or below the piriformis. A *sciatic hernia* passes through the lesser sciatic foramen. *Differential diagnosis* must be made between these conditions and (*a*) A lipoma or fibro-sarcoma beneath the gluteus maximus. (*b*) A tuberculous abscess. (*c*) A gluteal aneurysm. All doubtful swellings in this situation should be explored by operation.

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig, Germany.
Antonio Scarpa, 1747–1832. Professor of Anatomy, Pavia, Italy. Elected F.R.S. in 1791.

CHAPTER 55

URINARY SYMPTOMS. INVESTIGATION OF THE URINARY TRACT. ANURIA

URINARY SYMPTOMS

THREE symptoms, a veritable triple alliance, accompany most urinary affections. They are pain, frequency, and hæmaturia.

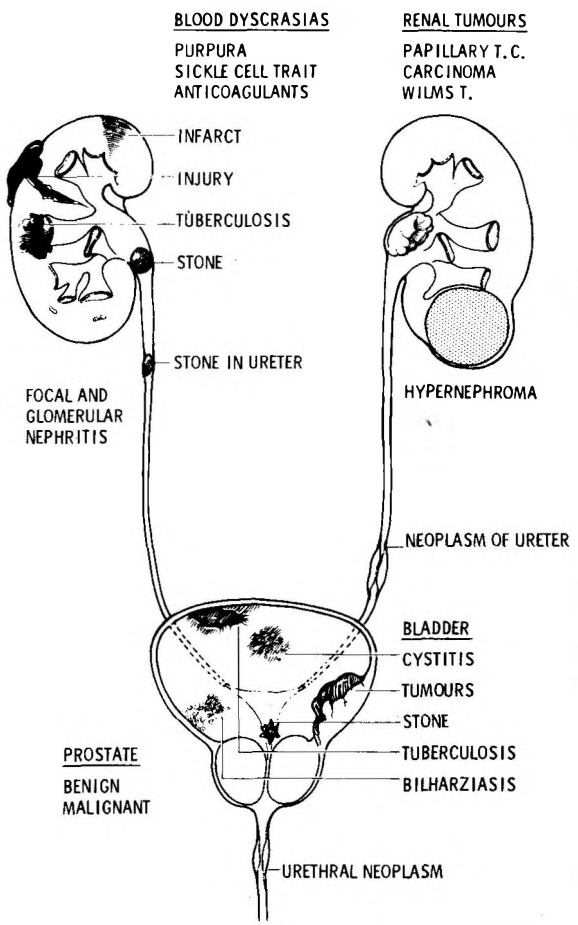


FIG. 1298.—The more common causes of hæmaturia.

Pain, associated with affections of the urinary tract embraces :

Renal pain is usually a fixed dull ache situated mainly in the costo-vertebral angle, but also in the upper and outer quadrant of the abdomen. It may be due to stretching of the renal tissue or irritation of the wall of the kidney pelvis.

Renal[1] *and ureteric colic,* a violent spasmodic pain, is due to obstruction producing distension and increased muscular activity.

Vesical pain varies from slight suprapubic discomfort to strangury, which is agonising pain referred to the external urinary meatus, accompanied by an intense desire to micturate, but resulting in the expulsion (by straining) of only a few drops of blood-stained urine.

Prostatic and seminal vesicular pain is deep-seated in the rectum ; it is often referred to the perineum, but sometimes to the suprapubic region or one or both iliac fossæ.

[1] Renal colic is a term that is deeply rooted. Pain passing from loin to groin is more accurately termed ureteric colic.

Urethral pain is scalding, and occurs during micturition.

Frequency may be due to incomplete emptying, as in prostatism, to irritability of the bladder as by inflammation or stone, to a contracted bladder, to a diuresis, or sphincter weakness.

Hæmaturia.—Blood in the urine, however transient, is a symptom that must never be ignored. Painless hæmaturia is so often a herald of new-growth in some part of the urinary tract that a complete urinary investigation should be implemented immediately. When hæmaturia is painful, the patient demands relief, and the task of persuading him to undergo a thorough examination is less difficult. The causes of hæmaturia are manifold, and the more common ones are depicted diagrammatically in fig. 1298.

Pink or red urine may occur after eating beetroot, while many drugs, e.g. Dindevan, Pyridium, and Furadantin may make the urine a deep orange red. Actual bleeding occurring while a patient is on anticoagulants should make the practitioner consider the possibility of unsuspected renal or vesical pathology.

INVESTIGATION OF THE URINARY TRACT

A general physical examination must be included in the investigation of any urinary tract condition. Of particular importance are the state of the heart, the blood-pressure, the chest (chest X-ray). A *hæmoglobin estimation* is essential, as anæmia is a close and aggravating partner to uræmia.

1. **The urine** is examined chemically and bacteriologically. For the latter a fresh midstream specimen of urine is essential for (*a*) it avoids contamination as far as possible and (*b*) it is without the risks of catheterisation.

2. **Tests of Renal Function.**—Because of the large reserve of renal tissue, considerable structural damage can occur before functional damage becomes apparent. Kidney function can be impaired in three principal ways: the renal plasma-flow may be reduced, the glomeruli damaged, or the function of the tubules impaired. In essential hypertension the chief fault lies in the renal plasma-flow. In glomerulonephritis the main effect is reduction in the glomerular filtration rate, while in pyelonephritis the brunt falls on tubular function. In back pressure due to obstruction all become impaired.

Range of Specific Gravity (concentration dilution test).—The power of the kidneys to concentrate and dilute urine is a good test of their functional integrity. Fluid is withheld for twelve hours overnight. The specific gravity of the first two morning specimens should reach 1·020; a specific gravity of 1·025 in a urine free of protein indicates good renal function. A litre of water is given by mouth. Within four hours the specific gravity should be as low as 1·002. A fixed specific gravity of 1·010 under these varying circumstances is good presumptive evidence of impaired function of the distal renal tubules.

The blood urea (or non-protein nitrogen) is normally between 20 and 40 mg. per 100 ml (3·3–6·6 mmol/l) of blood. A reading above 40 mg (6·6 mmol/l) is suspicious, and one more than 50 mg (8·3 mmol/l) is indicative that the kidney function is impaired by at least 50 per cent.

Creatinine Clearance.—This is a convenient method of measuring the glomerular filtration rate. One sample of blood and an accurately timed collection of urine for a period of about twenty-four hours are all that is required. The estimation of creatinine in each, enables a clearance to be arrived at. The normal clearance is between 80 and 120 ml. per minute. It decreases in old age.

3. **Intravenous Pyelography (I.V.P.).**—Strictly speaking this is *urography*, because more than the kidney pelvis is examined. It is a contrast

X-ray, and the radio-opaque medium employed is sodium diatrizoate (*syn.* Hypaque) which is mainly secreted by the proximal tubules.

Contraindications.—(*a*) When renal function is poor, secretion is insufficient to produce a reasonable film. (*b*) Idiosyncrasy to iodine must be excluded by a small preliminary test dose (1 ml. i.v.). A history of asthma or other allergic conditions should put one on guard in this respect. (*c*) A suspicion of myelomatosis. Precipitation of the myeloma protein can produce anuria.

If untoward signs develop during the injection, stop the injection at once. Give oxygen for anoxia, a pressor drug for low blood pressure, dextrose-saline solution for shock which is to be followed by dextran if needed. Important antidotal drugs in the treatment of acute sensitivity reactions are (*a*) anthisan, 100 mg. intravenously or (*b*) hydrocortisone, 100 mg. intravenously.

Preparation.—A mild vegetable aperient is given on two evenings preceding the examination. No fluid should be taken for 8 hours before the investigation, and the patient is instructed to empty the bladder just before the injection.

Technique.—40–60 ml. of sodium diatrizoate (Hypaque 45 per cent.) are injected into a suitable vein, usually an arm, with the patient recumbent. None of the solution must be permitted to escape into the tissues around the vein, for it is irritating. Rapid injection and exposure of a film immediately the injection is completed will give valuable nephrograms. If the kidneys are functioning normally, the medium appears simultaneously in the calyces and pelves of both kidneys two minutes later. As a routine, X-ray films are exposed at 5, 10, 15, and 25 minutes.

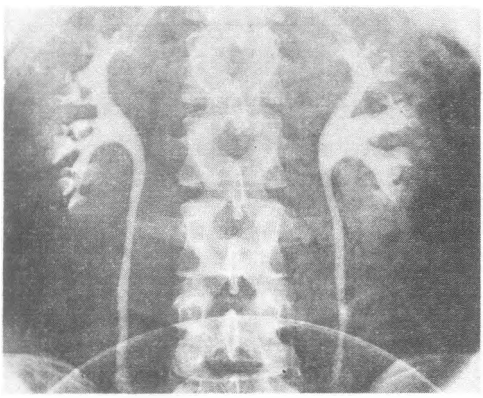


FIG. 1299.—Normal excretory pyelograph (I.V.P.).

Compression applied to the abdomen by a strap enhances the clarity of the pyelographs, because it delays emptying of the renal pelvis. The concentration reaches its maximum in 15 to 30 minutes (fig. 1299). After the 25-minute film has been taken, a film in the erect position is valuable: (i) to demonstrate the mobility of the kidneys, (ii) to visualise the ureters, (iii) to show emptying of the kidneys. Normally, visible excretion is completed in one to one and a half hours. The later radiographs show the bladder filled with the medium. In order to display the termination of the ureters, the patient should micturate immediately before the final radiograph. The amount of residual urine in the bladder, if any, can also be demonstrated in this way. When the renal pelves and calyces fail to empty within the normal limits, further radiographs are taken at half-hourly intervals.

Infusion Pyelogram.—In patients with a blood urea of 100 to 200 mg/100 ml (16·6–33·2 mmol/l), an infusion 50 per cent. Hypaque 2 ml/kg in an equal volume of normal saline, over a period of 10 minutes, may produce useful urograms.

4. **Instrumental Examination of the Urinary Organs** can be accomplished under local anæsthesia.

In the Male.—If the prepuce is present, it is retracted and all smegma is removed by washing with soap and water followed by swabbing with a mild antiseptic solution. Cocaine is dangerous, absorption via the urethral mucosa into the circulation sometimes causing collapse and even death. 1 per cent. lignocaine with 0·25 per cent. chlorhexidine gluconate as a local antiseptic in a jelly base (Lidothesin) is safe and effective. About 15 ml. is squeezed into the urethra and retained for five minutes with the aid of a penile clamp placed proximal to the glans.

In the Female.—The vulva is washed with soap and water followed by swabbing with a mild antiseptic solution. The labia minora are held apart and the local anæsthetic jelly is injected into the urethra.

Bouginage is described on p. 1298, and **Catheterisation** on p. 1268.

Cystoscopy.—By cystoscopy, the interior of the bladder is inspected and the ureteric orifices observed. If 7 ml. of a 0·4% solution (adult dose) of *indigo-carmine* are injected intravenously, the excretion of the dye down the ureters can be watched; normally it should appear within four or five minutes.

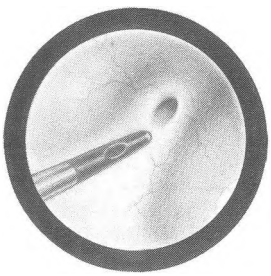


FIG. 1300.—A ureteric catheter about to enter the left ureteric orifice. Cystoscopic view.

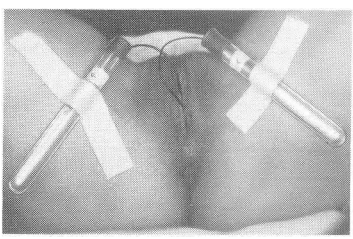


FIG. 1301.—Specimens of urine being collected after ureteric catheterisation. The test-tubes are capped with rubber finger-stalls.

Delay of excretion of the dye on one side is indicative of unilateral urinary obstruction or disease, while delay on both sides suggests bilateral renal impairment. By employing a catheterising cystoscope, ureteric catheters can be passed up each ureter (fig. 1300), and specimens of urine collected from each kidney (fig. 1301).

Retrograde (*syn.* **instrumental**) **pyelography** is employed when a clearer definition than that afforded by the intravenous pyelogram is required, e.g. suspected renal neoplasm or early tuberculosis, or to collect urine from each kidney separately for differential functional tests. The ureters are catheterised, and the patient is conveyed to the radiological table. A plain radiograph is taken and developed before injecting the medium in order to ensure that the ureteric catheters are not too advanced. 25% sodium diatrizoate (hypaque) is used for the injection. An initial film after 3 to 4 ml. have been injected will outline a normal pelvis (fig. 1302). Should a large hydronephrosis be present, more medium will be required to obtain clear

In 1877, Max Nitze, Professor of Urology, Berlin, in conjunction with Beneche, an Optician of Vienna produced the first cystoscope.

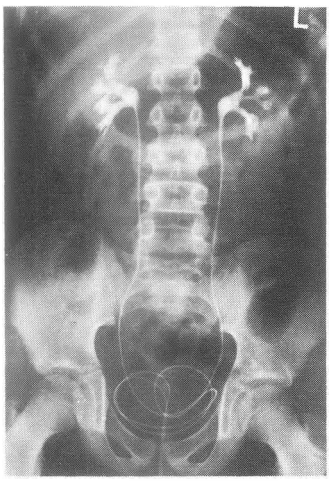


FIG. 1302.—Normal retrograde pyelogram. The definition is much clearer, consequently this procedure is invaluable in confirming doubtful abnormalities visualised by the excretory method, or where excretion is poor.

definition. By this method it is possible to inject the pelvis of both kidneys at the same sitting, but in cases of poor renal function it is essential to make each examination independently. In either event, after the X-ray exposure has been made, as much of the medium as possible is aspirated from the renal pelvis. Examination with the aid of an image intensifier gives immediate information of the size of the renal pelvis and calyces; the activity can be observed, and functional obstructions easily displayed.

Cystography: (*a*) *Excretory.*—The cystographs which accompany the later films of excretory pyelography may not be sufficiently dense to give a clear delineation of pathological conditions in the bladder.

(*b*) *Retrograde* is employed principally to confirm the presence and dimensions of a vesical diverticulum. A small catheter is passed and the urine in the bladder is evacuated. If a diverticulum is suspected, the patient lies on his face so as to favour drainage of urine from the diverticulum. A plain radiograph is taken to exclude a calculus in the diverticulum. The bladder is then filled with medium until the patient feels a desire to micturate. An excellent contrast medium for cystography is a 25 per cent. solution of iodoxyl (B.P.). A radiograph is taken with the bladder full, and again after the bladder has been emptied. If there is a diverticulum present, it will remain full of medium after the medium has left the main cavity (p. 1240). The extent of a very large neoplasm of the bladder can sometimes be demonstrated better by cystography than by cystoscopy.

(*c*) *Micturating cystograms* are best performed with an X-ray image-intensifier. The function of the bladder neck, and the presence of ureteric reflux of urine, can be observed on a monitoring screen as the patient empties his bladder. Simultaneous recording of bladder pressure with flow rates, alongside the X-ray display, on videotape are most useful in detecting unstable detrusor activity (fig. 1389).

Urethroscopy: (*a*) *Anterior urethroscopy* is employed when the presence of a urethral stricture (fig. 1298) or strictures is suspected, or in cases of chronic urethritis, in order to exclude or confirm the presence of an infected urethral crypt or a granuloma (a ' soft ' stricture). Anterior urethroscopy is conducted under air inflation of the urethra.

(*b*) *Posterior urethroscopy* permits inspection of the prostatic and membranous urethræ. It is carried out with an irrigating urethroscope. The most notable normal spectacle of posterior urethroscopy is the verumontanum, which presents as an eminence on the floor of the prostatic urethra. On the summit of this projection is the sinus pocularis (fig. 1303), with the ejaculatory ducts opening on either side. The verumontanum is reddened and enlarged in cases of chronic vesiculitis. In cases of chronic prostatitis, one or more of the numerous prostatic ducts, which normally are difficult to visualise, may be seen exuding pus. When the prostate is enlarged,

the lateral lobes project into the internal meatus producing an inverted V; a middle lobe or bar can be easily seen.

Urethrography.—Urethrography (fig. 1304) is especially valuable for gaining information concerning the length of a urethral stricture, of dilatation or diverticulum formation above a stricture, or of failure of the medium to pass a stricture. It also reveals dilated prostatic ducts in

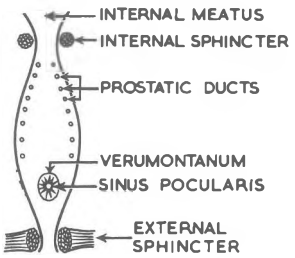


FIG. 1303.—Structures on the floor of the posterior urethra. *(After J. C. Ainsworth-Davis, F.R.C.S., London.)*

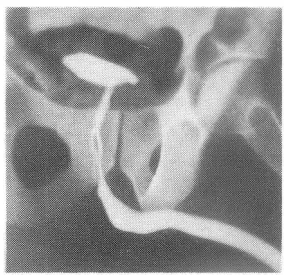


FIG. 1304.—Normal urethrograph.

chronic prostatitis and especially in tuberculous prostatitis. The one contraindication to its use is the presence of urethral hæmorrhage. The medium employed must be chosen with great care, for should there be a breach in the continuity of the lining membrane of the urethra, the medium may enter the circulation (fig. 1470).

Lipiodol brings with it the danger of oil embolus and this medium should never be employed. Even worse is to inject barium emulsion, and deaths have occurred from its use in cases of ruptured urethra.

The medium must therefore be harmless should some of it enter the circulation, and the injection must not be rapid or forceful. For general purposes the most satisfactory medium is Umbradil viscous V. This is a jelly which is squeezed from a tube into the butt end of a urethral syringe. It is injected easily and contains the local anæsthetic lignocaine. The injection is made most satisfactorily by employing Knutsson's apparatus which has a penile clamp attached (fig. 1305).

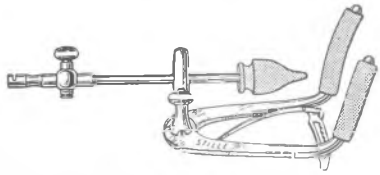


FIG. 1305.—Knutsson's apparatus for urethrography.

Renal Arteriography (fig. 127)

Renal arteriography can provide information unobtainable by retrograde pyelography. To be enabled to visualise the renal arterial architecture is of diagnostic value in the following conditions.

1. Hypertension, if unilateral ischæmia (e.g. due to renal artery stenosis) is suspected. 2. Renal neoplasm. 3. Renal cyst. 4. Chronic pyelonephritis. 5. Aneurysm of the renal artery.

There are two methods of performing renal arteriography.

(*a*) **Translumbar aortography** is carried out under general anæsthesia. A needle is passed into the abdominal aorta just above the renal arteries (level of L.I.), the contrast medium is injected and four films are taken in rapid succession. To minimise accidents a small quantity of medium is injected and a film taken to ensure that the needle is in its correct position (p. 141).

(*b*) **Retrograde Arteriography.**—A Seldinger's needle is used (fig. 128). The needle is passed into the femoral artery. The stilette is removed. A guide wire (a

Folke Knutsson, Contemporary. Director, Roentgen Department, University Hospital, Uppsala, Sweden.

' leader ') with a supple tip is passed into the artery and the needle is removed. The artery is compressed to avoid bleeding while a radio-opaque arterial catheter is threaded on to the leader and so guided through the skin and arterial puncture into the artery. The catheter is advanced to the desired level. The contrast fluid (Hypaque) is then injected. *Selective Renal Arteriography* can be achieved by using a hooked catheter, and guiding it into the renal artery (fig. 1306).

6–7 ml of contrast fluid are usually sufficient for selective arteriography; 30 ml for a free flush into the aorta. The dangers of the procedure (*a*) tubular necrosis of the kidneys, (*b*) paraplegia, often temporary, have been eliminated with modern contrast fluids, but damage to a plaque of atheroma, embolism, and dissecting aneurysm can occur.

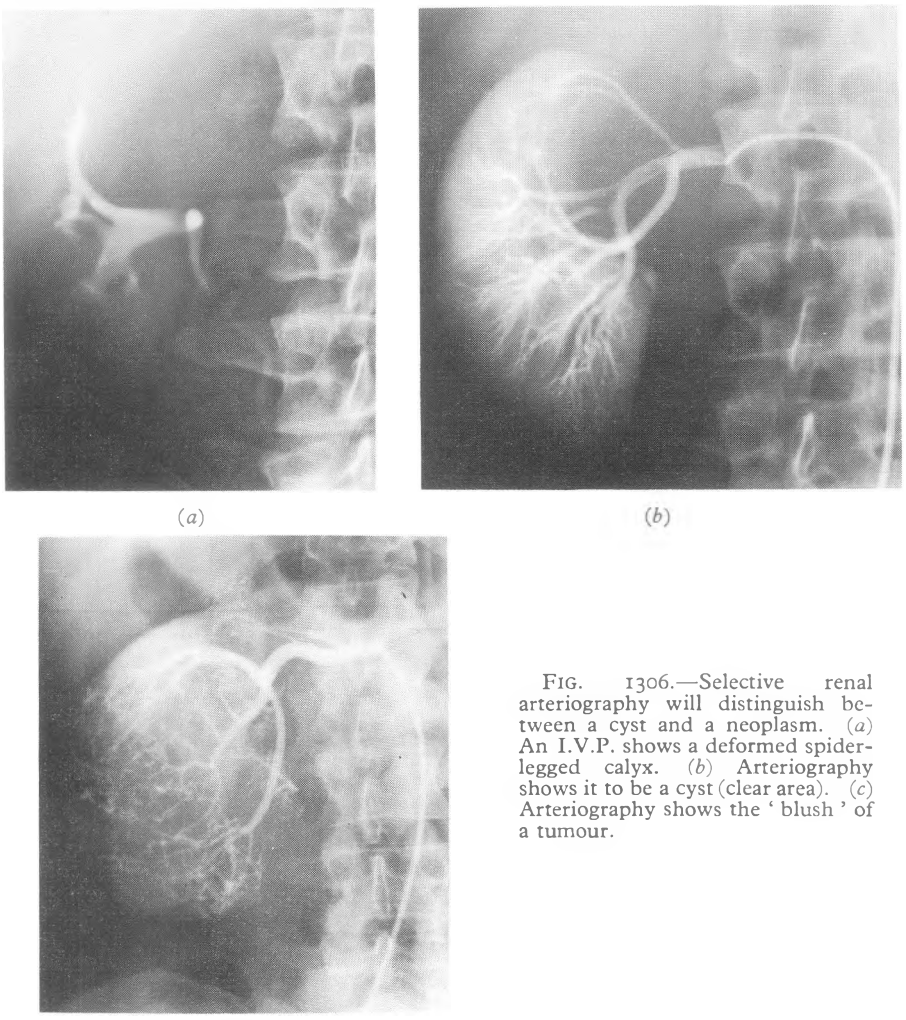


FIG. 1306.—Selective renal arteriography will distinguish between a cyst and a neoplasm. (*a*) An I.V.P. shows a deformed spider-legged calyx. (*b*) Arteriography shows it to be a cyst (clear area). (*c*) Arteriography shows the ' blush ' of a tumour.

Perirenal insufflation of oxygen to delineate a renal or adrenal swelling is seldom of use today.

Renal tomography is very useful in combination with other radiographic procedures as it can help in doubtful cases to differentiate between tumours and cysts and outline the renal substance.

Radio Isotope Renogram (fig. 1307).—A balanced pair of gamma-ray counters located over the kidneys measures the renal uptake and excretion of radioactive

Hippuran[1]. If urine, blood and an inactive body area are monitored at the same time additional information can be obtained. The rate of blood flow through the kidney, the rate of concentration, and the rate of clearance can be shown graphically. 1.

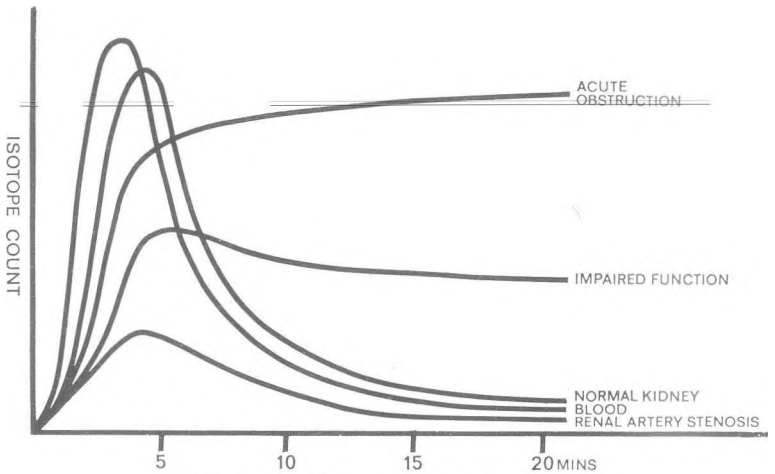


FIG. 1307.—Radio isotope renogram (see text).

Initial steep rise ($\frac{1}{2}$ minute) vascular phase. 2. Renal excretion and secretion in next 4–6 minutes. 3. Decline flattening to about or below blood levels. A comparison of function and even differential function can be assessed in this way.

Other scanning techniques.—*Gamma camera renography* with labelled Tectnetium provides excellent definition and improved functional assessments. *Ultrasound examination* which is valuable in differentiating solid from cystic lesions, and the *wholebody scanner with computerised axial tomography* (CATscan) which is very useful in differentiating the posterior abdominal wall masses (fig. 1308), both have the advantage of being non-invasive and do not subject the patient to any irradiation.

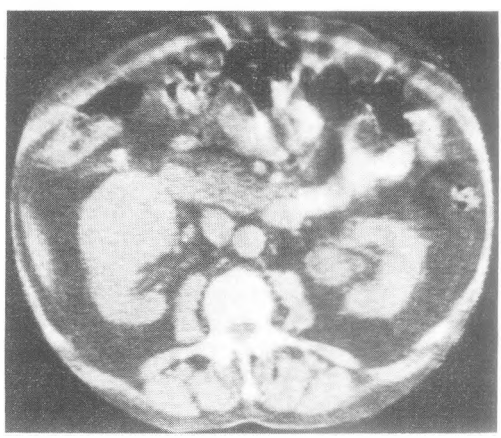


FIG. 1308.—CATscan showing hypernephroma. (N.B. CATscans are read from the feet of the patient. Here there is a right sided renal swelling on the left of the picture).

ANURIA (*syn.* SUPPRESSION OF URINE)

Oliguria should be defined as an excretion of less than 300 *ml. of urine in twenty-four hours. Anuria is an absence of excretion for twelve hours.*

The most helpful classification of anuria is into pre-renal, renal, and post-renal. Although suppression of urine is commonly and conveniently referred to as anuria, except in post-renal (ureteric obstruction) cases the suppression is seldom absolutely complete.

[1] Hippuran = Sodium iodohippurate. (Mallinckrodt, U.S.A.)

Pre-renal Anuria

The blood pressure in the glomeruli is normally about 90 mmHg (12 kPa); when the systolic blood pressure falls below 70 mm (9·3 kPa), filtration from glomeruli ceases. If the glomeruli are diseased, a higher pressure (up to 100 mmHg (13·3 kPa)) may be inadequate to maintain filtration. The causes of pre-renal anuria are traumatic shock, severe hæmorrhage, spinal anæsthesia, extensive burns, dehydration from vomiting, diarrhœa, or excessive sweating, and cardiac failure.

Treatment.—Blood transfusion in the case of hæmorrhage, the treatment of shock (p. 82) if that be the cause of the fall in the blood-pressure, or in cases of dehydration the administration of dextrose-saline solution intravenously often restores urinary excretion. If hypotension is long maintained, damage to the renal epithelium results, and the condition passes on to one of renal anuria, due to tubular necrosis.

Acute Renal Anuria (Acute tubular necrosis)

Acute renal anuria results from damage to, or ischæmia of, the renal tubular epithelium (fig. 1309). The principal causes met with in surgical practice are:

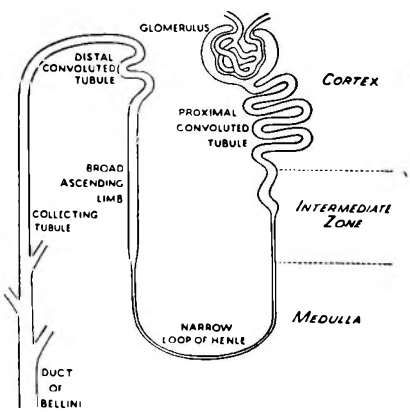


FIG. 1309.—Necrosis of the proximal convoluted tubule produces immediate anuria. (*After H. L. Sheehan.*)

1. Severe shock (hypotension) lasting two hours or more.
2. Incompatible blood transfusion.
3. Bilateral pyelonephritis.
4. The crush syndrome (p. 86).
5. Concealed accidental hæmorrhage and abortion.
6. Certain poisons. The most important are (*a*) media used for aortography, (*b*) the toxin of eclampsia, (*c*) chemicals—mercury salts, carbon tetrachloride.
7. Acute pancreatitis.
8. Operations on jaundiced patients.
9. Bacteræmic shock.

When renal anuria is reversible, and it is treated correctly, the condition is divided into three phases: 1. *The oliguric phase.* 2. *The phase of diuresis.* 3. *The phase of recovery.*

Clinical Features.—The average duration of the oliguric phase is ten to twelve days. Dark urine in small amounts and with a specific gravity of 1·010 is passed, or will be found on catheterisation. Anorexia is an early symptom which is followed by hiccough. Within four or five days there is copious effortless vomiting. Abdominal distension is common. Untreated, or treated incorrectly, the blood urea mounts by 20 to 30 mg (3·3–5·0 mmol) daily (fig. 1310). The systolic blood-pressure may be elevated—200 mm Hg (26·6 kPa) after two or three days.

Lorenzo Bellini, 1643–1704. Professor of Philosophy and Anatomy, Pisa, Italy.

Still untreated, or treated incorrectly, about the sixth day increasing drowsiness, thirst, a dry skin, and a dry brown tongue are characteristic findings. The respiratory and pulse-rates become slower and irregular, and Cheyne-Stokes' respirations are often in evidence. The final stages are usually characterised by muttering delirium followed by coma.

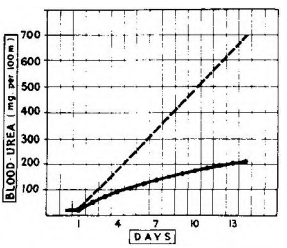


FIG. 1310.—Dotted line: rise of blood urea in untreated anuria. Solid line: same when treated by fluid limitation and protein-free diet. (*After G. M. Bull.*)

Treatment: Special Early Measures.—(*a*) If there is any doubt as to the cause, post-renal (obstructive) anuria must be eliminated by plain radiography and ureteric catheterisation (p. 1268). (*b*) In cases of incompatible blood transfusion, renal damage can sometimes be prevented by provoking a diuresis within an hour of the reaction—500 ml. of Rheomacrodex or mannitol (p. 82) is given intravenously. If no urine is secreted (the bladder is emptied naturally—or via a catheter), all fluids are restricted forthwith.

In all other circumstances of acute renal anuria it is imperative not to force fluids.[1] The important substances in the genesis of uræmia are not urea and other end products of nitrogen metabolism, but water and electrolytes, such as potassium. If patients receive 3,500 ml. or more per day, 75 per cent. die with signs of water overload, and evidence of pulmonary or cerebral œdema at necropsy. Another reprehensible practice is to prescribe potassium citrate for a patient on the threshold of suppression of urine; it has caused many deaths from hyperkalæmia.

Treatment of an Established Case.—The essentials are:

1. *Water Balance.*—In the absence of vomiting, the elimination of water by an anuric patient is limited to extra-renal routes, i.e. lungs, skin, and fæces. In a temperate climate this loss is 600 to 1,000 ml. per day. As it is estimated that there is a daily production of 400 ml. of water from the oxidation of body fat and proteins, the daily fluid intake of an anuric patient must be limited to 500 ml. plus an amount equal to that of the water vomited or recovered by gastric aspiration.

Additions must be made for diarrhœa and excessive sweating: regarding the latter, an extra 200 ml. is allowed for each 0·5 degree of the patient's temperature above 37·8° C. When the weather is exceedingly hot, the allowance is increased, but even in these circumstances it should rarely exceed 1,000 ml. per twenty-four hours.

Bedside evaluation—thirst, moisture of the tongue, and the skin—are excellent guides of adequate hydration.

2. *Electrolytic Balance.*—In the absence of diarrhœa and vomiting, the only effective channel for excretion of electrolytes is by the kidneys. Consequently, in anuria the administration of electrolytes often leads to gross

[1] According to Harrington, the pioneer of water closets, it does no good to keep trying to flush a lavatory when it is blocked!!!

John Cheyne, 1777–1836, and William Stokes, 1804–1878. (*See footnote, p. 100.*)

disturbances of electrolytic balance; in particular, hyperpotassæmia is liable to cause sudden death from cardiac arrest. The blood urea, of course, rises and the serum bicarbonate falls.

3. *Nitrogen Metabolism.*—The end-products of protein katabolism, other than urea, are possibly toxic. Furthermore, by the breakdown of both exogenous and endogenous protein, potassium is liberated. For these reasons it is essential to prohibit protein intake altogether, and to reduce endogenous nitrogen metabolism as much as possible. A high carbohydrate intake depresses endogenous nitrogen metabolism.

The Protein-free, Mineral-free, Strictly-limited-fluid Regimen.—The object is to provide 2,000 Calories (8,400 kJ) per day, and at the same time to limit fluid intake as detailed above. This is a diet upon which the patient can subsist without discomfort for periods up to three weeks (Bull). According to circumstances, the regimen can be varied:

(*a*) When the patient can take fluid by mouth without vomiting or undue nausea, a daily intake of 500 ml. of 40 per cent. lactose, given in small frequent amounts, is likely to be acceptable because the solution tastes less sweet than dextrose.

(*b*) When the patient is anorexic, nauseated, and prone to vomit, a plastic gastric tube is passed transnasally and the 500 ml. of 40 per cent. dextrose or lactose is gravitated by the drip method. A plastic tube is chosen because its relative stiffness makes it less likely to be expelled if the patient vomits. As far as possible the vomitus must be collected and measured, for obviously it is highly important to supplement the loss with precision. To stop the drip for two to four hours at a time helps some patients to retain the solution. Alternatively, 50 to 100 ml. of doubly centifruged cream is added to the dextrose solution. This enables the solution to be tolerated by some patients who otherwise would reject it.

(*c*) When the patient is unable to retain the fluid gravitated into the stomach, or toxæmia or a head injury makes him stuporous and therefore liable to inhale vomitus, there is but one alternative—to give the 40 per cent. dextrose solution intravenously. The modern method is to give this clot-provoking solution into the inferior or superior vena cava by a polythene tube passed from the long saphenous or cubital vein.

In order to discourage thrombosis, heparin (1,000 units per 500 ml.) is added to the infusion. Even so post-mortem examination has shown intravenous clotting in a high percentage of cases. For this reason the intravenous route should be employed only in cases of absolute necessity. Some advise passing the polythene tube but a few inches into a peripheral vein and to change the vein if clotting occurs. By gravitating the fluid in this way it is hoped that the menace of pulmonary embolism is lessened.

Vitamins.—Irrespective as to whether the alimentary or the intravenous route is employed, the daily vitamin intake should be identical with that of a patient subsisting wholly on parenteral feeding (pp. 94 to 99).

Prevention of Infection.—Anuric patients become infected easily, therefore all who come in contact with the patient should be gowned and masked. A nasal swab should be taken from the patient at the onset of anuria. If *Staphylococcus aureus* is cultured, a suitable antibiotic should be given at

Graham MacGregor Bull, Contemporary. Director, Medical Research Council, Clinical Research Centre, London.

once. Penicillin, 1 mega unit per twenty-four hours, can safely be given. Many antibiotics are cumulative even when given in greatly reduced doses. Tetracycline must be avoided because of its nephrotoxicity in poor renal function.

Watching for Hyperkalæmia.—Serial electrocardiographs are a better check to the development of this complication than reliance on plasma potassium determinations (p. 93).

Diuretic Phase.—In reversible lesions, usually about the eighth day, the epithelium of the lower nephrons regenerates sufficiently to prevent resorption of the glomerular filtrate, and a little urine is passed. This is a most hopeful sign, and given correct management and previously healthy kidneys (Causes 1 to 7, p. 1168) an increasing twenty-four-hourly output during the subsequent week is usual. When diuresis commences, an amount of water equal to the output of urine for the previous twenty-four hours is added to the daily allowance. It is more difficult to estimate an appropriate allowance of electrolytes, for the restitution of renal electrolyte-regulating function returns slowly. A heavy loss of sodium and potassium may occur and must be replaced. Estimation and quantitative replacement of the loss in the previous twenty-four hours is the most accurate way of management. Only when renal excretion reaches 1 litre per day does the blood-urea level commence to fall. Once diuresis exceeds 1 litre in twenty-four hours, the intragastric tube is removed and the patient is fed on a high calorie, low-protein diet containing an adequate daily amount of mineral salts.

Other methods of treatment in special circumstances:

1. **The Artificial Kidney.**—The principle of the artificial kidney is that of dialysis across a membrane which is permeable to crystalloids and not to colloids, with the patient's blood on one side of the membrane and a saline bath on the other; excessive crystalloids will leave the blood and enter the bath, which is constantly flowing. The indications for dialysis are: (*a*) Serum potassium above 7·0 mEq/litre (mmol/l), (*b*) Serum bicarbonate level below 12 m Eq/litre (mmol/l), (*c*) Gross overhydration, or (*d*) a blood urea above 300 mg/100 ml (65 mmol/l). Fits or coma may also be indications.

The artificial kidney requires a trained team to operate the machine, hence it is available only in a few centres. It is therefore necessary to describe other methods of ridding the patient of excessive electrolytes, particularly potassium.

2. **Peritoneal Dialysis.**—This method has been used with success. The simplest method and the one least likely to give rise to peritonitis is to insert a polythene tube into the peritoneal cavity by means of a trocar and cannula. Into the peritoneal cavity is gravitated moderately slowly a hypertonic solution (20% dextrose solution with 6 g sodium chloride per litre added). The fluid is retained by clipping the tube for two hours and then allowing it to flow out. The process is continued for up to forty-eight hours. Thus the peritoneum acts as a dialysing membrane.

3. **Ion Exchange Resins.**—If the patient does not vomit, then ion exchange resins in the sodium phase have been used successfully to withdraw potassium ions into the intestine in exchange for sodium ions. Five grammes of Resonium 'A', dissolved in the patient's allowance of dextrose solution, is given eight times a day. Calcium Resonium is often appropriate in uraemia as the serum calcium may be low. Rectal infusion of both resins is effective and most useful when the patient is vomiting.

Post-renal (*syn.* Obstructive) Anuria

1. **Calculus anuria** arises in one of the following ways:

a. A calculus becomes impacted in the ureter of the only functioning kidney, the other kidney being congenitally absent, previously removed, or destroyed by disease.

b. Both ureters become obstructed by stones or crystals.

Clinical Features.—Usually the patient is a man between forty and sixty years of age with a long history of urinary lithiasis, but in about 20 per cent. of cases calculous anuria arises without previous symptoms.

Onset.—Typically an attack of renal colic precedes the anuria, the onset of which is sudden. This pain may disappear and be replaced by a constant severe pain in the loin. In a few cases there is little, if any, pain, and the anuria is preceded by several days of increasing oliguria.

Stage of tolerance usually lasts from three to six days. The patient feels comparatively well and may continue his work. During this time the blood urea mounts steadily, and as it does so headache, sleeplessness, constipation, and lassitude supervene. By the end of this stage the blood urea is often over 200 mg/100 ml (33·2 mmol/l).

Stage of uræmia follows, and is characterised by the same clinical features described in late renal anuria.

Examination.—The obstructed kidney is tender and there is guarding by the overlying muscles. A large palpable kidney is probably functionless and the seat of a hydronephrosis or pyonephrosis; a recently obstructed kidney is never enlarged.

Action to be taken:

(1) *Pass a catheter* to exclude retention. A few ounces of blood-stained urine may be obtained.

(2) *Plain X-ray* may show the obstructing calculus, but more often visualisation is difficult. This is because the small size of the stone (it is seldom larger than an orange-pip), makes it difficult to see against superimposed bone and intestinal gas shadows. Sometimes a large branched renal calculus is displayed on the functioning side. Disparity in the size of the kidneys may be apparent.

FIG. 1311.—Stone impacted in the ureteric orifice as seen by cystoscopy.

(3) *Cystoscopy.* Pass a catheter up both ureters. This may pass or dislodge the stone and the emergency is over. Leave the catheter in position to drain, and when the general condition of the patient is satisfactory, remove the calculus by open uretero-lithotomy. (Just prior to this, further radiographs must be taken to confirm the site of the stone. An I.V.P. is permissible and helpful if the blood urea has fallen to within normal limits.) Occasionally at cystoscopy a stone is seen wedged in the ureteric orifice (fig. 1311) whereupon attempts

should be made to dislodge the stone with a ureteric catheter, or by meatotomy of the orifice.

(4) *Operation*—if a catheter will not pass. In this case the stone should be removed by open operation. If no stone is visualised by the X-ray, then the kidney on the side which was more painful should be explored. Pyelostomy, or nephrostomy (figs. 1312 and 1313) may be necessary. If the kidney explored is found to be hopelessly diseased, nephrostomy must be performed on the other side.

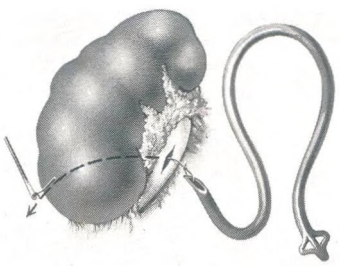


FIG. 1312.—Cabot's method of performing nephrostomy.

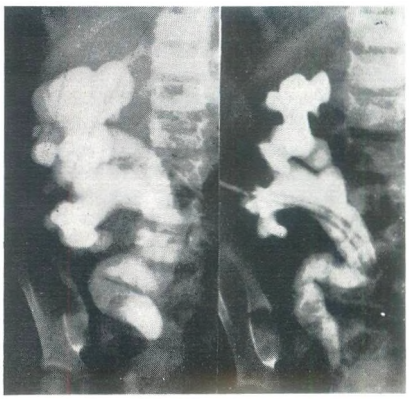


FIG. 1313.—Pyelograms before and after nephrostomy.

Through and Through, Loop, Ring, Nephrostomy (Tresidder).—A useful alternative to Cabot's method is to lead a single length of suitable tubing to the surface through the opening in the renal pelvis as well as through the kidney substance. Two holes are cut halfway along the tube where it is positioned in the pelvis. The ends of the tube are anchored to the surface or secured together and connected to the drainage bag. This is advantageous in affording (*a*) double drainage, (*b*) security against inadvertent removal, and (*c*) the continuous facility for replacement of the tube simply by railroading another into position.

(5) *When the obstruction has been relieved* either by ureteric catheterisation or operation, and until diuresis is established, the general treatment is similar to that described on p. 1168. In cases where nephrostomy has been necessary, preparations should be made for blood transfusion, for sometimes the hæmorrhage from the incised œdematous kidney is excessive.

2. **Anuria due to Sulphonamide Crystalluria.**—Sulphapyridine, sulphadiazine, and occasionally sulphathiazole in the presence of an acid medium are changed into acetyl salts which are insoluble. Crystals (fig. 1314) are deposited in the kidney tubules and ureters. The condition is now infrequent, and occurs mainly in patients with partial obstruction to the kidneys, and in those who are dehydrated.

FIG. 1314.—Acetylated sulphapyridine crystals resemble small wheat sheaves.

Treatment.—When a patient undergoing sulphonamide therapy develops renal colic and oliguria, the drug should be withheld and the high fluid intake further increased, and fruseamide 80 mg given i.v. Should anuria supervene there should be no delay in performing cystoscopy and attempting to catheterise the ureters. Provided the catheters can be inserted, the kidney pelves are washed out with 2·5% sodium bicarbonate solution. The crystalline mass can, on occasions, be

Hugh Cabot, 1872–1945. Professor of Surgery, Minnesota Graduate School of Medicine, Rochester, Minnesota, U.S.A.

Gerald Charles Tresidder, Contemporary. Senior Lecturer in Human Morphology, Southampton University, England.

dislodged with a stone dislodger (p. 1199). As an alternative to cystoscopic manœuvres, splanchnic block anæsthesia may succeed. If these measures are unsuccessful, unilateral or bilateral pyelostomy may be performed.

3. **Anuria due to Uric Acid Crystalluria** can occur in rapidly breaking-down neoplasms—more particularly lymphomas. The pelvis and ureters can become completely blocked by uric acid and urate crystals. Serum uric acid estimations should be performed on such cases having radiotherapy, and the urine should be kept alkaline. (The solubility of uric acid is vastly increased in a pH of 7–8.)

4. **Anuria due to Accidental Ligation of the Ureters.**—This is a hazard mainly of hysterectomy. Immediate exploration to divide the ligatures encircling the ureters should follow once the site of the obstruction has been determined by passing ureteric catheters. Pyelostomy should be reserved for cases unfit for this.

5. **Anuria due to Involvement of both Ureters in a Neoplastic Process, e.g. Carcinoma of the Cervix.**—Excluding terminal cases, the only treatment is transplantation of ureters into the bowel or bilateral nephrostomy.

6. **Anuria due to Involvement of Ureters in Retroperitoneal Fibrosis** (p. 1200).

CHAPTER 56

THE KIDNEYS AND URETERS

Embryology.—A bud from the lower end of the mesonephric (Wolffian) duct grows backwards behind the peritoneum to the sacral region (fig. 1315). The stalk of the bud forms the ureter and its dilated extremity the pelvis. Sometimes the bud is duplicated or the stalk becomes bifurcated, giving rise to congenital anomalies (see below). From six weeks to eight months, the primitive pelvis divides repeatedly to form generations of collecting tubules, the early (4th to 5th) generations ultimately forming the pelvis and calyces.

Later generations form the collecting tubules, and adjacent mesoblast produces a glomerular apparatus and nephric tubules which establish a continuity with the collecting system. It is failure of this junction which has been postulated as one cause for the development of polycystic kidneys. Between the fifth and eighth week of this development the kidney ascends on the posterior abdominal wall to reach its subcostal position. It also rotates so that the previously forward facing hilum faces medially.

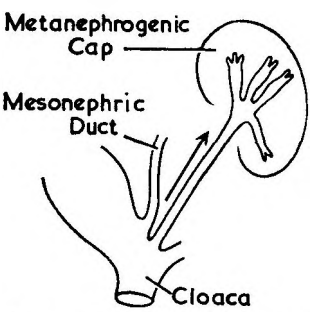


FIG. 1315.—The development of the kidney.

The fœtal kidney is at first lobulated, but in the human the lobules become welded together by the growth of a new cortex and the capsule. In some mammals, e.g. oxen and bears, fœtal lobulation is retained throughout life.

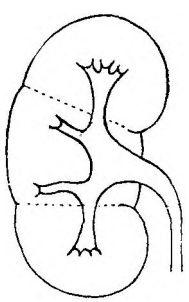


FIG. 1316.—The three divisions of the kidney render resection (partial nephrectomy) of the upper or lower division a practical proposition. *(After the late Carl Semb.)*

Surgical Anatomy.—The parenchyma of each kidney usually is drained by seven pairs of calyces, three in the upper and two each in the middle and lower segments (fig. 1316). Each of the three segments represents an anatomical and physiological unit with its own blood supply, an important point in view of the increased indications for partial nephrectomy (Carl Semb).

Surgical Physiology.—Ciné-pyelography indicates that there are sphincters at the junction of the minor and major calyces, and at the pelvi-ureteric junction. Each segment of the calyceal system fills before its contents are passed into the next chamber.

CONGENITAL ABNORMALITIES OF THE KIDNEY

Absence of One Kidney.—Sometimes pyelography reveals only one functioning kidney, and at cystoscopy only one ureteric orifice is present, alternatively a ureter and pelvis are present on the non-functioning side, but the parenchyma is almost or entirely absent. In either case the functioning kidney is hypertrophied. An absent or congenitally atrophic kidney is present in about 1:1,400 individuals.

Renal ectopia occurs once in 1,000 cases. The kidney is arrested in some part of its normal ascent, usually at the brim of the pelvis. As a rule the kidney of the opposite side is present and in its normal position. The left kidney is ectopic far more often than the right; the reason is unknown. Ectopia may present a diagnostic problem when acute disease develops in the ectopic kidney.

Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad, U.S.S.R.). Carl Boye Semb, 1895–1971 (see footnote, p. 748).

Horse-shoe Kidney.—The most medial subdivisions of the primary mesonephric bud of each side fuse and the kidneys fail to ascend completely. The adrenal glands, being developed separately, are in their normal positions. The abnormality (found once in every 1,000 necropsies) is more common in the male. Usually the bridge joining the lower poles lies in front of the fourth lumbar vertebra. Fusion occurs very early, when the embryo is but thirty to forty days old, at which time the two masses of mesoblast destined to form the kidneys lie very close together. Exceptionally, it is the upper poles that are fused.

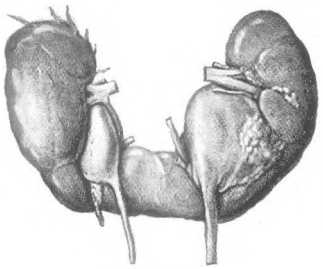


FIG. 1317.—Horse-shoe kidney. Note the ureters passing in front of the fused lower poles.

Clinical Features.—Horse-shoe kidneys are prone to become diseased, largely because the ureters are angulated as they pass over the fused isthmus (fig. 1317). This produces urinary stasis; consequently simple infection, tuberculosis, and calculus formation are common complications. Although a fixed mass below the umbilicus may suggest a horse-shoe kidney, the final diagnosis is established by pyelography. The most characteristic finding is that the lowest calyx on each side is reversed in position (i.e. directed towards the vertebral column). Rarely most, or all, of the calyces are reversed (fig. 1318). In a large percentage of cases the ureters curve like a flower vase. While horse-shoe kidney is not a contraindication to pregnancy, urinary complications are more frequent.

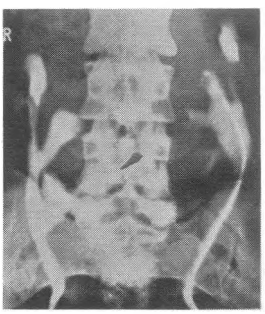


FIG. 1318.—Pyelogram of a horse-shoe kidney. Only rarely are all the calyces directed towards the spinal column. (*A. Jacobs, F.R.C.S., Glasgow.*)

Treatment.—In cases giving rise to pain, or when non-specific infection is present, division of the isthmus is usually curative. The operation can be conducted extraperitoneally, but a trans-peritoneal approach gives a much better view of the renal arteries and veins, and it is easier to decide whether a nephropexy is desirable. The area should be drained extraperitoneally. When one half of a horse-shoe kidney is irreparably diseased, e.g. calculi or neoplasm, heminephrectomy should be performed provided the remaining half has adequate function.

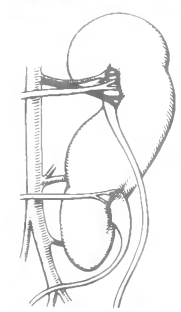


FIG. 1319.—Unilateral S-fusion of the kidneys.

Unilateral fusion (*syn.* crossed dystopia) is rare. Both kidneys are situated in one loin. One kidney, carrying its own blood-vessels and with the ureter opening into the bladder in a normal position, crosses the middle line, and its upper pole fuses with the lower pole of the normally placed kidney. In this instance both kidney pelves are situated one above the other medial to the renal parenchyma (unilateral long kidney). In some cases the pelvis of the crossed kidney faces laterally (unilateral S-shaped kidney (fig. 1319)) (See also fig. 1324).

Congenital Cystic Kidneys (*syn.* Polycystic Kidneys);—In 18 per cent. of cases there is a congenital cystic liver:

occasionally the pancreas and lung are affected similarly. The disease is hereditary and can be transmitted by either parent. This is important in relation to genetic counselling as the risk of transmitting the autosomal dominant trait is very high. The disease is not easily demonstrable on pyelography before the late 'teens' and rarely gives clinical manifestation until the fourth decade.

Pathology.—The organs may become enormously enlarged. The surface gives an appearance of many bubbles. On section the renal parenchyma is riddled with cysts of varying sizes, some containing clear fluid, others thick brown material, and still others coagulated blood.

Clinical Features in the Adult.—The condition is slightly more common in women than in men. There are six clinical features:

(*a*) *Renal Enlargement.*—The large knobby kidneys when discovered in the course of a routine examination can hardly be mistaken. Sometimes congenital cystic kidneys are revealed only at laparotomy for some other condition. *Unilateral Renal Swelling.*—One kidney contains larger cysts than the other, and the physical signs are similar to those of a renal neoplasm.

Patients with congenital cystic kidneys pass abundant urine of low specific gravity (1·010 or less) containing a slight trace of albumin but neither casts nor cells. On cystoscopy there is often considerably delay in the excretion of indigo-carmine, even in cases with a normal or only slightly elevated blood urea.

(*b*) *Pain* is due either to the weight of the organ dragging upon its pedicle or to tension within the cysts. Often the pain is a dull ache in the loin; sometimes it takes the form of renal colic, when a calculus, which is not rare in this condition, should be suspected.

(*c*) *Hæmaturia.*—In about 25 per cent. of cases, as a result of over-distension, one of the cysts ruptures into the renal pelvis and causes hæmaturia. Usually moderate hæmaturia lasts for a few days, and recurs at varying intervals; sometimes it is profuse.

(*d*) *Infection.*—The most common complication of congenital cystic kidneys in adult life is pyelonephritis.

(*e*) *Hypertension.*—About 75 per cent. of patients with congenital cystic kidneys above the age of twenty have hypertension. Why some escape this complication is not clear. Possibly the high blood pressure is due to a separate genetic factor frequently linked with congenital cystic kidneys.

(*f*) *Urœmia.*—The patient complains of anorexia, headache, and indefinite gastric symptoms, and is frequently given a placebo, until persistent symptoms demand a complete clinical examination. Later drowsiness and vomiting occur. Signs of uræmia often commence suddenly during middle life; only one quarter of patients with this condition survive the age of fifty-five years. Severe anæmia is common.

Pyelography.—Excretory pyelography is the best way of confirming the diagnosis, but when the concentration of Hypaque is insufficient to cast a clear shadow, retrograde pyelography is necessary. Only one side should be injected, the other being deferred for a week lest œdema further impairs

renal function. The shadows of the kidneys are enlarged in all directions. The renal pelvis is elongated and may be compressed. The calyces are stretched over the cysts and are often narrow (like the legs of a spider) or bell-like (fig. 1320).

FIG. 1320.—Polycystic kidney. Pyelographic appearance. Note length of kidney and elongated bell-like calyces stretched over cysts.

Treatment :

(*a*) *Expectant.*—Routinely the patient should drink a large quantity of water, have a low-protein diet, and take iron to prevent anæmia. Infection, when it occurs, should be treated by an appropriate drug.

(*b*) *Operative.*—By relieving pressure on the remaining renal parenchyma, Rovsing's operation, if performed early, improves renal function. In many instances, life is prolonged five years or more.

(*c*) *Renal Failure* should be treated by dialysis, possibly bilateral nephrectomy and kidney transplantation.

Rovsing's Operation.—One kidney and then the other is exposed. The cysts are incised with a narrow-bladed scalpel, first on the convex border, then on the posterior surface and, finally, as the kidney becomes more manageable in size, the peritoneum is peeled away and the anterior surface is dealt with similarly.

So improved by bilateral Rovsing's operation was one of our patients who was admitted in a state of uræmia, that two and a half years later he was serving in the Life Guards! Rovsing's operation should be performed, not as a last resort, but as soon as possible after the condition is recognised.

Infantile Polycystic Disease is an unrelated condition—autosomal recessive—and much rarer. The kidneys are large, may obstruct birth and many cases are stillborn. Most others die in early life from renal failure.

Unilateral Multicystic Disease is much commoner. It presents as a mass in the flank. No function is detectable on pyelography, and exploration and removal is the treatment of choice. In the differential diagnosis, Wilms's tumour (p. 1215), neuroblastoma and hydronephrosis are all rarer conditions.

Solitary Renal Cyst.—While the term 'solitary' serves to distinguish the condition from congenital cystic disease of the kidneys, it is found that not infrequently one or two similar but smaller cysts are also present.

Ætiology.—The origin of the cyst may be identical (but on a smaller scale) with that of congenital cystic kidney (p. 1176); or it could arise from bygone trauma or infection causing blockage of a tubule. The mature age at which the cyst is usually found is in favour of the latter supposition.

Clinical Features.—There are no pathognomonic symptoms or signs. Sometimes there are no symptoms until a swelling is noticed, at others there is a dull ache in the loin. The cyst can become infected and if a swelling is palpable, and particularly if the swelling is tender, the creditable diagnosis of pyonephrosis will be made. A spontaneous hæmorrhage into the cyst occurs from time to time, in which case sudden renal pain is likely to be experienced. Occasionally the cyst presses on the pelvi-ureteric junction and causes urinary symptoms.

Radiology.—(*a*) Pyelography reveals a filling defect with one of more calyces stretched over it, simulating the deformity produced by a neoplasm, but typically more rounded. (*b*) It is possible to distinguish a renal cyst from a renal neoplasm by selective renal arteriography (fig. 1306) or by puncture percutaneously, or by an ultrasound scan.

Nils Thorkild Rovsing, 1862–1927. Professor of Surgery, Copenhagen.

Treatment.—Exploration should be advised. Renal cysts can be caused by a tumour blocking tubules and interfering with the vascular supply to the same area of the kidney. Having displayed the kidney the operation is varied according to circumstances.

(*a*) *Kirwin's Method.*—When the cyst is blue-domed and aspiration reveals clear fluid, that portion of the cyst wall lying above the surface is excised. The cavity is filled with perinephric fat and after securing hæmostasis of the cut edges, a corrugated drain is left down to the area, the fascia of Gerota closed over it, and the wound closed in layers.

(*b*) *Partial nephrectomy* is performed if the contents of the cyst are blood-stained (one-third of such cysts contain a papilliferous neoplasm).

(*c*) *Nephrectomy* is performed if there are indications that the cyst is a malignant degeneration of a neoplasm, e.g. irregular induration at the periphery of the cyst.

Differential Diagnosis.—In sheep-raising districts, *hydatid cyst of the kidney* is common. On the right side (if hydatid disease is suspected) the swelling is liable to be mistaken for a hydatid cyst of the liver. Occasionally the patient complains of passing 'grape skins' (ruptured daughter cysts) in the urine. The treatment is excision of the cyst, but in many cases the cyst is so large that nephrectomy is advisable.

Aberrant renal vessels are found on the left side more frequently than on the right; in females more often than males; unilateral examples are three times more common than bilateral.

Aberrant renal vessels can cause hydronephrosis (p. 1185 and fig. 1339); more frequently, however, aberrant vessels accentuate a hydronephrosis present on account of another reason (fig. 1336).

Usually the aberrant artery is small; occasionally it is comparatively large, supplying one-quarter of the renal parenchyma. Division will cause infarction of the corresponding portion of renal tissue. On the other hand, an obstructing aberrant renal *vein* can be divided with impunity, because the venous collateral circulation is very generous.

CONGENITAL ABNORMALITIES OF THE RENAL PELVIS AND URETER

Duplication of a renal pelvis is the most common congenital anomaly of the upper renal tract and is found in about 4 per cent. of patients. It is usually unilateral, and is somewhat more common on the left side than on the right. The upper renal pelvis is comparatively small (fig. 1321) and drains the upper group of calyces; the larger lower renal pelvis drains the middle and lower groups of calyces.

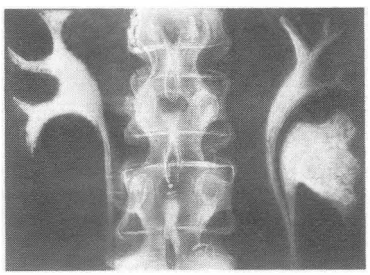


FIG. 1321.—Pyelogram showing a kidney with a double pelvis.

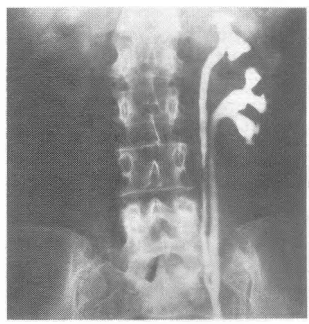


FIG. 1322.—Double ureter. Retrograde pyelogram.

Thomas Joseph Kirwin, 1889–1959. Director of Department of Urology, Fourth and Fifth Avenue Hospitals, New York, U.S.A.
Dumitra Gerota, 1867–1939. Professor of Surgery, Bucharest, Rumania.

Reno-renal reflux, from one segment of a duplex kidney to the other may be a cause of pain. It is usually associated with pyelonephritic changes in the segment affected by the reflux.

Duplication of a Ureter.—Double ureters are present in addition to double renal pelves in about 3 per cent. of cases submitted to pyelography. The ureters often join, usually in the lower third of their course (fig. 1322), and have a common orifice into the bladder. Less frequently the ureters open independently into the bladder (fig. 1323), in which case the ureter draining the upper pelvis crosses its fellow, and opens below and medial to it.

Clinical Features.—In many instances a double renal pelvis or a double ureter is found by chance in the course of an investigation of the urinary organs. A double pelvis is more liable to become the seat of infection, calculus formation, or hydro-

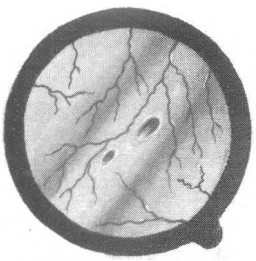


FIG. 1323. — Complete duplication of the left ureter as seen through the cystoscope.

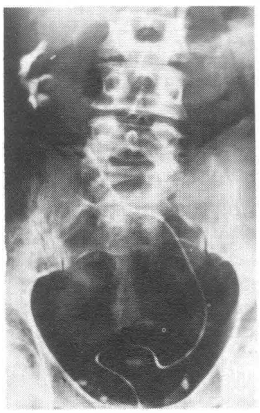


FIG. 1324.—Crossed dystopia. The ureteric orifices are situated normally. *(R. K. Garg, F.R.C.S., Bombay.)*

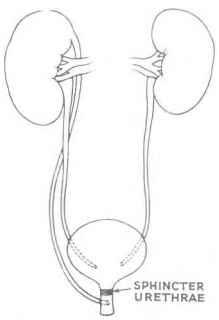


FIG. 1325.—Ectopic ureter in a female.

nephrosis than a normal pelvis. Initially such disease is confined to one part of the duplication. In cases of complete duplication of the ureter the lower ureteric orifice is sometimes the site of congenital atresia.

Treatment.—Ureteric meatotomy can be performed in some early cases of stricture of a ureteric meatus. Difficulty is encountered in cases of Y-shaped bifid ureter, because a ureteric catheter cannot be made to enter the smaller orifice. Hemine-phrectomy with removal of its ureter is eminently satisfactory when disease normally requiring total nephrectomy is confined strictly to one half of a kidney that has a double pelvis. The exception to the rule is, of course, a neoplasm.

Ectopic Ureteric Orifice.—Should a second ureteric bud arise from the meso-nephric duct later than usual, the orifice of the accessory ureter is prone to occupy a grossly abnormal position. This is a rare anomaly. In both sexes the existence of double ureter is determined by excretory pyelography.

In the female an ectopic ureter opens either into the urethra below the sphincter urethræ (fig. 1325) or into the vagina, and causes an intractable incontinence of urine. The diagnosis can nearly always be made from the history alone. A girl or woman who has dribbled for as long as she can remember, despite the fact that she has a desire to void, and does urinate, has an ectopic ureteric orifice. The demonstration of this orifice is often extremely difficult, because it is guarded by a valve. The diagnosis may be established by giving an intravenous injection of indigo-carmine, and placing one swab into the vestibule and another in the vagina; the one coloured

blue signifies the position of the ectopic orifice. Sometimes laparotomy is the only way of finding or excluding the additional ureter.

In the male the aberrant opening may be situated at the apex of the trigone, in the posterior urethra, in a seminal vesicle, or in an ejaculatory duct. As in all these diverse situations, the ectopic ureteric orifice is situated above the sphincter urethræ, the male patient is continent, but recurrent acute or chronic pyelonephritis is apt to supervene in the renal tissue served by the ectopic ureter. Posterior urethroscopy, after the intravenous injection of indigo-carmine, may be of help in demonstrating that a male patient has an ectopic ureter.

Treatment.—*In the female* excision of that segment of the kidney served by the ectopic ureter is usually advisable, because it is so often hydronephrotic and chronically infected. In the rare event of the urine draining from it being sterile, the ectopic ureter can be implanted into the bladder, or joined to its fellow.

In the male—Often no treatment is required. Should urinary infection supervene hemi-nephrectomy is indicated.

Congenital mega-ureter may be unilateral (fig. 1326) or bilateral, and in late stages is accompanied by hydronephrosis. Commoner in males the condition is often symptomless until infection has occurred. If, however, the patient experiences pain, a diagnosis can be made before the onset of infection. The lower end of the ureter and orifice appear normal but constitute an obstruction. Reflux is not present; a ureteric catheter passes easily. Vigorous peristaltic activity can be seen in the dilated ureter under the image intensifier, but efflux is small, while marked delay in emptying of the pelvis and ureter is seen in late films many hours after.

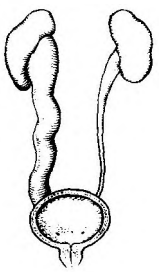


FIG. 1326.—Unilateral congenital mega-ureter.

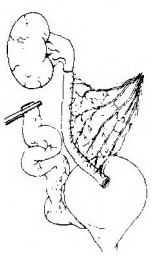


FIG. 1327.—Substitution of a segment of ileum for the mega-ureter, which is shown attached to the bladder.

Treatment.—In infected cases, percystoscopic meatotomy and drainage of the dilated ureter with a ureteric catheter for about six weeks has been found to be effective in clearing up the infection. Reflux afterwards is likely however, and the condition is better treated by initial nephrostomy and then when the infection is completely controlled by open excision of the lower ureter, followed by excision of excessive length of tortuous ureter, refastening the end by wedge excision to narrow the lumen, and tunnel re-implantation to prevent reflux. When the renal parenchyma is severely damaged, nephro-ureterectomy is curative. In bilateral cases where function is diminishing, cutaneous ureterostomy should be considered.

Post-caval Ureter.—The right ureter passes behind the inferior vena cava (fig. 1328) instead of lying to the right of it. This may cause obstructive symptoms. Should these symptoms warrant it, the ureter can be divided in the dilated portion and reanastomosed in front of the cava. Occasionally the retrocaval portion of the ureter is fibrotic and must be excised. A long oblique anastomosis should be made of the normal lower ureter to the dilated upper portion.

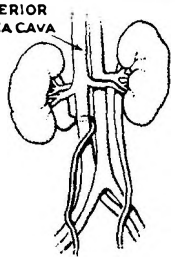


FIG. 1328.—Post-caval ureter.

Ureterocele is due to congenital atresia of a ureteric orifice which causes a cystic enlargement of the intramural portion of the ureter. Usually the wall of the cyst is composed of mucous membrane only; infrequently the muscle coat is included. This condition may be discovered in childhood; often it is not recognised until adult life. Women are more often affected than men, and in 10 per cent. of cases the condition is bilateral. The condition, producing some obstruction, does render the patient more liable to stone formation and recurrent infection. The cystoscopic findings are characteristic: when the wall consists of mucous membrane there is a translucent cyst over which blood-vessels radiate (fig. 1329); in the rare variety

FIG. 1329.—Right-sided ureterocele. (*After J. C. Ainsworth-Davies, F.R.C.S., London.*)

containing muscle in the wall, the cyst is opaque. In either case the sac is seen to enlarge rapidly with each efflux of urine, and then slowly to collapse. The pyelographic findings are characteristic (fig. 1330).

Treatment.—Many cases are symptomless and no treatment is required. Hydronephrosis, stone, or recurrent infection may necessitate treatment. Cauterising a hole through the wall of the cyst with a diathermy electrode is a simple and satisfactory procedure. A micturating cystogram should be performed three months later to exclude urinary reflux. In advanced unilateral cases associated with a hydro- or pyonephrosis and dilatation of the whole of the ureter, nephrectomy is needed.

INJURIES TO THE KIDNEY

In civil life blows or falls upon the loin are the most fruitful sources of such injuries, while blows from in front and crushing accidents add their quota (road traffic accidents). Hæmaturia following minor injuries should suggest the possibility of pre-existing renal abnormality, e.g. stone, hydronephrosis, or tuberculosis.

The degree of injury varies considerably from a small subcapsular hæmatoma to a complete tear involving the whole thickness of the kidney (fig. 1331); in addition, the kidney may be partially or wholly avulsed from its pedicle. Tears of the renal parenchyma follow the lines of the uriniferous tubules. The whole of one pole may be detached.

The injury is extraperitoneal, except very occasionally in children in whom there is little perinephric fat; in these cases the peritoneum, being intimately related to the kidney, is liable to be torn in addition to the renal capsule, allowing blood and, perhaps, urine to escape into the peritoneal cavity.

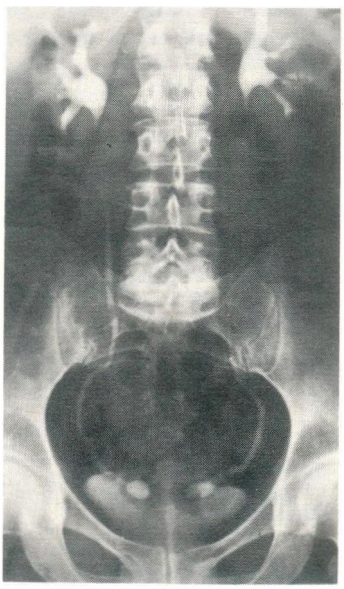


FIG. 1330.—Intravenous pyelographic appearance of ureterocele. Typical adder-head appearance of lower ureters which remain filled with Hypaque (diatrizoate).

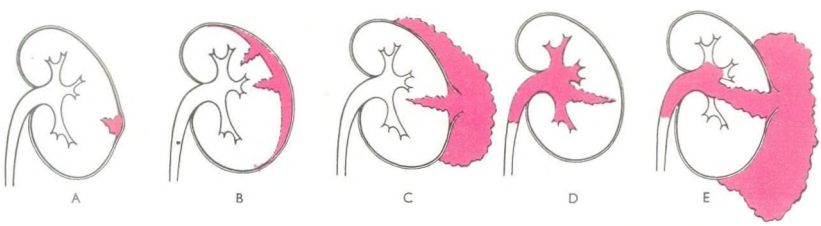


FIG. 1331.—Various types of renal injuries: A, small subcapsular hæmorrhage; B, large subcapsular hæmorrhage; C, cortical laceration with perinephric hæmatoma; D, medullary laceration with bleeding into the renal pelvis; E, complete rupture. (*After P. Adams.*)

Clinical Features.—Rarely is there superficial bruising, but there is likely to be local pain and tenderness.

Hæmaturia is a cardinal sign of a damaged kidney, but it may not make its appearance until some hours after the accident. If the hæmorrhage is profuse, it may be followed by clot colic.

Severe Delayed Hæmaturia.—Sudden profuse hæmaturia can occur between the third day and the third week after the accident in a patient who appears to be progressing favourably. It is due to a clot becoming dislodged.

Meteorism.—In many cases of renal injury, abdominal distension comes on about twenty-four to forty-eight hours after the accident. In all probability it is caused by a retroperitoneal hæmatoma implicating splanchnic nerves.

A perinephric hæmatoma should be suspected if there is even a slight flattening of the normal contour of the loin (fig. 1332), always provided there is no scoliosis.

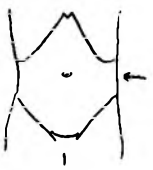


FIG. 1332.

Management and Treatment

Conservative treatment is usually successful and must be instituted at once. However, it must be remembered that injury to other organs may be associated with injury to the kidney.

1. The patient must rest flat in bed until macroscopic hæmaturia has been absent for one week.
2. Morphine should be given for pain, and as a sedative.
3. Hourly pulse and blood-pressure chart must be kept.
4. Give a sulphonamide or antibiotic to prevent infection of the hæmatoma.
5. Save a sample of each specimen of urine passed.
6. Group and cross-match blood. A transfusion may be required for shock or continuing hæmorrhage.
7. An intravenous urogram (I.V.P.) should be obtained urgently to show that the other kidney is normal. A leak of urine on the damaged side is often demonstrated.

Under this regimen the hæmaturia should diminish, pain and guarding decrease, and pulse and colour of the patient improve.

Exploration is necessary in 20 to 25 per cent. and is undertaken if : 1. There are signs of progressive blood loss. 2. A swelling develops in the loin. 3. Signs of peri-renal infection develop. The aim is to conserve the kidney or part of it if possible.

In all cases the peritoneum should be opened to exclude damage to other organs.

Should the necessity for an urgent operation arise and facilities for pyelography not be available, the presence of a functioning kidney on the contralateral side can often be confirmed by chromo-cystoscopy (p. 1163). When the kidney is found to be ruptured in several places, or the kidney pedicle is damaged, nephrectomy must be undertaken. Small tears can be sutured over Oxycel or a piece of detached muscle, either of which promotes hæmostasis. Larger single rents in the middle of the kidney are best dealt with by performing nephrostomy through the rent and suturing the kidney on either side of

the tube. If laceration is confined to one pole of the kidney, partial nephrectomy may be practicable.

When a sole existing kidney is sufficiently damaged to necessitate exploration, it must be repaired. Failing this, the wound is packed firmly with gauze in the hope that not only will the bleeding be controlled but that the ruptured kidney may heal.

Multiple Injuries.—Simultaneous splenectomy and left nephrectomy have been carried out successfully when both organs have been ruptured. The mortality of cases of rupture of the kidney with damage to the liver or hollow organs, often complicated by fractured ribs, pelvis, or vertebræ, is high.

FIG. 1333.—A pararenal pseudo-hydronephrosis.

Complications.—1. Clot retention in the bladder can be cleared with a plastic catheter and a glass syringe.

2. Pararenal pseudo-hydronephrosis (fig. 1333) may occur in the course of a few weeks, due to a combination of a complete tear of the renal cortex and ureteric obstruction.

3. Hypertension may follow fibrosis of the kidney, usually after three months or more. Nephrectomy will be necessary.

4. **Aneurysm of a renal artery** (fig. 1334) is a rare complication of injury to a kidney. The most frequent symptom is pain in the renal region, and, when the aneurysm is large, a non-tender swelling will be felt. Only occasionally is it pulsatile, and a bruit may be heard. An X-ray may show a shadow in relation to the pelvis of the kidney. An I.V.P. demonstrates that the swelling is extrarenal with the renal pelvis displaced laterally. Aortography will confirm the diagnosis. Attacks of hæmaturia occur, and are probably due to congestion of the renal parenchyma. The aneurysm may rupture into the peritoneal cavity or into the perirenal tissues, with fatal results.

Treatment by nephrectomy and excision of the aneurysm is usually required. In a few cases a saccular aneurysm has been resected with preservation of the renal vessels and the kidney.

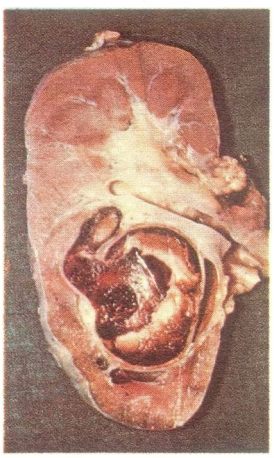


FIG. 1334.—Aneurysm of the renal artery containing laminated thrombus.

INJURIES TO THE URETER

Rupture of a ureter can occur as the result of an accident causing hyperextension of the spine. The clinical diagnosis is impossible until a diminished quantity of urine is passed, followed by a swelling in the loin or iliac fossa. An I.V.P. shows a diffuse shadow below the kidney on the injured side.

Injury of one or both ureters during a pelvic operation is considerably more common than the foregoing and occurs most often during the course of a difficult hysterectomy. Preliminary catheterisation of the ureters prevents such accidents, for with catheters within them, the ureters can be felt and seen unmistakably. The accidents include division of the ureter, ligating it, crushing it in forceps or removing a portion of its wall.

Injury recognised at the Time of the Operation.—The uretero-vesical continuity should be restored by one of the methods described below, unless the patient's condition is poor, when ligation of the proximal end of the ureter is the best course. If the patient rallies within two days, temporary nephrostomy is carried out, and a reparative operation undertaken later.

Injury not recognised at the Time of the Operation:

(*a*) **Unilateral Injury.**—There are three possibilities:

1. *No Symptoms.*—If one ureter is ligated securely with unabsorbable material and the contralateral kidney and ureter are normal, in 68 per cent. of cases the kidney on

the obstructed side undergoes silent atrophy. Thus the accident remains unsuspected unless the patient, some time later, undergoes a urological examination.

2. *Development of loin pain and a fever*, possibly proceeding to a pyonephrosis, occurs in less than a half of the remainder. Excretory pyelography reveals no excretion on the side of the lesion. If this is discovered early, and the patient is fit, re-exploration to free the ureter is best: alternatively, nephrostomy, and later repair of the ureter.

3. *A urinary fistula develops* through the abdominal incision or, following hysterectomy, through the vagina. Again, a temporary nephrostomy may be performed, and a reparative operation postponed until œdema and infection have abated after an interval of about six weeks.

(*b*) **Bilateral Injury.**—In cases of anuria following ligation of both ureters during hysterectomy an attempt is made to pass ureteric catheters, when both ureters will be found to be occluded. The wound should be explored and ligatures removed.

Repair of the Injured Ureter

1. If the ends are clean cut and no length is lost, each end should be split for 1·5 cm and the edges united as a flat ribbon (fig. 1335). A soft latex T-tube is left with one limb across the gap for two weeks. This is to be preferred to end-to-end suture which frequently results in stricture formation.

2. In division low down, it may be possible to reimplant the ureter into the bladder.

3. *Boari's Operation.*—A flap of bladder is turned up to replace the lower ureter.

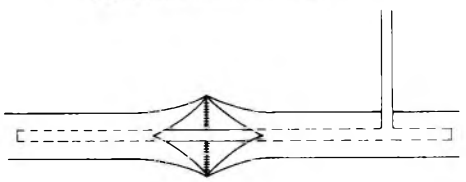


FIG. 1335.—Repair of the ureter using a T-tube.

4. Occasionally, where conservation of all renal tissue is vital, replacement by a segment of ileum is necessary.

Nephrectomy may be the best course, for example, in the elderly patient with a malignant condition, the other kidney being normal.

HYDRONEPHROSIS

A hydronephrosis is an aseptic dilatation of the whole or part of the kidney due to a partial or intermittent obstruction to the outflow of urine.

A hydronephrosis may be unilateral or bilateral.

Unilateral hydronephrosis is due to some form of *ureteral* obstruction, often at the pelvi-ureteric junction; any length of ureter above the obstruction is dilated also. The causes are:

Extramural.—1. 'Aberrant Vessels'. In a number of cases of pelvic hydronephrosis a lower polar artery or vein arising from the main vessels in an aberrant position obstructs the upper ureter where it crosses in front of it. The pyelographic appearance is shown in fig. 1339 (*b*). In many more instances this is not an aberrant vessel, but a normal inferior renal artery or vein that has been displaced downwards by the enlarged renal pelvis. This displaced vessel, while adding considerably to the rapid increase in the size of the hydronephrosis, is not the cause thereof (fig. 1336).

2. Involvement of ureter by growth outside it, e.g. carcinoma of the cervix, prostate, rectum, colon, or cæcum.

3. Idiopathic retroperitoneal fibrosis (p. 1200).

Intermural.—1. Congenital stenosis, physiological narrowing, or achalasia at the pelvi-ureteric junction.

2. Ureterocœle and congenital small ureteric orifice.

Achille Boari. Nineteenth-century Italian surgeon.

3. Inflammatory stricture of the ureter following removal of a stone that was lodged in one portion of the ureter for a considerable time, or tuberculosis of the ureter. Stricture of the ureter may follow uretero-ureteric anastomosis or trauma to the ureter during a pelvic operation.

4. Neoplasm of a ureter, or the bladder involving a ureteric orifice.

Intramural.—A calculus in the ureter or small calculus in the renal pelvis. The latter often gives rise to intermittent hydronephrosis.

Bilateral hydronephrosis is generally the result of some form of *urethral* obstruction, but it can also be caused by one of the lesions described above occurring on both sides.

When due to lower urinary obstruction, the cause may be:

(*a*) *Congenital:*

1. Congenital stricture of the urethral meatus or, very rarely, phimosis.

2. Congenital valves of the posterior urethra, or congenital contracture of the bladder neck.

(*b*) *Acquired:*

1. A neoplasm of the bladder which involves both ureteric orifices.

2. Prostatic enlargement or carcinoma; acquired contracture of the bladder neck.

3. Carcinoma of the cervix (occasionally carcinoma of the rectum), involving both ureters.

4. Inflammatory or traumatic urethral stricture. Phimosis.

When the obstruction lies in the urethra, muscular hypertrophy of the bladder occurs, and the ureters, by reason of this hypertrophy, become partially obstructed in the intramural portion of their course.

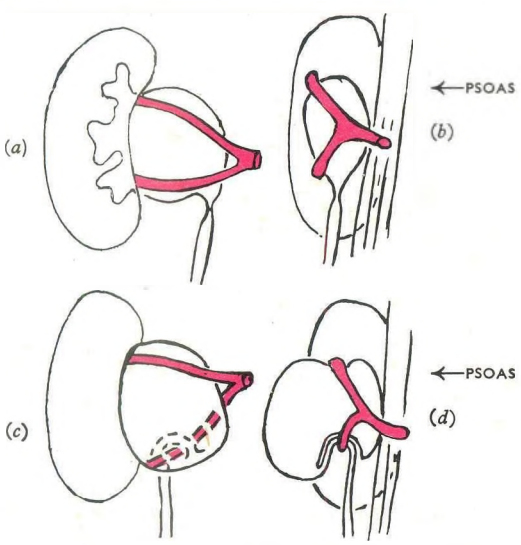


FIG. 1336.—'Congenital' hydronephrosis with obstruction at pelvi-ureteric junction. (*a*) Initial arrangement. (*b*) Lateral view of the hilum. (*c*) and (*d*) show pelvis of kidney prolapsing forward between branches of the renal artery.

Pathology. — The pathological changes differ in some respects according to whether the kidney has an extrarenal or an intrarenal pelvis (fig. 1337), the former

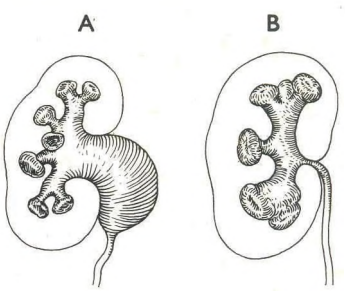


FIG. 1337. — Hydronephrosis occurring in an (A) Extrarenal, (B) Intrarenal pelvis of the kidney, the former being more liable to be obstructed by a blood-vessel.

being much more common. In a kidney with an extrarenal pelvis, at first the pelvis alone becomes dilated (pelvic hydronephrosis) (fig. 1336). As time goes on, if the obstruction is not relieved the calyces become increasingly dilated and the renal parenchyma is progressively destroyed by pressure atrophy. In a kidney with an intrarenal pelvis the destruction of the parenchyma occurs more rapidly. In either case, it may be a matter of years before the diagnosis is made, by which time the kidney may be merely a lobulated sac containing pale uriniferous fluid of low specific gravity. On bisecting the specimen longitudinally a series of compartments representing the dilated calyces will be found. Rarely is the renal parenchyma destroyed in its entirety, although in advanced cases the amount remaining is very small.

CLINICAL FEATURES

Unilateral Hydronephrosis

The female/male ratio is 2:1; the right side is more commonly affected. Presenting features include:

1. *Insidious.*—Pain is slight; often there is only a dull ache or a sense of weight in the loin which may be brought on by alcohol or excessive fluid intake. On examination an enlarged, sometimes a greatly enlarged, kidney is found. In these cases the obstruction is usually at the pelvi-ureteric junction.

2. *Attacks of renal colic* occur. Often there is no palpable swelling.

3. *Intermittent Hydronephrosis.*—After an attack of renal colic with its accompanying abdominal rigidity passes off, a swelling in the loin is found. Some hours later, or on the next day, following the passage of a large quantity of urine due also to some reflex polyuria, the pain is relieved and the swelling disappears (Dietl's crisis).

Bilateral Hydronephrosis

1. *Due to Lower Urinary Obstruction.*—There is little to call attention to the hydronephrosis except, perhaps, a dull ache in the loins. The symptoms are overshadowed by those of the causative lesion (above). Symptoms of uræmia—thirst, loss of appetite are commoner than loin pain. There is often polyuria of low specific gravity containing a small quantity of albumin. The kidneys are unlikely to be palpable because before the hydronephroses become large enough to be felt, renal failure will have occurred.

2. *Due to Bilateral Upper Urinary Obstruction.*—This is rare compared with a unilateral lesion. Symptoms may be bilateral, but more frequently are on one side only, an I.V.P. revealing hydronephrosis on the other side.

3. *Due to Pregnancy.*—Dilatation of the ureters and renal pelves occurs in every pregnancy. Both renal pelves and ureters are affected, but the right side to a much greater extent than the left. The dilatation commences during the first few weeks of gestation, and reaches its zenith between the fifth and sixth months. It then remains stationary until after delivery, when involution occurs within two to twelve weeks. The condition is due to atony of the ureteric musculature brought about by the hormone progesterone. Possibly pressure of the fœtal head on the ureters plays some part during

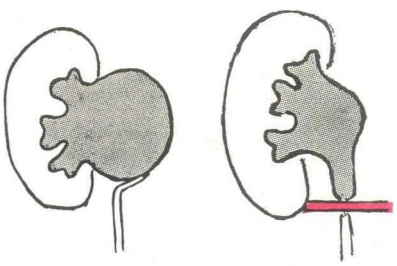
Joseph Dietl, 1804–1878. Professor of Pathology and Therapeutics, Cracow. In his later years he achieved fame as a politician.

the last months of pregnancy. The main importance of the condition is an increased liability to infection (p. 1202). Exceptionally the hydronephrosis persists.

Pyelography (I.V.P.).—Except during pregnancy, when X-rays are contraindicated for fear of causing a congenital malformation, the diagnosis is made by *excretory pyelography*. When the hydronephrosis is advanced, films taken six to twenty-four hours after the injection of the medium may show a hydronephrotic kidney which was imperceptible on films taken at the routine times. As a rule the earliest pyelographic manifestation of a hydronephrosis is dilatation of the renal pelvis, for in the majority of cases the pelvis is extrarenal. The next change is decreasing concavity and then flattening of the minor calyces and dilatation of the major calyces, which are

FIG. 1338.—The progress from normal to clubbing of the minor calyces in hydronephrosis, as seen radiologically. (*After J. B. Macalpine.*)

the first changes when the pelvis is intrarenal. In moderate-sized hydronephroses the minor calyces become convex and finally club-shaped (fig. 1338) and the major calyces considerably broadened. In advanced cases there is extreme dilatation of the renal pelvis and the calyces (fig. 1339), or no excretion of opaque media. Doubtful cases may be clarified on occasion by inducing a diuresis during excretion pyelography which may 'blow up' a hydronephrosis. *Retrograde pyelography* (fig. 1340) is required only when the results of excretory pyelography are inconclusive. Delay in emptying may be shown by a film taken ten minutes after the pelvis has been filled retrogradely; by this time a normal pelvis should have emptied.



(*a*) (*b*)

FIG. 1339.—(*a*) Pyelographic appearance in 'congenital' hydronephrosis with obstruction at the pelvi-ureteric junction. (*b*) Pyelographic appearance in obstruction due to a polar vessel.

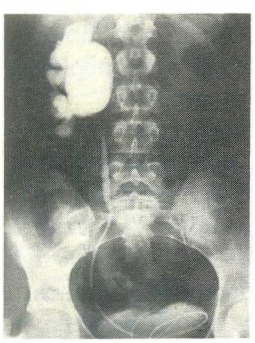


FIG. 1340.—Retrograde pyelogram of a hydronephrosis showing a greatly enlarged pelvis and dilated calyces.

The indications for operation are bouts of renal pain, increasing dilatation of the renal pelvis and calyces, or evidence of infection.

Treatment.—*Conservation* of renal tissue is the aim. Nephrectomy should only be performed when the renal parenchyma has been largely destroyed. Mild bilateral cases with few symptoms should be followed up, but only operated upon if dilatation is increasing.

Careful freeing of the ureters (ureterolysis) is an essential preliminary to specific procedures:

Ligation and division of an obstructing blood-vessel, together with clearance of adhesions in the neigh-

bourhood of the pelvi-ureteric junction, gives a lasting cure in the few cases due to this cause. The objection to dividing the obstructing artery is that ischæmic infarction ensues in the portion of kidney supplied.

Reconstruction of a hydronephrosis is employed in cases of advanced pelvic hydronephrosis providing a reasonable thickness of renal parenchyma remains. The Anderson-Hynes operation (fig. 1341) is probably the best. The affected kidney is displayed and the upper third of the ureter and the renal pelvis are mobilised. A renal vein overlying the pelvi-ureteric junction can be divided, but an artery in this situation, whether aberrant or not, should be preserved. The anastomosis is made in front of such an artery. The upper end of the ureter is divided a short distance below the pelvi-ureteric junction, or any narrow segment of ureter. The redundant part of the renal pelvis, together with the ureteropelvic junction is resected as shown in fig. 1341 A, a

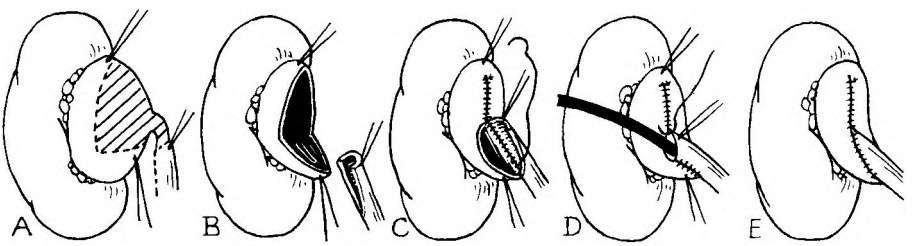


FIG. 1341.—The Anderson-Hynes plastic reconstruction of a hydronephrosis. Note the placement of the stay sutures.

flap of the lower part of the pelvis being retained. The upper two-thirds of the opening thus created in the renal pelvis is closed with a 0000 plain catgut suture traversing all coats (fig. 1341 B). The upper end of the ureter is slit for 2–3 cm and its anterior edge is anastomosed to the anterior edge of the pelvic flap by a continuous suture of 0000 plain catgut (fig. 1341 C). A ureteric catheter is passed into the ureter for a short distance; this is merely a temporary expedient to keep the deep sutured layer out of the way while the superficial edges are drawn together (fig. 1341 D) in the same manner as were the deep. The catheter is withdrawn just before the anastomosis is completed.

Intubated Ureterostomy (Davis).—Longitudinal incision of a narrowed segment of ureter, leaving a T-tube across the split to allow regrowth round the tube is a simple and effective treatment when the pelvis is not greatly enlarged.

RENAL CALCULUS

Ætiology.—The subject is a complicated one, and the following is an epitome of current opinion. (Compare with the ætiology of gallstones, p. 916.)

1. **Dietetic.**—A deficiency of vitamin A causes a desquamation of epithelium. The cells form a nidus around which the stone is deposited. From a study of economic conditions in districts where urinary calculi are common, it is evident that the inhabitants suffer from dietetic imbalance. This is more applicable to bladder calculi.

2. **Altered Urinary Solutes and Colloids.**—In hot climates the concentration of solutes will rise. It has been postulated that any reduction of the urinary colloids which adsorb solutes, or excess of muco-proteins which may chelate[1] calcium, predispose to the formation of an insoluble complex.

3. **Decreased Urinary Output of Citrate.**—The presence of citrate in the urine, 300–900 mg/24h (1·6–4·7 mmol/24h as citric acid), tends to maintain in solution otherwise relatively insoluble calcium phosphate and carbonate. The excretion of citrate is under hormonal control and decreases during menstruation.

4. **Renal Infection.**—Infection favours the formation of urinary calculi. Both clinical and experimental stone formation is common when the urine is infected with a urea-splitting streptococcus, staphylococcus, or proteus. The predominant bacteria found in the nuclei of urinary calculi are a staphylococcus and *Esch. coli*.

5. **Inadequate Urinary Drainage.**—Stones are prone to occur in patients with obstruction to the free passage of urine.

[1] Chelate—to seize hold of. Chētē, Gr. = the prehensile claw of a crab or scorpion.

James Christie Anderson, Contemporary. Consulting Surgeon, Royal Hospital, Sheffield, England.
Wilfred Hynes, Contemporary. Surgeon, Plastic and Jaw Department, United Sheffield Hospitals, Sheffield, England.
David Melvin Davis, Contemporary. Professor Emeritus of Urology, Jefferson Medical College, Philadelphia, U.S.A.

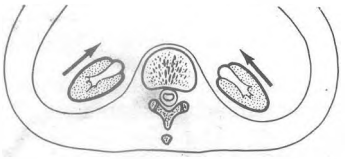


FIG. 1342.—In prolonged recumbency the fact that the urine has to travel uphill favours urinary stasis and consequent stone formation. (*After A. C. McEachern.*)

6. **Prolonged immobilisation** from any cause, e.g. paraplegia, is prone to result in skeletal decalcification and an increased output of calcium in the urine. This, combined with the mechanical effects of recumbency on renal drainage (fig. 1342), favours the deposition of calcium phosphate calculi. In uninfected cases spontaneous dissolution sometimes occurs.

7. **Hyperparathyroidism,** although rare, occurring perhaps in 5 per cent. of cases, should always receive consideration. In cases of multiple or recurrent urinary calculi this cause should be eliminated by biochemical tests (p. 647). Hyperparathyroidism results in a great increase in the elimination of calcium in the urine. It has been aptly remarked that these patients pass their skeleton in their urine. A parathyroid adenoma should be removed before the urinary calculi are treated.

8. **Randall's Plaque. Microliths.**—Randall showed that the initial lesion in many cases of renal calculus is an erosion at the apex of one of the renal papillæ. On this erosion are deposited urinary salts = Randall's plaque. It has further been shown that minute concretions (microliths) occur normally in the renal parenchyma and Carr postulates that these particles are carried away, like carbon particles from the bronchi, by lymphatics. Should some of the renal lymphatic vessels become blocked by inflammatory exudate, a subendothelial calculus may form. Later the endothelium becomes ulcerated, leaving the calculus in contact with the urine.

Varieties of Renal Calculi

Oxalate Calculus (Calcium Oxalate).—Popularly known as the mulberry stone, it is covered with sharp projections (fig. 1343). These cause the kidney to bleed, and altered blood is precipitated on the surface of the stone.

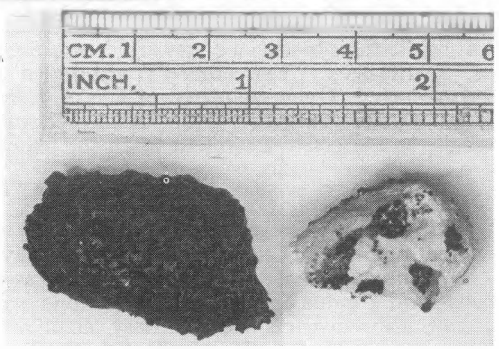


FIG. 1343.—Two oxalate calculi. The larger one removed from the right kidney and the smaller from the left of the same individual. The larger is black, owing to altered blood. The smaller is beginning to be discoloured around its sharp projections. These specimens illustrate clearly the process by which the oxalate calculus changes its complexion.

An oxalate calculus, which is usually single, casts an exceptionally good shadow radiologically, and this is fortunate, for often by virtue of its rough surface it gives rise to symptoms when comparatively small. A calcium oxalate stone is very hard, and on section is laminated concentrically. 'Envelope' crystals can be identified in the urine, viz. →

Phosphatic calculus (usually calcium phosphate, although sometimes combined with ammonium magnesium phosphate and, rarely, composed of the latter only) is smooth and dirty white. In an alkaline urine it enlarges rapidly, and often fills the renal calyces, taking on their shape (staghorn calculus, fig. 1344). Because it is smooth, a phosphatic calculus gives rise to few symptoms until it has attained a large size. By reason of its size rather than its density, it is demonstrated readily by X-rays.

Alexander Randall, 1883–1951. Professor of Urology, University of Pennsylvania, Philadelphia, U.S.A.
Reginald Joseph Carr, Contemporary. Radiologist, Bradford Royal Infirmary, Bradford, England.

Uric acid and urate calculi are hard and smooth and, because they are usually multiple, they are typically faceted. Their colour varies from yellow to reddish brown. Pure uric acid calculi are not opaque to X-rays, but absolutely pure uric acid calculi are uncommon; the majority contain enough calcium oxalate crystals to render them opaque. Calculi of ammonium and sodium urate are sometimes found in children. Such stones are yellow, soft, and friable, and unless they contain impurities they do not cast an X-ray shadow.

FIG. 1344.—Phosphatic calculus (actual size). Note its branched nature (staghorn calculus).

Cystine calculi are wont to appear in the urinary tract of patients with cystinuria and are sometimes encountered in young girls. Cystinuria is *not* an inborn error of metabolism; it results from a greatly diminished or absent resorption of cystine from the renal tubules. Cystine crystals are hexagonal, white, translucent, and appear only in acid urine (fig. 1345). Cystine calculi are usually multiple and may assume a cast of the renal pelvis and calyces. These calculi are soft, like beeswax, and pink or yellow when first removed. On exposure the colour changes to a greenish hue. They are radio-opaque owing to the sulphur they contain.

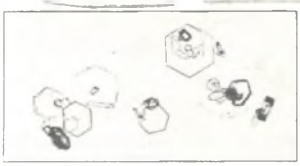


FIG. 1345.—Cystine crystals from the urine of a patient with cystinuria.

Xanthine calculi are extremely rare. They are smooth and round, brick red in colour, and show a lamellar structure.

Indigo calculi are curiosities. Blue in colour, they are derived from indican.

Clinical Features.—Fifty per cent. of patients with renal calculus present between the ages of thirty and fifty. The male/female ratio is 4:3.

The symptoms are not stereotyped and sometimes the diagnosis remains obscure until a radiological examination has been made.

Quiescent Calculus.—Some stones, especially those composed mainly of phosphates, lie dormant for a long period, during which time there is progressive destruction of the renal parenchyma, and uræmia may be the first indication. Secondary infection usually supervenes.

Pain is the leading symptom in 75 per cent. of cases.

Fixed renal pain is located in the renal angle posteriorly (fig. 1346), in the

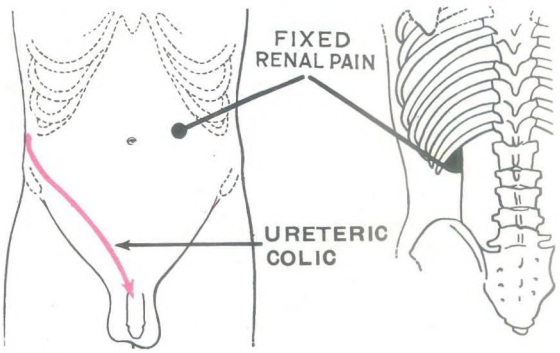


FIG. 1346.—The usual distribution of renal pain.

hypochondrium anteriorly, or in both situations simultaneously. It is often worse on movement, particularly on walking upstairs.

Ureteric[1] *colic* is an agonising pain passing from the loin to the groin, coming on suddenly, causing the patient to draw up his knees and roll about. It is often accompanied by vomiting and profuse sweating. Strangury[2] may occur if the stone is in the intramural ureter. The pulse quickens, and as the attack progresses the temperature becomes subnormal. An attack of colic rarely lasts more than eight hours. The condition is often due to a stone entering the ureter, but also occurs when a stone in the renal pelvis temporarily blocks the pelviureteric junction. Colic may also be caused by the passage of a shower of oxalate crystals, e.g. after eating an excess of strawberries or rhubarb (p. 1196).

Abdominal Examination.—During an attack of ureteric colic there is rigidity of the lateral abdominal muscles but not, as a rule, of the rectus abdominis. Percussion over the kidney produces a sharp stab of pain; tenderness of the kidney may be apparent on bimanual palpation. Only when a calculus-containing kidney becomes the seat of a hydronephrosis or pyonephrosis of considerable size does it give rise to a palpable swelling.

Hæmaturia.—Infrequently, hæmaturia is a leading, or the only symptom. As a rule hæmaturia occurs in small amounts (enough to render the urine 'smoky') during or after an attack of pain.

Pyuria.—Infection of the kidney is liable to supervene, and pus will be found in the urine in varying amounts. Stones lead to an increase in the number of white cells in the urine even in the absence of infection.

Investigation of a suspected case of Renal Calculus

Radiography.—Before the examination the bowels should be emptied by giving a *vegetable* laxative, for minerals in the intestine cast a shadow (fig. 1347). Radiographs of both kidneys, ureters, and the bladder are taken.

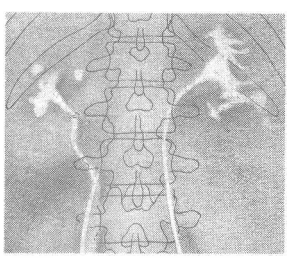


FIG. 1347.—The two shadows in the region of the right kidney were mistaken for calculi. They were due to enteric-coated pills of ammonium chloride.

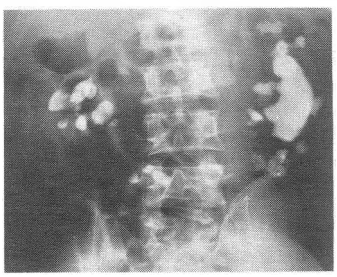


FIG. 1348.—Plain radiograph showing horse-shoe kidney full of stones. The patient, a man of forty-two, stated that he had enjoyed good health until one week previously.

[1] See footnote p. 1160

[2] Strangury is the passage of a few drops of urine, often blood-stained, after painful straining.

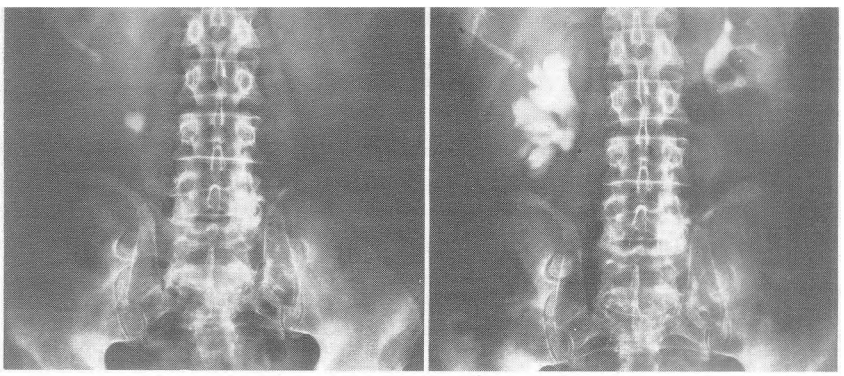
When a renal calculus is branched, there is no doubt concerning the shadow it casts. If a shadow *may* be a calculus in the kidney, exposures are made during full inspiration and full expiration. If the opacity moves with the kidney, and measurements from the lower pole of the kidney to the opacity remain constant, then the shadow is intrarenal.

The following structures and substances from time to time cast a shadow which at first sight may appear to be a renal calculus:

(i) A calcified lumbar or mesenteric lymph node.
(ii) A gall-stone or a concretion in the appendix.
(iii) Drugs (e.g. fersolate) or foreign bodies in the alimentary canal.
(iv) Phleboliths.
(v) The ossified tip of the twelfth rib.
(vi) A chip fracture of the transverse process of a lumbar vertebra.
(vii) Calcified tuberculous lesion of the kidney.
(viii) A calcified suprarenal gland.

A doubtful shadow in relation to the kidney can often be proved to be extrarenal by a *lateral radiograph*. When the shadow can be shown to lie anterior to the bodies of the vertebræ, *ipso facto* it is anterior to the kidney. Such is the finding in calcified mesenteric lymph nodes, gall-stones, and opacities in the alimentary tract.

Excretory pyelography is of value in confirming that the opacity is intrarenal, in determining in which part of the kidney the stone is situated (fig. 1349), and in revealing the function of the other kidney.



(*a*) (*b*)

FIG. 1349.—(*a*) Plain X-ray showing a renal calculus. In which part of the kidney is it situated? (*b*) Same case. Excretory pyelogram. The stone is obstructing the pelvi-ureteric junction. Pyelolithotomy is needed urgently.

The presence of a non-opaque calculus also can be demonstrated by pyelography, for it causes a filling defect in the shadow cast by the medium. A similar defect is seen in papilloma of the renal pelvis (p. 1220). Retrograde pyelography may be required to give a clearer delineation.

Cystoscopy.—A urethral stricture or prostatic obstruction, both of which may be a predisposing factor in renal stone formation, will be revealed by instrumental examination. Unless cystitis is present, the bladder wall looks

normal. The urinary efflux from the affected side is increased if the stone is irritating the renal pelvis, or decreased when the pelvi-ureteric junction is blocked or when the parenchyma is severely diseased.

Treatment of Ureteric Colic (p. 1199).

OPERATIONS FOR THE REMOVAL OF RENAL CALCULI

Pre-operative Treatment.—If urinary infection is present, antibiotic treatment is instituted, and continued after operation as necessary. A bacteriology swab for culture should be taken of urine in the renal pelvis.

Operation—General Remarks.—All the operations about to be described have certain features in common. Via a lumbar incision (p. 1221) the kidney is exposed and mobilised. When delivery is impossible, the choice of procedure is more limited and the operation more difficult. An X-ray showing the calculus or calculi clearly should be displayed in an illuminated viewing-box in the operation theatre. The stone or stones removed should coincide in every respect with the X-ray findings.

It is advisable to X-ray the exposed kidney (using a special film encased in a sterile plastic cover). This ensures that no stone or portion of stone within the kidney has been overlooked, and reduces the number of so-called recurrences.

Nephroscopy.—The inspection of the interior of the pelvis and calyces by means of a urethroscope is feasible in some kidneys. The development of rod lens systems, and glass fibres for transmission of increased light, has enabled the production of a nephroscope with flushing system and small forceps with which the interior of calyces can be examined, small calculi picked off, and subepithelial plaque visualised. This teaches one that even a clear X-ray is not proof that kidneys are cleared of foci on which other calculi can form.

At the conclusion of all the operations to be described the kidney is replaced in its bed, the fascia of Gerota closed, and the lumbar wound closed with drainage of the perinephric space. If nephrostomy has been performed, the tube is either brought through the lumbar incision or through a stab incision.

Pyelolithotomy is indicated when a stone (it is usually solitary) lies in the renal pelvis, provided the pelvis is extrarenal. In nearly 50% of cases a renal calculus is so situated. The posterior wall of the renal pelvis is dissected free from its surrounding fat. The kidney is grasped in the left hand so that the tips of the index and middle fingers lie beneath the renal pelvis, making it more prominent, while the thumb anteriorly prevents the stone slipping into one of the calyces. An incision is made on to the stone in the long axis of the renal pelvis. The stone is removed with gall-stone or suitable forceps, care being taken to avoid breaking it. If pyelography showed a stone in a calyx and the renal pelvis is large, the stone can be located by the little finger introduced into the renal pelvis: should the stone be accessible, it can be grasped in forceps and removed by this route. After a stone has been removed via the renal pelvis, a bougie is passed through the pelvi-ureteric junction to calibrate it and to exclude any obstruction lower down. If the kidney is known to be uninfected, the incision in the renal pelvis is closed by interrupted sutures of fine plain catgut. If the kidney is grossly infected, nephrostomy (p. 1173) is performed before closing the incision in the renal pelvis.

Extended Pyelolithotomy (Gil-Vernet) by dissecting in the renal sinus posteriorly, a plane between the pelvis and calyces and the renal vessels leads to the necks of the calyces. Incisions continued into the calyces enable a direct view into the calyx

Gil-Vernet, Contemporary. Surgeon, Madrid, Spain.

to be obtained in many cases, facilitating the removal of large and small stones. Thermocouple-regulated cooling of the kidney, by ice chips in polythene bags, or liquid nitrogen circulating through coils placed around the kidney, enables the renal pedicle to be clamped to provide a bloodless field and unhurried surgery.

Nephrolithotomy is frequently indicated if extended pyelolithotomy is employed. It may be necessary when there has been previous surgery, with adhesions making approach to the pelvis difficult. The kidney is cooled, and the renal pedicle is clamped as an incision into the renal parenchyma is accompanied by considerable hæmorrhage. The incision is made just behind and parallel to the most prominent part of the convex border of the kidney (Brödel's line). As the terminal branches of the anterior and posterior renal arteries meet along this line, no large artery is divided, thereby minimising the amount of subsequent cortical necrosis. The incision, usually about 2·5 cm. long, is made over that calyx containing the stone or stones, which are removed with lithotomy forceps. All bleeding points are underrun and tied with 0000 chromic gut, the edges of the opened calyx closed if necessary. The renal incision can be closed by interrupted catgut sutures passing through the kidney substance but not penetrating the calyx, and tied over a piece of Oxycel or muscle to enhance hæmostasis. If infection is present, nephrostomy is carried out by placing a small self-retaining catheter through the incision into the renal pelvis. The incision is then closed on either side of the tube in the manner just described.

Pyelonephrolithotomy is used to remove a stone situated deeply in a calyx, especially if it is present in addition to one in the renal pelvis, and in cases of stones situated in more than one calyx. The pelvis is opened, the little finger is introduced and the calyx containing the stone is palpated. If pressure is exerted on the corresponding convex border of the kidney, even a small stone can usually be palpated. Otherwise, the kidney is punctured with a round-bodied needle so that the point grates on the stone. An incision is made through the parenchyma, and with the finger pressing on the calculus from within, it is grasped by lithotomy forceps introduced through the cortical incision, and withdrawn. If there are stones in other calyces the procedure may be repeated, making two, or even three, cortical incisions. The incisions are closed in the manner already described. If drainage of the interior of the kidney is necessary, the catheter is introduced through the cortical incision or, if there are more than one, through the most dependent.

Partial Nephrectomy.—When the stone is in the lowermost calyx—a fairly common position—and there is considerable damage to the calyx, as in multiple stones, or 'grit' in the renal substance, then lower partial nephrectomy is the best operation (p. 1222). If the damaged calyx is not removed, its dependent position encourages further stone formation.

Nephrectomy (p. 1221) is indicated when the contralateral organ has been proved to be perfectly healthy and the stone or stones have destroyed much of the renal parenchyma. Large branched calculi are notorious for recurring, so nephrectomy is often the best treatment in such cases.

Post-operative Treatment.—The patient should lie on the side of the operation or on his back to encourage drainage and obliteration of dead space in the wound. Wound drains should be shortened after 36–48 hours and removed as soon as any leak has ceased.

If a nephrostomy tube has been inserted then it is wise to give a wide spectrum antibiotic to decrease the risk of infection and possible secondary hæmorrhage.

Treatment of Bilateral Renal Calculi.—Usually the kidney with the better function is operated upon first, the operation on the contralateral side being postponed for two or three months. Exceptions to this rule are (*a*) if there is pain on one side, that side is operated upon first, for pain usually signifies that the stone is obstructing the outflow of urine from the kidney, the function of which will become increasingly impaired; (*b*) if there is a

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The first operation of nephrolithotomy was performed by Ambroise Paré (1510–1590). His patient was a criminal condemned to death by hanging, instead of which Paré removed two stones from his kidney. Fifteen days later the patient was cured, secured his remission, and was given a grant of money.

Max Brödel, 1870–1941. Artist of Pathology and Founder and Director of the Department of Art as Applied to Medicine (the first school of its kind in the world), Johns Hopkins University, Baltimore, U.S.A.

pyonephrosis on one side this should be treated by nephrectomy, or, if the patient's condition is poor, by nephrostomy. In cases of bilateral staghorn calculi without infection and with few symptoms especially in the elderly, it is probably wiser not to operate. The patient should drink large amounts of fluid.

Prevention of Recurrence

Frère Jacques, that famous lithotomist of the Middle Ages, used to say, " I have removed the stone, but God will cure the patient." With the advance of knowledge our responsibilities do not end with the removal of the stone; provision must be made to prevent recurrence. Recurrent calculi can be divided into two varieties:

(*a*) *False Recurrence.*—Even a tiny fragment overlooked at the time of the operation is liable to become the nucleus of a new stone. Similarly infection and obstruction are factors in recurrence and should be eliminated in the treatment of the first stone.

(*b*) *True Recurrence.*—Ideally all stone formers should be investigated to exclude metabolic factors. The following investigations in bilateral and recurrent stone formers are essential:

(i) Serum Calcium, measured starving on three occasions to exclude hyperparathyroidism. This series may need to be repeated (p. 647).

(ii) Serum Uric Acid to exclude gout. Elevated serum uric acid levels can predispose to the formation of calcium phosphate stones.

(iii) Urinary urate and calcium and phosphate excretions in 24-hour urine, and screening for cystine.

(iv) Analysis of any stone passed.

(*c*) *Dietary Advice:*

(i) There should be a high fluid intake at all times, regardless of the chemistry of the stones. This is clearly indicated in cystinuria where the liability to stone formation is directly related to the concentration of the cystine in the urine. It is equally important to keep the urine dilute at night. Avoidance of milk, cheese and other items of the diet containing a great deal of calcium and, if renal function is satisfactory, the use of sodium cellulose phosphate (5 g t.d.s. with meals) to reduce calcium absorbtion may be considered.

(ii) **Uric-acid and Urate Calculi.**—Red meats, offal, and fish which are rich in purine should be avoided. Sufficient sodium citrate and sodium bicarbonate are given to keep the urine alkaline. Allopurinol in doses sufficient to overcome a high serum uric acid is essential.

(iii) **Calcium Oxalate Calculi.**—Rhubarb, strawberries, plums, spinach, and asparagus, which are rich in oxalate, should only be taken if they are accompanied by cream or milk, as then oxalates are precipitated as an insoluble calcium salt in the intestine, and are not absorbed. At all times a diet adequate in magnesium is necessary; calcium oxalate is rendered less soluble in the presence of magnesium ions.

(iv) **Phosphate Calculi.**—Excessive alkalinity of the urine should be treated by giving ammonium chloride or another urinary acidifier. Phosphates in the diet should be restricted. Aluminium gel, 40 ml. t.d.s., p.c. and at bedtime, drastically reduces the incidence of recurrence.

(v) **Cystine Calculi.**—Sulphur-containing proteins such as eggs, meat, or fish are restricted, and proteins with a low sulphur content substituted. Carbohydrates and fats are unrestricted. The urine must be kept dilute at all times as the formation of calculi is directly related to the concentration of cystine in the urine. Fluids must be drunk during the night as well as during the day. Solubility is increased by keeping the urine strongly alkaline, but this is often difficult to achieve. Penicillamine is a costly and by no means always successful drug in preventing recurrent cystine calculi.

(vi) **Recumbency Calculi.**—In patients doomed to long recumbency, sodium phytate taken by mouth forms insoluble calcium phytate, thus removing by the bowel one source of hypercalciuria. Regular timing is essential.

(vii) **Idiopathic Hypercalcuria.**—A strict low calcium diet (400 mg daily) should be tried first. 5 mg Bendrofluazide reduces calcium loss in the urine. Water softening to remove calcium in the local water may be required in order to allow a high fluid intake. Sodium phytate taken orally at regular intervals is the last line of treatment.

Frère Jacques, 1651–1719. Itinerant Italian lithotomist, after serving as a trooper in the French army, adopted semi-religious habit and cut for stone in the bladder.

URETERIC CALCULUS

A stone in the ureter nearly always has its birth in the kidney. When it enters the ureter the calculus is rounded or oval in shape. Should it become arrested in its descent to the bladder, in time it becomes enlarged and elongated, resembling in shape a date stone (fig. 1350). In 90 per cent. of cases the stone is single.

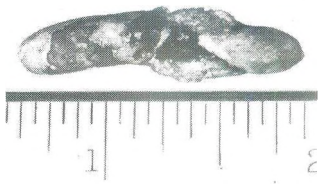


FIG. 1350.—Ureteric calculus removed by operation.

Clinical Features.—When a stone descends into the ureter there is an attack of ureteric colic, which is repeated at longer or shorter intervals, until the stone is ejected into the bladder or becomes impacted in the ureter. There are five sites of anatomical narrowing of the ureter where a stone may be arrested (fig. 1351).

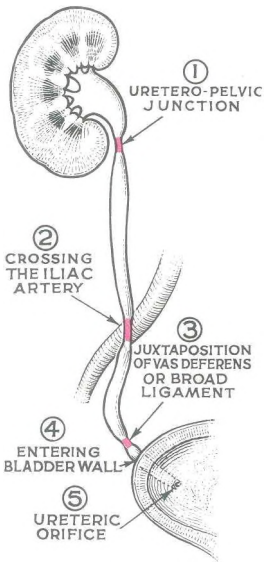


FIG. 1351.—Normal anatomical narrowings of the ureter. (*After C. C. Higgins.*)

Ureteric Colic.—A stone in the upper third of the ureter produces symptoms identical with those of a stone blocking the pelvi-ureteric junction (p. 1191). When a calculus enters the lower third of the ureter, the colic it produces often commences anteriorly at a lower level than the kidney, and is frequently accompanied by pain referred along the two branches of the genito-femoral nerve, to the testis in the male (fig. 1352), the labium majus in the female, and to the anterior surface of the thigh in both sexes. The testis usually becomes retracted by spasm of the cremaster, and tender, the tenderness persisting for some time after the colic has ceased. When the stone enters the intramural portion of the ureter, the pain is referred to the tip of the penis in the male. In both sexes there is strangury. In more than 50 per cent. of cases the stone is passed spontaneously.

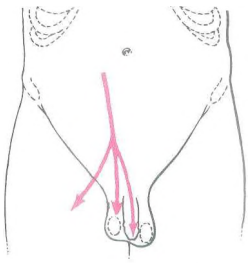


FIG. 1352.—Radiations of ureteric colic produced by a stone in the lower third of the ureter.

Impaction.—When the stone becomes impacted the attacks of colic pass off and give place to a dull pain, usually in the iliac fossa, for impaction usually occurs in the pelvic portion of the ureter. The pain is increased by exercise, and relieved by rest. Such pain varies in intensity and is often associated with backache due to distension of the renal pelvis. Cessation of pain sometimes occurs by the stone forming for itself a false diverticulum in the wall of the ureter by a process of pressure necrosis; subsequent perforation of the ureter occurs but rarely. Severe renal pain persisting for one to two days and then gradually subsiding suggests that complete obstruction has occurred.

If an I.V.P. performed one or two weeks later shows no function, the stone must be removed, otherwise complete atrophy of the kidney will occur.

Hæmaturia.—Some degree of hæmaturia occurs after an attack of ureteric colic, and it lasts for a few hours or a day. It is sometimes so slight as to require microscopic identification.

Abdominal Examination.—There is tenderness and often rigidity over some part of the course of the ureter. On rare occasions a stone in the lower end of the ureter can be felt on rectal or vaginal examination. The principal difficulty on the right side is distinguishing the symptoms and signs produced by a ureteric calculus from those of acute appendicitis. The presence of blood in the urine does not necessarily rule out appendicitis, for an inflamed appendix lying in juxtaposition to the ureter can give rise to ureteritis which may cause a small amount of red cells in the urine. Pain on percussion over the kidney is unlikely in acute appendicitis.

FIG. 1353.—Excretory pyelogram revealing hydronephrosis and hydroureter due to stone in lower ureter.

Radiography.—Occasionally a typical elongated shadow in the line of the ureter confirms the diagnosis; more often either no shadow is seen, owing to the small size of the stone and the presence of intestinal gas shadows, or a shadow is seen which may or may not be a ureteric calculus.

Excretory Pyelography.—In, and for some time after, an attack of renal colic there is either no excretion or greatly delayed excretion on the affected side. Films taken later, even up to forty-eight hours after the intravenous injection of Hypaque (diatrizoate) may outline the ureter down to the site of obstruction. A stone not visible in the plain X-ray may become outlined in the ureter by the contrast solution. After a stone has been impacted for a week or more, a varying degree of hydronephrosis and dilatation of the ureter is often apparent (fig. 1353).

If pyelograms taken during an attack of pain show normal excretion on the affected side, then the patient has not got a ureteric colic.

Cystoscopy.—When a calculus is in the upper part of the ureter, the ureteric orifice shows no abnormality. When a calculus has reached the lower third of the ureter, or sometimes when it is in the middle third, the ureteric orifice becomes patulous and its immediate vicinity bespattered with minute petechial hæmorrhages. As the calculus descends to just above the bladder wall, these hæmorrhagic spots coalesce to form larger, bright-red extravasations. The stone having entered the intramural portion of the ureter, the ureteric orifice becomes grossly œdematous. Finally, the pouting calculus may be seen (fig. 1311).

The passage of a ureteric catheter provides much information, and in conjunction with radiography confirms the diagnosis. Usually a catheter fails to pass the obstruction and a radiograph shows the tip of the catheter abuttingon the lower margin of the calculus. However, when the urinary efflux is increased, a catheter sometimes passes into the renal pelvis without hindrance, or, if arrested, after partial withdrawal and reinsertion (fig. 1354).

Retrograde pyelography is seldom required. The only indications for its employment are when there is doubt concerning a shadow being a ureteric calculus and excretory pyelography shows absence or ineffective delineation of the corresponding renal pelvis and the ureter. It sometimes proves a means of displaying the presence of a ureteric calculus non-opaque to X-rays.

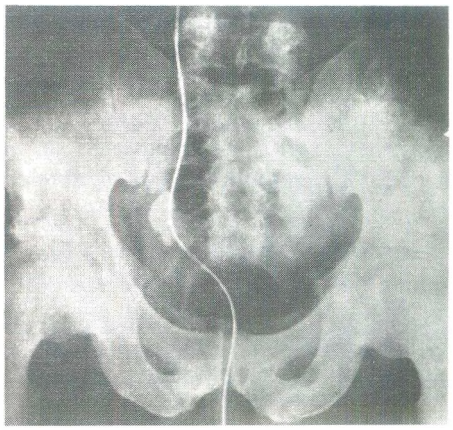
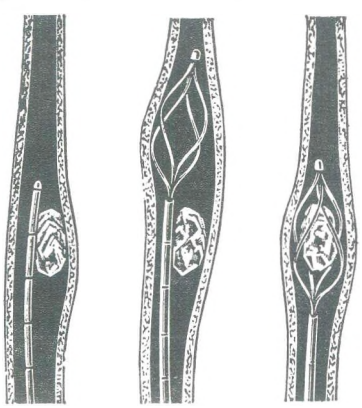


FIG. 1354.—Stone in the ureter. Catheter passed alongside the stone.

Treatment.—1. Of the Colic. Morphia (15 mg) is the best drug to relieve severe pain and does not produce spasm in ureteric muscle. The stone produces spasm and one dose of probanthine (15 mg) should be given at the same time. Repeated doses of probanthine can easily produce retention of urine by paralysis of the detrusor muscle of the bladder.

2. **Of the Stone.**

(i) **Expectant.** Provided the urine is sterile, most small calculi will pass naturally. This may take many months. Progress of the stone is observed by plain X-rays every six to eight weeks, and occasional pyelography to see that the function and appearance of the kidneys are satisfactory.



(*a*) (*b*) (*c*)

FIG. 1355.—Dormia stone catching basket in use. (*a*) basket introduced past stone. (*b*) opened and (*c*) enclosing stone ready for withdrawal. (*Porgès, Paris*).

(ii) **Removal of the stone** may be required if:

(*a*) Repeated attacks of colic produce no advance of the stone.

(*b*) The stone is enlarging but not moving.

(*c*) Complete obstruction of the kidney occurs, or a hydronephrosis is increasing.

(*d*) The urine is infected.

(*e*) The stone when first seen is thought to be too large to pass.

Instrumental Treatment

(*a*) *Ureteric catheterisation.*—A small stone half a centimetre or less in diameter arrested in any part of the ureter often passes after ureteric catheterisation.

(*b*) *Dormia Basket* (fig. 1355).—This can be used for stones in the lower third of the ureter.

Dormia, Contemporary. Assistant Professor of Urology, University of Milan, Italy.

(*c*) *Ureteric Meatotomy.*—The ureteric orifice is the narrowest part of the whole ureter. By enlarging the opening a stone is often enabled to pass. However, this procedure almost invariably leads to urinary reflux afterwards and should therefore be avoided.

(*d*) *Intra-ureteric Manipulation* with stone catchers, loop catheters and corkscrew stone dislodgers should never be used in mid or upper ureter.

Operative Treatment

Uretero-lithotomy.—The patient should be submitted to radiography just prior to the operation, in order to detect any change in the position of the calculus.

(*a*) **Stone Impacted in the Upper Third of the Ureter.**—An incision similar to that for exposing the kidney (p. 1221) is made; it can be prolonged downwards if necessary.

(*b*) **Stone Impacted in the Lower Two-thirds of the Ureter, but above the Spine of the Ischium.**—With the patient in the dorsal position, an incision is made from a point 4 cm above the anterior superior iliac spine to the middle of the inguinal ligament. The flank muscles are divided in the direction of their fibres. The peritoneum is displaced medially by gauze dissection until the dilated ureter is found adhering to its under-surface. The stone is sought by palpation, and, if possible, is milked upwards or downwards to a convenient point for extraction. The ureter is incised longitudinally and the stone withdrawn by a scoop or forceps. If the stone is impacted firmly, the only course is to incise the ureter directly over it, a practice to be avoided whenever possible, for the mucous lining is ulcerated at the site of impaction, and healing is impaired. The incision in the ureter is closed by sutures of 0000 plain catgut passing through the muscular coats only. The lower end of the wound is drained down to the incision in the ureter.

(*c*) **Stone Impacted in the Juxtavesical or Intramural Portions of the Ureter.**—A midline, sub-umbilical extraperitoneal approach is employed, with the patient in Trendelenburg's position. The peritoneum is stripped from the dome of the bladder and from the side wall of the pelvis. A dissection is then made between the posterior surface of the bladder and the wall of the pelvis. Often at this stage the stone will be felt. If this is not possible, the ureter must be identified where it crosses the iliac vessels, and traced downwards. Once the stone has been located it is removed as described above.

After removal of a stone at any site, a ureteric catheter or bougie should be passed to prove the patency of the distal ureter.

Idiopathic retroperitoneal fibrosis.

This condition is rare. The patient, who typically has complained of backache for many months, suddenly becomes anuric. In other circumstances when kidney disease has been suspected pyelography reveals rapidly developing unilateral or bilateral hydronephrosis associated with a rising blood urea. Characteristically the ureters are nearer the mid-line than normally and this is due to an area of fibrosis centred over the bifurcation of the aorta which strangulates the ureters. The cause is largely unknown. Some cases have been noted following the use of methysergide for the treatment of migraine, and methyl-dopa may also cause the condition.

Treatment.—In the anuric patient, if an attempt at ureteric catheterisation is successful, the catheters should be left *in situ* to relieve the obstruction. If this fails, bilateral nephrostomy is necessary although the use of the artificial kidney (p. 1171) may be beneficial. Steroids may be considered.

In non-urgent cases the involved ureter or ureters should be freed from the fibrous mass by sharp dissection and fixed laterally to the psoas muscles to prevent recurrence. Should this prove impossible an ileal loop ureter should be constructed (p. 1262).

INFECTIONS OF THE KIDNEY

Ætiology.—Renal infections arise in the following ways :

1. *Hæmatogenous.*—From infected tonsils, carious teeth, or from cutaneous

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig, Germany.

infections, particularly boils or a carbuncle, and in the case of tuberculosis from affected lymph nodes in the neck, chest or abdomen.

2. *Ascending the peri-ureteral lymphatics* from the cervix or the prostate.

3. *Ascending the lumen of the ureter* from the bladder is the commonest route. It may occur in lower urinary tract obstruction with dilated ureters as a result of vesicourethral reflux. This is an important route also in children with recurrent urinary tract infection.

On complete urinary investigation, which should always be undertaken after the acute phase of the infection has subsided, about 30 per cent. of males and a smaller number of females will be found to have an obstructive lesion in some part of the urinary tract. Moreover, with or without such a lesion, a focus of infection in the genital organs may be demonstrable.

Bacteriology.—By far the most common organisms responsible are *E. coli* and other Gram-negative bacilli. When *Strept. fæcalis* is present it is usually associated with another organism. In *E. coli* and streptococcal infections the urine is acid. Both Proteus and Staphylococci split urea into ammonia, rendering the urine alkaline and so may promote stone formation.

PYELONEPHRITIS

Renal infection, by whatever route it originates, is never confined to the renal pelvis. In hæmatogenous infection the renal parenchyma is attacked first, and the infection spreads to the renal pelvis. In ascending infection the bacterial inflammation is not limited to the renal pelvis; it implicates the calyces, the pyramids, and at least some part of the adjacent parenchyma. It is, therefore, more correct to discard the term ' pyelitis ' in connection with the latter type of infection, in favour of the more accurate designation ' pyelonephritis '.

ACUTE PYELONEPHRITIS

Acute pyelonephritis is more common in females, especially during childhood, at puberty, soon after marriage (' honeymoon pyelitis '), during pregnancy, and the menopause. It occurs more often on the right side than on the left, and is frequently bilateral.

Clinical Features.—While there may be prodromal symptoms of headache, lassitude, and nausea, the onset is usually sudden, often commencing with a rigor and, even, vomiting. There is acute pain in the flank and the hypochondrium. In a few cases the pain radiates from the loin to the groin (ureteric colic). The temperature rises to 38·8° or 39·5° C, and is remitting. Soon after the onset, increased frequency of micturition, due to cystitis, sets in, and when, as is often the case, the urine is acid, micturition is accompanied by a scalding pain in the urethra. On examination there is tenderness in the hypochondrium and in the angle between the last rib and the sacrospinalis, accompanied by a varying degree of muscular rigidity. In the early stages, when it is imperative to make a correct diagnosis, the urine is clear macroscopically.

In cases of severe bilateral pyelonephritis, particularly when there is an associated obstruction, there are likely to be symptoms of uræmia.

Bacteriological Examination of the Urine.—A mid-stream specimen should be collected in a sterile bottle; the urine is centrifuged and examined microscopically. In early acute pyelonephritis there are typically a few pus cells and many bacteria. When pyelonephritis has been present for twenty-four hours or more, the urine is often cloudy and pus cells abound. Quantitative estimations of the cells and bacteria in the specimen are of real value, whereas vague impressions reported as . . . ' many, some, a few ' are usually misleading. In all cases the investigation includes an examination of the sediment stained by Gram's method, culture of the specimen, and an investigation of the sensitivity of the organism or organisms to various antibiotics and chemotherapeutic agents.

Severe Cases.—There are repeated rigors and the temperature rises to 40° or 40·5° C, often without a corresponding rise in the pulse-rate. There is vomiting, sweating, and great thirst, and the patient looks and feels ill. The blood culture, if the specimen is taken soon after a rigor, often gives a positive result. After some hours the pain is localised in one or, rarely, both loins. Urine is scanty and highly concentrated, and is frequently teeming with coliform organisms and pus cells, in which case the diagnosis is simplified. In blood-borne infections the organisms and pus cells do not appear in the urine until the infection has spread from the cortex to the medulla—a matter of many hours, or even days. As the abdominal rigidity abates, the enlarged affected kidney may be palpable.

Differential Diagnosis.—When the symptoms and signs are typical the diagnosis is straightforward. In other circumstances there may be difficulty in distinguishing the condition from pneumonia, acute appendicitis, and acute cholecystitis. The urgent need is to differentiate acute appendicitis from right-sided pyelonephritis. The fact that the pain commenced in, and did not pass to the right side, greatly favours the latter condition. Excretory pyelography may prove of assistance, for an early acute pyelonephritis limited to the right kidney the concentration of medium in the renal pelvis and calyces on the affected side may be so poor that no shadow, or a very indefinite shadow, is cast. A radio-isotope renogram using Chlormerodrin (^{203}Hg) 1·25 μCi per kg body weight will show scattered areas of diminished uptake (fig. 1307).

Pyelonephritis of Pregnancy.—The condition usually develops between the fourth and sixth months of gestation. It is commoner in women who have had recurrent bouts of cystitis or pyelonephritis in the past. In 90 per cent. of cases the right side alone is affected. The initiating causes are often chronic urethritis or a cervical erosion. The symptoms do not differ from those of pyelonephritis occurring in the non-pregnant. In about 10 per cent. of cases the disease runs a more severe and protracted course, and occasionally it results in abortion or premature birth. In all patients who have had pyelonephritis of pregnancy, periodic examinations of the urine are necessary during the rest of the pregnancy, the puerperium and for several months afterwards, for if the infection has not been eradicated, recrudescence is liable to occur during this period.

Urinary infection in childhood is not uncommon, and is important to recognise because it frequently involves the kidneys. If recurrent it may lead to severe renal impairment or renal failure in early adult life. In young children there may be no urinary symptoms, although the mother may notice a cloudy or offensive urine. If a child fails to thrive, refuses to eat, has a high fever (temp. 39–40° C) with *rigors or convulsions*, the possibility must always be considered. Pain or screaming on micturition may occur. The older child may complain of loin pain, or suddenly develop frequency and enuresis.

Hans Christian Joachim Gram, 1853–1938. Professor of Medicine, Copenhagen.

Many cases (50%) are associated with an underlying anatomical or congenital abnormality. Once the diagnosis has been confirmed by a clean catch specimen or by a specimen obtained by suprapubic needle puncture of the bladder, a full urological investigation is essential.

Reflux of urine occurs up the ureters during micturition in about 35% of cases with a proven urinary infection. It may be intermittent and is certainly more frequent when infection is present. It can only be demonstrated clearly by micturating ciné-cystography, but this does necessitate catheterisation: this must be performed gently and with strict asepsis, and it is wise to leave a solution of neomycin (50 ml 0·5% freshly made) in the bladder afterwards. Renal function tests and full blood analysis should be done.

Reflux also occurs when the intravesical pressure is high, and the obliquity of the intramural ureter is lost. A thin bladder wall and the presence of a saccule near the ureteric orifice may lead to funnelling of the orifice during ureteric peristaltic retraction.

Mild and moderate reflux up to the upper third of the ureter may not require reimplantation. Severe reflux into the pelvis should be treated by reimplantation to increase the obliquity of the course of the ureter through the muscle wall. Children with reflux should be taught to perform ' double micturition ' in order to limit the urinary stasis if operation is not going to be carried out.

Urinary infection must be treated with the appropriate drug until the clean catch specimen is sterile on culture and free from white cells on microscopy. A repeat urine sample 10–14 days after cessation of treatment must be examined to exclude recurring infection.

ACUTE PYELONEPHRITIS ASSOCIATED WITH RETENTION OF BLADDER URINE

Acute Pyelonephritis with retention of bladder urine occurs most frequently in men suffering from prostatic obstruction or stricture of the urethra, and in cases of fracture of the spine with injury to the spinal cord (p. 483). The retention is not necessarily complete and the patient may be able to urinate, but there is a varying amount of residual urine, from a few ounces to several pints. In most cases the infection is bilateral. Often it follows the passage of a catheter or other instrument, or operations on the lower urinary tract, but it may arise spontaneously. The old name for this condition was ' surgical kidneys ', and in the days of unsterile catheterisation it was frequent and dreaded. At post-mortem examination the kidneys are enlarged and miliary abscesses can be seen beneath the capsules. On bisection there are numerous yellow streaks of pus in the parenchyma radiating from the medulla to the convex border. The renal calyces, pelves, and ureters are dilated.

Treatment of Acute Pyelonephritis

The essential factors in curing pyelonephritis are *early*, *correct* and *prolonged* treatment, and careful follow-up with repeat cultures and white cell counts of the urine after treatment has finished. A full investigation to exclude abnormalities in the urinary tract should be undertaken when the acute attack is controlled.

A mid-stream specimen of urine should be taken and sent for culture and tests of the sensivity of the organisms to antibiotics. Bed rest is important.

If the urine is acid, as it is in the common coliform infections, alkalinisation of the urine has a most beneficial effect in relieving the symptoms and in inhibiting the growth of these organisms. Mist. Pot. Cit. et Hyocyamus, 10 ml. 6 hourly is an old but useful treatment.

When pain is severe, an antispasmodic is given and heat is applied to the affected loin. Morphia, in addition, may be necessary sometimes.

The patient should be instructed to drink large quantities of bland fluid, at least 3 litres in the twenty-four hours. In severe cases with vomiting or

dehydration, intravenous dextrose-saline is given until the dehydration has been rectified and the vomiting has ceased.

While awaiting the bacteriological report and the result of the sensitivity tests, in comparatively mild cases a soluble sulphonamide can be given as directed below, but in severe cases an antibiotic with a wide range of activity, e.g. ampicillin, should be substituted. When the bacteriological report is to hand, the drug of choice should be given.

Three and seven days after commencing the treatment of pyelonephritis the urine must be sent for culture. Even when a full initial course (ten days) of an antibiotic or chemotherapeutic agent has rendered the urine sterile, it is advisable to continue treatment with another drug for a further two to three weeks, or until the hourly white cell excretion rate has fallen to normal levels (less than 200,000 per hour). During this treatment further investigations to rule out abnormalities in the urinary tract should be carried out (p. 1161). Despite the undoubted efficacy of modern antibacterial drugs, none is likely to succeed in sterilising the urine of a patient with an abnormality of the urinary tract. A history of previous urinary infection also reduces the likelihood of a cure, although to a lesser extent (Garrod).

Colomycin, *Gentamicin* and *Carbenicillin* are the best drugs currently available for combating infections with the more resistant strains of *Pseudomonas Pyocyanea*, Proteus, and Klebsiella.

Chemotherapeutic and Antibiotic Agents

Sulphonamides: *Sulphafurazole* (Gantrisin) is capable of high urinary concentration without risk of crystalluria, and is of low toxicity. It is capable of controlling many *Esch. coli* infections and some strains of *proteus* and *Ps. aeruginosa*. The dose is 2 g *statim* followed by 1 g six-hourly until the temperature has remained normal for three days, and cultures of the urine have proved sterile on three consecutive occasions.

Sulphamethiazole (Urolucosil) has similar properties, but unlike other sulphonamides it is unnecessary to insist on a high fluid intake while the patient is taking this drug. The dose is 100 mg four times daily.

Antibacterial Nitrofuran: *Nitrofurantoin* (Furadantin) is a synthetic antibacterial agent. Although when taken by mouth the blood levels remain low, the concentration in the urine is significant; hence it is of value only in genito-urinary infections. It is particularly effective against *Esch. coli*, *A. aerogenes* and *proteus*; often it has proved lethal to antibiotic-resistant strains of these organisms. The average dose by mouth is 5 to 8 mg per kg of body weight. One-quarter of this dose is administered immediately after food three times daily and a further dose with cold milk last thing at night. If nausea or vomiting occurs, the dose must be reduced.

Antibiotics: *Broad Spectrum Antibiotics.*—While these are effective if employed in accordance with bacteriological findings, when it is necessary to continue treatment for a prolonged period (which is not unusual) a number of dangers loom large. Firstly, there are the toxic properties, producing nausea, vomiting, and diarrhœa. Secondly, there is the alteration in the intestinal micro-flora these antibiotics produce, sometimes leading to moniliasis or staphylococcal enterocolitis (p. 18). Thirdly, the frequency with which resistant variants emerge, often in a comparatively short time, is disturbing.

CHRONIC PYELONEPHRITIS

Chronic pyelonephritis is an important, common, and dangerous disease. It is the most frequent cause of death from uræmia. Like its acute counterpart it can be primary, or secondary to abnormality of the urinary tract.

Lawrence Paul Garrod, 1895-1979. Professor of Bacteriology, St. Bartholomew's Hospital, London.
Theodor Albrecht Edwin Klebs, 1834-1913. Professor of Bacteriology successively at Prague, Zurich and Rush Medical College, Chicago, Illinois, U.S.A.

Pathology.—The inflammation is interstitial, and microscopically scars resulting from the destruction of adjacent parenchyma can be seen. These scars are unevenly distributed and nearly always are more evident in one kidney than in the other. Histologically it can be seen that the brunt of the onslaught has been borne by the renal tubules; they are atrophic, often dilated, and sometimes cystic. In time many of them are destroyed and disappear in scar tissue. On the other hand, the glomeruli retain their normal structure until late in the disease.

Chronic pyelonephritis is almost three times as common in females as it is in males. Two-thirds of the affected females are under forty years of age, while 60 per cent. of the males are over forty.

Clinical Features.—It is possible, but unusual, for chronic pyelonephritis to remain asymptomatic until advanced renal insufficiency appears.

Lumbar pain, dull in character, is present in 60 per cent. of cases.

Increased frequency and dysuria are leading symptoms in most cases.

Hypertension is present in 40 per cent. of cases, being more common in primary than in secondary pyelonephritis. It develops slowly and thus is more in evidence in long-standing cases. In 20 per cent. of cases the hypertension is of the accelerated (' malignant ') type.

Constitutional symptoms comprising lassitude, malaise, anorexia, nausea, and headache constitute the main complaint in 30 per cent. of cases. The disease often remains unrecognised in these sufferers, perhaps for years.

Pyrexia. Attacks of pyrexia exceeding 37·8° C are the main symptoms in 20 per cent. of cases. The need to explain these attacks often leads to a thorough investigation including the urinary tract, and the disease is then brought to light.

Anæmia.—A normochromic anæmia due to unsuspected renal impairment may be the presenting feature.

The Urine. *Proteinuria.*—On account of the late destruction of the glomeruli, contrary to glomerulonephritis, the amount of protein passed in the urine is small (less than 3 g daily).

Casts are found infrequently.

White cells are numerous. A fresh three-hour urine should be examined and a count of over 200,000 per hour is abnormal—often it may be as high as several million. Ureteric specimens may reveal considerable difference in this respect between the kidneys, and on the ability of the individual kidney to concentrate and acidify urine.

Bacteriological Examination of the Urine.—Commonly found are *Esch. coli*, *Streptococcus fæcalis*, *proteus* and *pseudomonas*.

Treatment is not easy. Two principles must be followed: (1) to eradicate predisposing causes such as obstructions or stones, and (2) by repeated cultures and sensitivity tests to control and, it is hoped, ultimately to eradicate the infection with repeated courses of suitable drugs. In secondary cases, even if the cause can be removed, the renal pelves have frequently lost their musculature and are fibrotic and non-contractile. Moreover,

once the parenchyma has been damaged it is a locus of predilection for blood-borne organisms. Consequently chemotherapy and antibiotics confer as a rule only temporary benefit. The longer the infection has persisted, the more difficult is it to eradicate. When prolonged treatment is required, there is a definite place for the following inexpensive drug.

Mandelic acid and its salts are often curative in coliform infections and those caused by streptococcus fæcalis. Ammonium mandelate 2 g (or better, mandamine, which is mandelic acid and hexamine combined), together with a capsule of 1 g ammonium chloride, is given six-hourly. Fluids are restricted to 1·5 litres in the twenty-four hours. It is essential that the urine be kept acid (pH 5·3 or less). Contraindications to this form of therapy are impairment of renal function and infections producing ammoniacal decomposition of the urine.

In nearly 50 per cent. of patients with pyelonephritis due to infection by one organism only, in whom infection persists in spite of treatment with antibacterial drugs, it is found that the original organism has been replaced by another, e.g. *Streptococcus fæcalis*, instead of *Esch. coli*.

It is highly important to eradicate distant foci of infection whenever possible. In occasional cases when the disease is unilateral and associated with the accelerated type of hypertension, nephrectomy is sometimes curative. Partial nephrectomy, removing a badly damaged portion of renal tissue, may be necessary by helping to sterilise the urine and free the patient of pain.

Recurrent Dialysis.—In advanced cases, diminishing renal function may require institution of recurrent dialysis using an artificial kidney. Difficulty in controlling hypertension or renal infection may necessitate bilateral nephrectomy.

Transplantation of a kidney from a donor or a cadaver may be possible (p. 1341).

Investigation of a case of Pyelonephritis

The following investigations are carried out in chronic cases a week or more after treatment with antibacterial drugs, and in acute cases after the infection has subsided with treatment. These investigations are to discover any cause of obstruction to the upper or lower urinary tract, and/or a source of infection, which is present most often in the genital organs or the urethra.

(*a*) *The Clinical Examination is Repeated.*—The kidneys are palpated for tenderness and enlargement, and the bladder for chronic retention of urine. In middle-aged or elderly males the prostate is examined for enlargement or fibrosis. In male children, atresia of the meatus or meatitis is sought. Inspection of the cervix uteri often displays cervicitis or cervical erosion. Examination of the secretion expressed by prostatic massage sometimes accounts for the presence of infection. If no infection is found in these situations, a possible focus in carious teeth or infected tonsils is sought.

(*b*) *Renal Function Tests.*—To assess the glomerular and tubular function (p. 1161).

(*c*) *Excretory Pyelography.*—The plain film of the urinary tract may reveal the presence of a urinary calculus. In chronic pyelonephritis, and after an attack of severe acute or recurring acute pyelonephritis, changes in the pyelographic shadow or shadows will be manifest. Some of these are similar to those of early hydronephrosis, but they are more unequally distributed. Although the calyces become flattened or clubbed, this is more in evidence in one part of the kidney than another. Persistent poor definition in one group of calyces is presumptive evidence of chronic infection. In cases of longer standing the renal pelvis becomes slightly dilated, but, unlike hydronephrosis, the dilatation of the calyces predominates. The most important X-ray sign is narrowing of the cortex, often localised, and giving rise to depressed

scars seen as notches in the outline, or considerable contracture of one pole. Nephrotomography can be very helpful in demonstrating these scars.

(*d*) *Cysto-urethroscopy.*—After the instrument has been passed, residual urine, if any, is measured. Examination of the urethra may show chronic urethritis or the presence of a stricture. In the female urethro-trigonitis is commonly found. Hitherto unconfirmed obstruction to the prostatic urethra by an enlarged middle lobe or contracture of the bladder neck may be demonstrated. Evidence of cystitis is the most common finding. Sometimes the ureteric orifice, or orifices in bilateral cases, is seen pouting, œdematous, or gaping. Delay in efflux is usual on the affected or more affected side. Ureteric catheters are passed and specimens collected from each kidney. These specimens are examined for cells and bacteria. By these means it is ascertained whether the infection is bilateral or unilateral. In male children valves in the posterior urethra, or a contracture of the bladder neck, are sometimes revealed.

(*e*) *Micturating Cine-cystography*, using a cine-camera coupled to an X-ray image intensifier, may reveal reflux of urine up one or both ureters. This can be a transient phenomena due to infection. It is commonly a factor in childhood.

HYPERTENSION AND A UNILATERAL RENAL LESION

Renal ischæmia regularly produces increased arterial tension, as ischæmia of the renal parenchyma leads to the formation of a vasopressor substance. Too often cases are labelled essential hypertension without thorough investigation. Sometimes in a case of hypertension a unilateral diseased kidney is demonstrated and nephrectomy is followed by permanent lowering of the blood pressure. The most amenable lesion in this respect is chronic pyelonephritis with a short history of high blood pressure. Occasionally patients with a renal neoplasm, or renal calculi with infection, have been permanently benefited, in so far as hypertension is concerned, by removing the diseased kidney. Renal artery stenosis is a cause of hypertension (fig. 127). Direct arterial surgery (p. 146 and p. 157) may be curative.

PYONEPHROSIS

The kidney is converted into a multilocular sac containing pus or purulent urine, with a varying amount of renal parenchyma in its walls. A pyonephrosis can result from infection of a hydronephrosis, follow acute pyelonephritis or, most commonly, arise as a complication of renal calculus (calculous pyonephrosis). As a rule the condition is unilateral.

Clinical Features.—The classical triad of pyonephrosis is anæmia, fever, and a loin swelling. When the condition arises as an infection of a hydronephrosis the swelling may be a large one. When the pyonephrosis is open, an evening rise of temperature to 37·2° or 37·7° C is usual; if the pyonephrosis becomes closed, the temperature is higher. Pyuria, when present, gives rise to cystitis, and sometimes symptoms of that condition are the chief complaint.

Investigation.—A plain X-ray may show a calculus. Excretory pyelography shows the same vagaries as advanced hydronephrosis, in that the appearance of the shadow of the dilated renal pelvis and calyces is long delayed and may be absent. Cystoscopy often reveals cystitis with a purulent efflux from the ureter of the affected side. In closed cases the bladder may be normal and a ureteric catheter either becomes arrested, usually at the pelvi-ureteric junction, or passes the obstruction and gives exit to pent-up purulent urine.

Treatment.—Infection in an obstructed hydronephrotic kidney should be treated as an emergency. Antibiotics should be given, and the kidney drained by a large urethral catheter as a nephrostomy tube. In cases known

to be due to stone, the stone should be removed. Where obstruction is known to have destroyed most of the renal substance, and function on the other side is good, nephrectomy should be performed. Should the capsule prove to be densely adherent to the surrounding structures subcapsular nephrectomy (p. 1222) is performed. When a pyonephrosis arises in a single kidney, or the condition is bilateral, permanent nephrostomy may be the best possible measure. Drainage by an 'ileal conduit' to bladder or the skin is another possibility (p. 1262).

CARBUNCLE OF THE KIDNEY

The source of origin of the organism is usually a cutaneous lesion, such as a boil, carbuncle, whitlow, or an abscess of the breast, and the *Staphylococcus aureus* is conveyed to the kidney via the blood-stream. Occasionally a blow on the loin determines the issue, when injury to the kidney produces a small hæmatoma.

Morbid Anatomy.—On splitting open the kidney there is a necrotic mass of tissue, usually localised, involving the parenchyma (fig. 1356).

Clinical Features.—There is an ill-defined, tender swelling in the loin, persistent pyrexia and leucocytosis, signs that closely simulate a perinephric abscess. In early cases the urine contains neither pus cells nor organisms, but after a few days staphylococci can sometimes be isolated. Pyelography often shows compression or obliteration of a group of calyces, an appearance that does not differ from adenocarcinoma of the kidney.

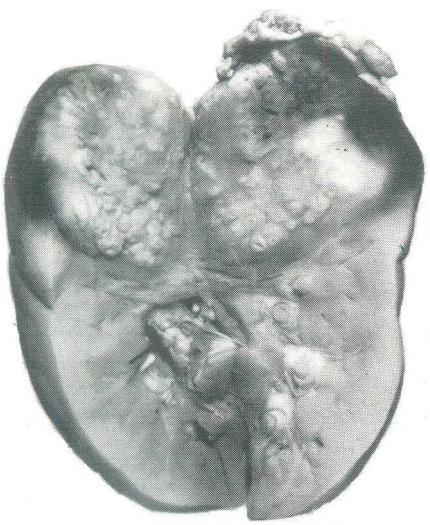


FIG. 1356.—Carbuncle of the kidney.
(J. G. Yates-Bell, F.R.C.S., London.)

Treatment.—Antibiotics have cured some early cases. When the response to antibiotic therapy is not dramatic the kidney must be exposed, the carbuncle incised, and the necrotic material removed. Hæmorrhage is not excessive and can be controlled by pressure with a moist warm pack. A drainage tube is passed down to, but not into, the resultant cavity and the wound is closed around the tube. Antibiotics are continued for at least ten days. Primary nephrectomy is indicated only when the major part of the kidney is involved by the carbuncle.

PERINEPHRIC ABSCESS

The most common causes are depicted in fig. 1357. Other causes are infection of a perirenal hæmatoma, and perforation of the renal capsule from undue delay in operating upon a pyonephrosis or a renal carbuncle. A tuberculous perinephric abscess can arise from an advanced pyonephrosis or from tuberculosis of a nearby vertebra.

Clinical Features.—The classical symptoms and signs of a perinephric abscess are a high, swinging temperature, rigidity, tenderness, and fullness in the loin (fig. 1358). If the suppuration commences in the lower part of the perinephric fat, local signs present early, but when the suppuration is confined to the upper portion of the perinephric fat which lies beneath the lower ribs, it produces no visible swelling, and even rigidity and tenderness may be

absent. The leucocyte count is always raised, often to 20,000/mm^3 (20×10^9/l). As a rule no pus or organisms are present in the urine.

Radiography shows obscuration of the psoas shadow, with one or more of the following additional signs—scoliosis with concavity towards the abscess, and elevation and immobility of the diaphragm on the affected side.

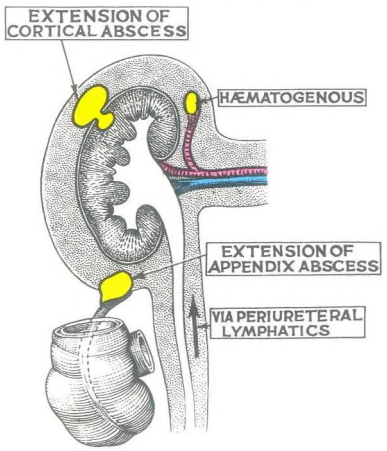


FIG. 1357.—Sources of perinephric abscess.

FIG. 1358.—A large perinephric abscess.

Mathé's Sign.—Absence of the downward displacement that occurs in the erect posture in every normal kidney. Two radiographs, one in the lying posture and one in the erect posture, during excretory pyelography are required. In established cases the kidney can be shown to be displaced forwards if a lateral view is obtained.

Treatment.—Under antibiotic cover a lumbar incision is made large enough to enable the surgeon to open up pockets both above and below the kidney. The surface of the kidney is palpated for an unruptured cortical abscess, which, if present, should be incised. A specimen of pus having been obtained for bacteriological examination, the wound is closed with ample drainage. Appropriate antibiotic treatment follows.

RENAL TUBERCULOSIS

Ætiology and Pathology.—Tuberculosis of the urinary tract cannot arise except as a blood-borne infection from a distant focus, which, in many cases, is impossible to locate.

When clinically recognisable, in the great majority of cases renal tuberculosis is apparently confined to one kidney. A group of microscopical lesions coalesce and discharge pus and tubercle bacilli along one set of tubules, and the pyramid which gives exit to the discharge becomes ulcerated (fig. 1359 A).

Course : *Tuberculous bacilluria* is a convenient term to designate a very early lesion of the renal cortex when no abnormality in the affected kidney can be detected, even by retrograde pyelography. It indicates the presence of active tuberculosis disease which may progress, and as such must be treated in the same way as the demonstrable lesions.

Charles Pierre Louis Mathé, Contemporary. Formerly Urologist-in-Chief, St. Mary's Hospital, San Francisco, California, U.S.A.

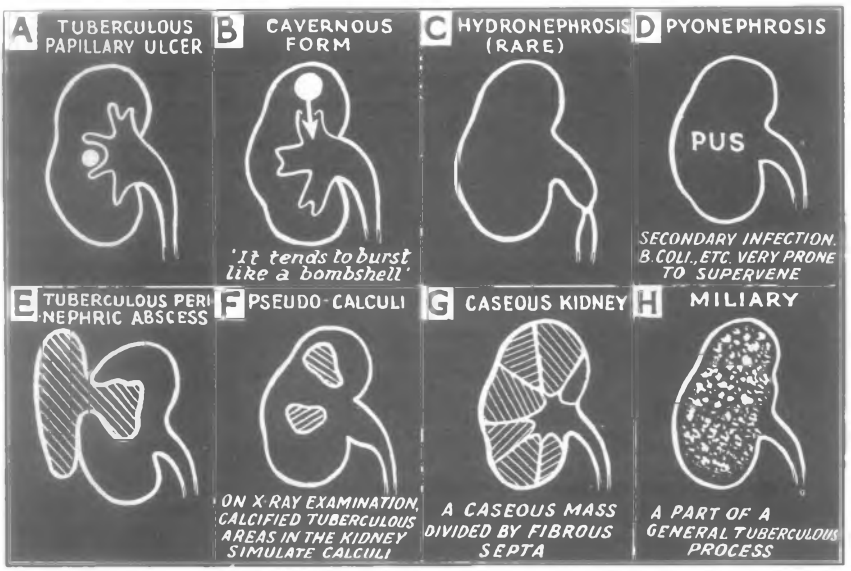

A TUBERCULOUS PAPILLARY ULCER
B CAVERNOUS FORM
'It tends to burst like a bombshell'
C HYDRONEPHROSIS (RARE)
D PYONEPHROSIS
PUS
SECONDARY INFECTION. B. COLI, ETC. VERY PRONE TO SUPERVENE
E TUBERCULOUS PERI-NEPHRIC ABSCESS
F PSEUDO-CALCULI
ON X-RAY EXAMINATION CALCIFIED TUBERCULOUS AREAS IN THE KIDNEY SIMULATE CALCULI
G CASEOUS KIDNEY
A CASEOUS MASS DIVIDED BY FIBROUS SEPTA
H MILIARY
A PART OF A GENERAL TUBERCULOUS PROCESS

FIG. 1359.—Blackboard sketches of types of tuberculous kidney (macroscopic pathology).[1]

A macroscopical lesion progresses rather slowly, and other similar lesions appear in the same kidney. The infection can be confined to the affected kidney for months or years, causing one or other of the changes depicted in fig. 1359 C to G. More often the disease spreads along the ureter (tuberculous ureteritis) to give rise to tuberculous cystitis in both sexes and prostatitis, vesiculitis and later epididymitis in the male.

In the male, renal and genital tuberculosis can be associated before the bladder and posterior urethra are involved, in which event the genital infection probably occurs in the following way. An urgent desire to micturate causes the internal sphincter to open, while at the same time, because there are no immediate facilities for emptying the bladder, voluntary closure of the external sphincter is called into play. At that moment infected urine is forced under pressure into the mucosal crypts of the prostate and the common ejaculatory ducts (Wells).

Clinical Features.—Renal tuberculosis usually occurs between twenty and forty years of age. Men are affected twice as commonly as women, and the right kidney somewhat more commonly than the left.

Frequency.—The earliest symptom is increased frequency of micturition. Often this is the only symptom. It is progressive, the patient complaining that (over a period of months) the frequency has increased both by day and by night. Frequency is occasionally due to polyuria as the result of the renal tubular inflammation: it is usually due to patchy tuberculous cystitis.

Pyuria.—In early cases the urine is pale and slightly opalescent. The presence of pus cells without organisms in an acid urine is very suggestive of tuberculous urinary infection (Bacteriological examination, p. 1201).

[1] G is known also as the ' putty kidney ' and by the French as the ' cement kidney '. It is nature's method of performing (ineffective!) autonephrectomy.

Charles Alexander Wells, Contemporary. Professor Emeritus of Surgery, Liverpool, England.

Painful Micturition.—Once tuberculous cystitis has set in, micturition becomes increasingly painful. First there is suprapubic pain if the bladder cannot be emptied immediately; later a burning pain accompanies micturition, and when secondary infection has occurred there is superadded agonising pain after micturition referred to the tip of the penis or to the vulva, often associated with terminal hæmaturia (strangury).

Renal pain is often absent, but sometimes the patient complains of a dull ache in the loin.

Hæmaturia.—In 5 per cent. of cases the disease is ushered in with painless hæmaturia occurring from an ulcer situated on a renal papilla. During the hæmorrhage, and after it has ceased, investigations may or may not demonstrate the presence of a renal lesion and tubercle bacilli in the urine. In the latter event more typical symptoms commence some months later. Apart from a few drops of blood at the end of micturition in cases complicated by severe cystitis, macroscopical hæmaturia occurs only occasionally.

A tuberculous kidney is œdematous and friable, and is therefore more likely to be damaged by trauma than is the normal pliable kidney.

Constitutional Symptoms.—A continuous slight loss of weight is usual. Evening pyrexia to 37·3° C occurs when the disease is fully established. A high temperature is indicative of a secondary infection or disseminating tuberculosis.

On Examination.—It is unusual for a tuberculous kidney to be palpable.

When a patient with renal tuberculosis has an enlarged kidney which can be felt, it is by no means certain that this kidney is the one that is diseased, for compensatory hypertrophy sometimes renders the healthy kidney both large and tender.

The prostate, vesicles, each vas, and epididymis should be examined for nodules or thickening, which may indicate tuberculous invasion.

Investigation :

Bacteriological Examination of the Urine.—Prompt examination must be made of an early morning specimen before contaminants can overgrow. When, after staining a specimen of the sediment with Ziehl-Neelsen's stain, acid-fast bacilli are found, it is highly probable that they are tubercle bacilli, but the proof lies in culture or guinea-pig inoculation, either of which is reliable. **Most important—specific therapy should never be started until a positive culture has been obtained.** If this precaution is neglected, an exacting course of treatment may be started for a non-tuberculous condition, and at a later stage it is impossible to prove or disprove the diagnosis.

In few fields of medicine is the diagnosis more exact. The microscope demonstrates tubercle bacilli in 72%, the guinea-pig test in 94%, and the culture medium in 98% of cases.

A Plain X-ray may show calcification in healing foci. In gross disease this may be a rim around a caseous mass, or complete dense spheres. In minimal lesions specks of calcification may be scattered in one part of the cortex or pyramid. Ureteric calcification is late except at the lower end, and here, in males, the vesicles may also show patchy calcification.

Franz Heinrich Paul Ziehl, 1857–1926. Neurologist, Lübeck, Germany.
Freidrich Carl Adolf Neelsen, 1854–1894. Prosector, Stadt-Krankenhaus, Dresden, Germany.

Excretory pyelography (fig. 1360).

Changes in the Pyelogram commence with loss or dysfunction of one or more calyces, and irregular density in the nephrogram. Spasm or œdema may obliterate a calyceal neck, and if this becomes severe a hydrocalyx will develop. Complete obstruction leads to an abscess displacing adjacent calyces and the appearance of a space-occupying lesion. The bladder tends to become round and spastic when involved, and ulceration may produce spasm and flattening of the bladder in this area. Ureteric fibrosis leads to loss of the natural curves of the ureter, and a wider, straighter and shorter line between kidney and bladder.

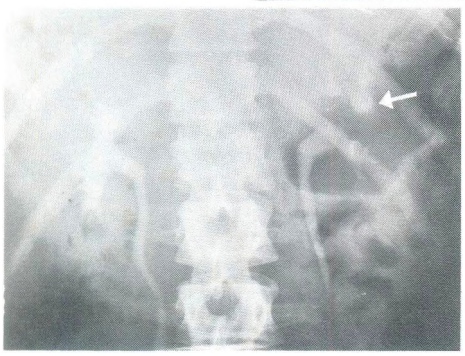


FIG. 1360.—Excretory pyelogram showing a small localised lesion with hydrocalyx. Healing took place under conservative treatment. (*Professor E. Wildbolz, Berne.*)

In late cases, the contralateral ureter becomes dilated also; this is not necessarily an indication of disease on the opposite side, for the dilatation may be due to the cicatrised bladder causing obstruction to the ureter in its intramural course.

Cystoscopy.—When increased frequency is the only symptom and it has not been present long, the bladder and the ureteric orifices are normal.

A unilateral cloudy efflux is sometimes observed. Either more frequent efflux or a delay in excretion of indigo-carmine is indicative of the side that is diseased. In 10 per cent. of cases there is an absence of efflux on the affected side, and no pathological changes in the bladder. These are examples of a closed ureter leading to so-called autonephrectomy, or a pyonephrosis.

Œdema causes pallor around a ureteric orifice and is the earliest cystoscopic sign of vesical involvement. Infiltration follows, and the mucous membrane of the affected ureteric orifice pouts. Tubercles appear, usually lateral to the ureteric orifice, and later in the dome of the same half of the bladder. By confluence of a circumscribed group of tubercles, a tuberculous ulcer, which has an irregular periphery, may form. In long-standing cases, as a result of sclerosing peri-ureteritis, which causes shortening of the ureter, the ureteric orifice becomes displaced upwards and its mouth remains open (the so-called ' golf-hole ' ureteric orifice). As the disease progresses, the capacity of the bladder steadily diminishes. At any stage, if a secondary infection supervenes, the intense generalised inflammation of the bladder wall obliterates many of the characteristic features of urinary tuberculosis just described above.

Retrograde pyelography may be necessary when investigating a suspicious pyuria if renal function is poor and tubercle bacilli have not been seen in the Ziehl-Neelsen film. Appearances, such as shagginess of a calyx or an abscess of the parenchyma connected to a calyx by a narrow neck, are often displayed by this method when excretory pyelograms appear normal. Retrograde pyelography is also of great assistance

in cases of unilateral renal tuberculosis too advanced to concentrate dye given intravenously (fig. 1361). Remember, however, that the diagnosis depends on finding bacilli and *not* on the pyelographic appearances.

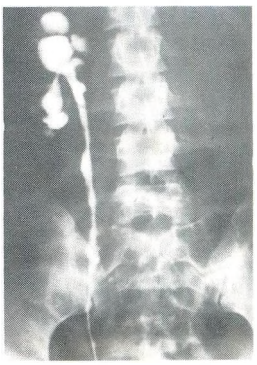


FIG. 1361. — Retrograde pyelogram showing advanced tuberculosis of the right kidney and ureter.

Examination of the Lungs.—A radiograph is necessary to exclude active or previous pulmonary tuberculosis.

Treatment.—(See p. 37). It is desirable to have the patient under observation when commencing treatment. The liability to serious side-effects from the drugs used will usually become obvious within the first three weeks.

The drug regimes used in the treatment of renal tuberculosis have depended to some extent on the cost and availability of the drugs, and the difficulty in some parts of the world in keeping contact with the patients to ensure that treatment regimes are followed through. Short courses of effective drugs are much the best in such a situation. Bacteriocidal drugs, Rifampicin, INAH, Pyrazinamide are the drugs of choice. The first two months should be with Rifampicin 600 mg, INAH 300 mg, Pyrazinamide 1 g daily, and the next four months with Rifampicin and INAH only. The Rifampicin kills the actively dividing cells, and the Pyrazinamide is effective against dormant bacilli in the macrophages. Pyrazinamide is hepatotoxic if 2 g or more is given. Streptomycin, due to side effects and the need to give it by injection, has largely been superceded.

Prognosis in renal tuberculosis has improved enormously since the advent of chemotherapy and antibiotic treatment, and if the regimen detailed is carried out properly there should be a very low incidence of recrudescence of infection.

Operative Treatment.—Some lesions are surrounded by so much necrotic and fibrous tissue that antibiotic agents do not reach the organisms in sufficient concentration to kill them. Unilateral lesions of this character, and those in which the disease is more advanced, call for operative treatment, the optimum time for which is after six to twelve weeks of antibiotic treatment.

According to circumstances one of several operations is carried out. It must be emphasised that conservative surgery should be the aim, and several conservative procedures may prove necessary on one or both kidneys and ureters during the course of the disease. The fact that a horseshoe kidney is involved does not alter this principle, neither does the fact that one kidney may have been removed already.

1. **Renal Cavernotomy** (Hanley).—As the primary lesion of the calyx heals (fig. 1359 A and B) fibrosis tends to cause a stricture at the neck of the calyx. Radiological disappearance of such a cavity should be viewed with suspicion, and usually indicates that there is a closed pyocalyx which remains infected, as its avascularity prevents the drugs from reaching the tubercle bacilli present. Sometimes such a pyocalyx will re-open and discharge its contents some six months after starting treatment, but if this does not occur after a year and a calyx is radiologically ' missing ', a drainage operation must be undertaken.

Howard Granville Hanley, Contemporary. Consulting Surgeon, St. Paul's Hospital, London.

The kidney is approached as described on p. 1221 and after clearing the perinephric fat, the abscess is aspirated with a syringe and wide-bore needle. The roof of the abscess is removed with scissors and its caseous contents swabbed out until healthy granulation tissue is reached. Any bleeding from the cut edges is controlled with fine catgut stitches (fig. 1362). The wound is closed without drainage. As the cavity is closed off from the pelvis (if it is not, the operation is unnecessary), there is no leakage of urine. Multiple abscesses in both kidneys can be dealt with in this way.

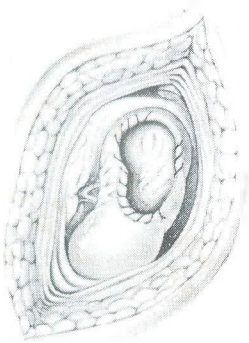


FIG. 1362.—Renal cavernotomy completed. (*After H. G. Hanley, London.*)

2. **Nephro-ureterectomy.**—The indication is a unilateral lesion with gross impairment of renal function on the affected side.

In such advanced cases the ureter as well as the kidney usually proves to be diseased, and nephro-ureterectomy (fig. 1363), in which, through a separate incision, the ureter is divided and ligated at its entrance to the bladder before commencing lumbar nephrectomy, is the operation of choice. By total removal of the ureter a possible source of continued infection of the bladder is removed, and the incidence of post-operative wound infection and sinus formation is reduced.

3. **For Hydronephrosis.**—This is due to tuberculous cicatricial contracture and healing of the bladder around the lower end of the ureter or at the pelvi-ureteric junction. A procedure similar to the Anderson-Hynes operation (p. 1189) should be performed.

(*a*) (*b*)

FIG. 1363 (*a* and *b*).—Nephro-ureterectomy. Tuberculous pyonephrosis.

4. **For Systolic bladder** (*syn.* '**thimble bladder**' (fig. 1402)).—This is due to healing by fibrosis and often leads to extreme frequency of micturition. The best operation is the type of ileo-cystoplasty, described by Hanley, in which a 15 cm opened-out loop of terminal ileum is sutured to the bladder, from which the dome has been removed to provide a maximum opening (fig. 1403).

ABACTERIAL PYURIA

That sterile pyuria indicates urinary tuberculosis is almost axiomatic. Nevertheless, a small group of cases of abacterial pyuria is definitely non-tuberculous. The symptoms simulate renal tuberculosis very closely, and it is only when repeated examinations of the urine, and in the male the seminal fluid, fail to reveal *mycobacterium tuberculosis* or other bacteria, and cultures and guinea-pig inoculations are negative, that the diagnosis can be made. Pyelography occasionally shows slight dilatation of the renal pelvis and ureter of one or both sides; more usually this examination is negative. Cystoscopy reveals a severe cystitis and often diminished bladder capacity. Occasionally amœbiasis is responsible.

Treatment.—Aureomycin usually cures the condition in three days. In cases resistant to this treatment 0·3 g of neoarsphenamine intravenously weekly for four weeks is often successful. In rare instances, when amœbiasis is responsible, treatment with emetine hydrochloride will eradicate the infection (p. 1010).

NEOPLASMS OF THE KIDNEY

Benign Neoplasms

Adenoma.—Pea-like cortical adenomas are sometimes found at necropsy. They give rise to no symptoms, and are only of academic interest.

Angioma may give rise to profuse hæmaturia.

Truly benign tumours of the kidney are so rare that a good rule is that *all neoplasms of the kidney which can be recognised clinically should be considered malignant and treated as such.*

Malignant Neoplasms

Clinically neoplasms of the kidney are divided into two classes:

In children between the ages of one and seven.

In adults after the age of forty.

Between the ages of seven and forty malignant neoplasms are unusual.

RENAL NEOPLASMS IN CHILDREN

Wilms's tumour (*syn.* nephroblastoma) is a mixed tumour containing epithelial and connective tissue elements arising from embryonic nephrogenic tissue, situated originally in one or other pole of the kidney. In infancy such tumours are very occasionally bilateral. Far more common is a unilateral tumour. They usually occur during the first four years of life. Cases arising under one year seem to have a much better prognosis.

Pathology. — *Macroscopically.* — On section the neoplasm is a greyish-white or pinkish-white colour; its consistence varies with the rapidity of the growth, i.e. the more rapid the growth, the softer the tumour (fig. 1364).

FIG. 1364.—Wilms's tumour.

Max Wilms 1867–1918. Professor of Surgery, Heidelberg, Germany.

Microscopically it is composed mainly of two types of cells—epithelial and connective-tissue. Cartilage, bone, and smooth or striped muscle fibres are occasional findings. The epithelial and connective-tissue cells exist side by side, but one type is usually predominant. Thus the tumour is composed of mixed cellular elements, some of which are radio-sensitive and some radio-resistant. Consequently, the radio-resistant elements show a continuance of activity in spite of radiotherapy.

Clinical Features:

Abdominal Tumour.—The symptomatology is always the same. An abdominal tumour appears and progresses rapidly, while the general health deteriorates. Examination of the abdomen reveals a mass which may be enormous; the bulk of the tumour is on one side of the abdomen. Wilms's tumour tends to grow within a capsule, pushing the rest of the kidney aside; thus the reniform shape of the kidney is lost early.

Pyrexia.—Half these patients have some elevation of temperature, which disappears if the tumour is removed.

Hæmaturia is a sign of ill omen. It denotes that the previously encapsulated tumour has burst into the renal pelvis, and all patients suffering from a Wilms's tumour with this sign die within nine months (Scott).

Pyelography shows gross deformity of some of the calyces.

Metastases.—Wilms's tumour metastasises early, mainly by the blood-stream to the lungs, less commonly to the liver, rarely to bones, and exceptionally to the brain. Lymphatic dissemination is much less common.

Differential Diagnosis.—From a practical standpoint there is but one swelling with which Wilms's tumour can be confused, and that is retroperitoneal neuroblastoma, but as the treatment is precisely the same such differentiation is an academic exercise. However, if radiography reveals bone metastases, this distinctly favours a neuroblastoma. Although these abdominal tumours arouse considerable diagnostic interest, for fear of accelerating dissemination, multiple examinations by, for instance, a class of students, must be forbidden.

Treatment.—*Immediate nephrectomy and post-operative radiotherapy.* The earlier nephrectomy can be carried out, the better. A few hours may be necessary to correct anæmia by transfusion. Radiotherapy should be commenced immediately after operation, and 200 rad given daily to a total of 4,000 to 4,500 rad.

Prognosis.—Under the age of one year 80 per cent. survive five years, over this age it drops to 30 per cent. Recurrences usually occur within a year, so a child surviving eighteen months is probably cured.

Bilateral Cases and Inoperable Cases.—Radiotherapy should be given to the renal areas. There is some hope that chemotherapeutic agents and particularly actinomycin D may prove of value in these cases.

RENAL NEOPLASMS IN ADULTS

1. **Hypernephroma** (*syn.* Grawitz tumour) is an adenocarcinoma and is the commonest neoplasm of the kidney (75 per cent). It arises in the cortex, possibly from a pre-existing adenoma, probably *per primam* in cells of the uriniferous tubules.

Lyall Stuart Scott, Contemporary. Surgeon, Urology Department, Victoria Infirmary, Glasgow, Scotland.
Paul Albert Grawitz, 1850–1932. Professor of Pathology, Greifswald, Germany.

Pathology.—A tumour of moderate size is spherical in shape, and it often occupies one or other pole, the seat of election being the upper pole; less often it is in the central portion of the kidney. On section it is characteristically yellow (due to lipoid); less often it is dull white, or semi-transparent. Hæmorrhagic areas are often seen. The tumour is divided into numerous lobules by fibrous septa (fig. 1365). The larger the tumour the more extensive is central hæmorrhage and necrosis.

FIG. 1365.—Adeno-carcinoma of the kidney.

Microscopical Structure.—The most common appearance is solid alveoli of cubical or polyhedral clear cells, with deeply stained small rounded nuclei and abundant cytoplasm containing lipoids, cholesterol, and glycogen. The cells may also be arranged in the form of papillary cysts or tubules. In a much smaller percentage the cells are granular (dark). Clear and dark cells can co-exist in different parts of the same tumour. In all cases the stroma is scanty but rich in large blood-vessels, the walls of which often appear, in places, to consist of tumour cells.

Spread.—As the tumour enlarges, it encroaches upon a group of calyces and is prone to grow into the renal veins. Pieces of growth becoming detached are swept into the circulation, to become arrested particularly in the lungs to produce 'cannon ball' secondary deposits (fig. 1368), and in bones. Occasionally, when a secondary growth appears in a long bone, it remains the only metastasis for perhaps a year or more.

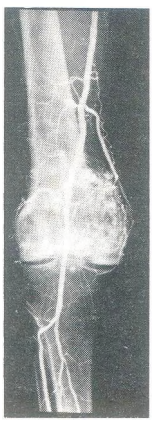


FIG. 1366.—Arteriogram showing vascular 'blush' due to a metastasis from a Grawitz tumour.

While spread by the blood-stream predominates, spread by lymphatics occurs also. If the tumour bursts through the renal capsule into the perirenal tissues, it then metastasises to the lymph nodes in relation to the hilum of the kidney, and from there to the para-aortic and other lymph nodes.

Clinical Features.—Men are more often affected than women, the ratio being 2 : 1. Typically the first sign is intermittent hæmaturia. Clot colic may follow. Dragging pain in the loin is sometimes a leading symptom. Occasionally the first manifestation is a palpable renal swelling. In the adult male a rapidly developing varicocele is a rare but impressive sign.[1]

Atypical Cases.—(*a*) In 25 per cent. of cases the primary growth remains 'silent', the patient presenting because of secondary deposits such as a painful enlargement of a bone (fig. 1366), a spontaneous fracture, persistent cough, or hæmoptysis.

(*b*) Occasionally persistent pyrexia (37·8°–38·9° C) is the only symptom, there being no infection to account for the temperature. It is presumably

[1] A left-sided varicocele is more likely, as the left spermatic vein joins the left renal vein.

due to absorption of blood and necrotic material. Such pyrexia usually disappears after nephrectomy: should it persist metastases are present.

(*c*) In a small group of cases the patient presents on account of lassitude and is found to be extremely anæmic, and anæmia being out of proportion to that which could be expected from the hæmaturia, if such be present.

(*d*) 'Polycythæmia' occurs in 4 per cent. of cases. The sedimentation rate is *always raised* above the 1 to 2 mm found in idiopathic polycythæmia vera. The blood count will return to normal after nephrectomy, but a return of the polycythæmia has been known with development of metastases.

(*e*) A nephrotic syndrome has been the presenting feature in some cases of hypernephroma.

(*f*) Endocrine disturbances. Renin secreting tumours producing hypertension have been recorded.

Early Diagnosis.—By the time a patient has the classical triad of symptoms, viz. hæmaturia, pain, and a palpable renal tumour, he nearly always has metastases. It is therefore of paramount importance to endeavour to make an early diagnosis, and any one of these symptoms calls for a thorough renal investigation. Patients with hæmaturia should be examined cystoscopically while the bleeding is in progress. If blood is seen issuing from one ureteric orifice, and there is a clear efflux from the other, the information gained is of considerable importance, but is by no means conclusive evidence of a renal neoplasm. Extremely suggestive is bleeding occurring from a palpable kidney. When blood is not seen issuing from a ureter, but an enlarged middle lobe of the prostate or a papilloma of the bladder is discovered, it must not be assumed that the source of the hæmorrhage has been found. Investigation must proceed until each kidney has been proved either to be normal or culpable.

Pyelography.—The early diagnosis rests almost entirely on pyelography.

Excretory pyelography may be inconclusive in early cases, because of lack of precise definition, and in late cases because there is poor or absent concentration of the medium, but at all times it is of great value in determining the function of the contralateral kidney. The presence of a projection in the cortical outline is important.

Tomography during the nephrogram phase can be helpful in outlining the periphery and may distinguish cyst and tumour.

Retrograde pyelography is often required because of the better delineation it affords. The principal pyelographic changes are:

(*a*) Filling defects due to invasion of one or more of the minor or major calyces. Very characteristic is failure of the medium to enter one major calyx.

(*b*) Elongation and compression of one or more calyces and sometimes of the renal pelvis causing the 'spider-leg' deformity (fig. 1367). In congenital cystic kidneys the 'spider legs' are broader, more clearly cut, and involve most if not all of the calyces; also, the condition is bilateral.

(*c*) Displacement of the renal pelvis and the distorted calyces downwards

in growths occupying the upper pole. Growths of the lower pole often displace the ureter inwards.

(*d*) In advanced cases (which may give no shadow on excretory pyelography) the interior of the calyces and renal pelvis are so encroached upon that they are represented by a few, irregular, widely separated shadows.

Chest X-ray.—A radiograph of the thorax should always be taken before deciding the best course of treatment. It is not rare for a hitherto un-

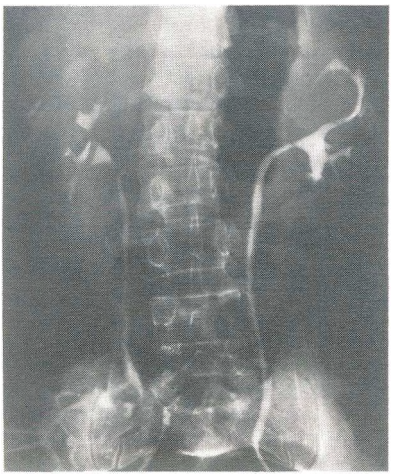


FIG. 1367.—Pyelogram in a case of hypernephroma of the left kidney. The only symptom was one attack of painless hæmaturia.

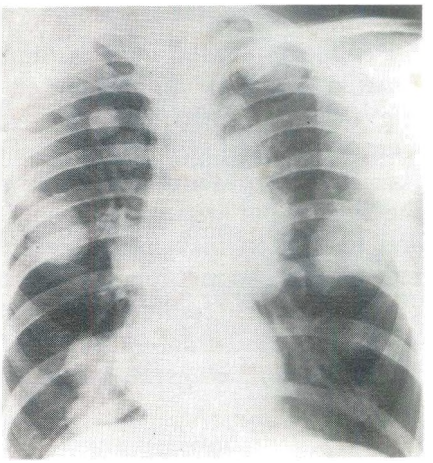


FIG. 1368.—Cannon ball secondaries from a hypernephroma.

suspected metastasis or metastases in a lung to be revealed in this way (fig. 1368).

Aortography by the retrograde route (fig. 1306), will show an avascular area in the case of cyst, and abnormal vessels and pooling in the case of tumour.

Bone Scans should, if possible, be carried out as this may reveal metastases which fail to show on X-ray.

Differential Diagnosis.—(*a*) Hæmorrhage into a hydronephrosis, (*b*) congenital cystic kidney with hæmaturia, or (*c*) a solitary cyst of the kidney can closely simulate a renal neoplasm, or (*d*) aneurysm of a renal artery.

Treatment.—In cases without metastases the treatment is nephrectomy with removal of the perinephric fat.

The incision is a matter of personal preference, the objective being to gain wide exposure of the enlarged kidney and especially of its pedicle. The abdominal approach (p. 1222) is best for accomplishing the following manœuvre.

Preliminary Division of the Renal Pedicle.—It is advisable to deal with the renal pedicle before manipulating the kidney, otherwise blood-borne metastases may occur as a direct result of the operation. Before undertaking ligation and division of the renal pedicle the renal vein and vena cava should be palpated delicately between the finger and thumb for a tumour-thrombus which, if present, calls for opening the vein and extracting the thrombus.

In other respects the details of the operation are those described on p. 1221.

When an extremely large tumour is encountered a paramedian incision is easily enlarged by a transverse cut.

Radiotherapy.—In general terms these tumours are resistant to irradiation, and some series suggest that post-operative radiotherapy makes things worse.

Infarction.—In large masses preliminary deliberate infarction (embolism) of the kidney at the time of aortography, by means of selective renal artery catheterisation and the injection of polystyrene granules or small metal springs, can reduce the size considerably, and make nephrectomy performed 4–7 days later much easier.

Drug Treatment.—There is some suggestion that preliminary infarction of the kidney enhances the immune response and improves the response to drug treatment (vinblastine and progesterone) of the secondary deposits existing at the time of presentation.

Prognosis.—Even some of the largest neoplasms have been followed by a permanent cure. In operable cases 44% of the patients are alive and well after three years, and 30% after five years. If there is macroscopic involvement of the renal vein or its branches, the prognosis is poor.

2. **Papillary Tumours of the renal pelvis** (fig. 1369) resemble those in the bladder but are less common. They tend to invade the kidney proper, and to take on malignant characteristics. Spread downwards by seeding may give rise to multiple ureteric and bladder tumours, but a diffuse metaplasia may be the result of some carcinogenic agents in the urine (p. 1251).

Clinical Features.—Hæmaturia is often continued for months at a time. A renal swelling is absent, except in rare cases where the pelvi-ureteric junction

FIG. 1369.—Papillary tumour of the renal pelvis.

FIG. 1370. — Pyelogram showing a filling defect of the renal pelvis due to papilloma.

becomes occluded and a hydronephrosis results, in which event there is lumbar pain. Clot colic may occur.

Pyelography reveals a characteristic filling defect of the renal pelvis (fig. 1370).

Treatment.—Nephrectomy with extended ureterectomy is the correct

treatment. The intramural portion of the ureter along with the ureteric orifice must be excised in continuity by sleeve resection of the bladder wall.

3. **Squamous-celled carcinoma of the renal pelvis** is the least common variety of malignant disease of the kidney. In some instances it appears to be preceded by leukoplakia, and in most cases a stone or stones are present.

Unless the stone gives rise to symptoms, this is the most elusive of all renal neoplasms, for it causes indefinite abdominal pain which finally becomes localised in the region of the involved kidney. Hæmaturia is slight, or to be found only on microscopical examination of the urine. Excretory pyelography shows a kidney excreting little or no medium. Retrograde pyelography sometimes reveals a deformed renal pelvis. Owing to diagnostic difficulties, by the time nephrectomy is performed there are often widespread metastases that are radio-resistant. This neoplasm has the worst prognosis of any renal neoplasm.

Primary neoplasms of the ureter are rare, and similar to those of the renal pelvis. The symptoms are identical with those of a neoplasm of the renal pelvis, although hydronephrosis, hæmonephrosis, and pyonephrosis are more frequent accompaniments. Occasionally a portion of a papillomatous growth can be seen protruding from a ureteric orifice. Usually the diagnosis is made by pyelography.

Papillary tumours of the ureter should be treated by nephro-ureterectomy, as the epithelium frequently shows changes of carcinoma in situ at many sites. The intra-mural ureter should be removed, or the epithelium destroyed by fulguration. Only rarely is it justifiable, because of reduced renal reserve, to perform resection of the ureter, and replacement by an ileal conduit viz.——————————————→

On the whole, the prognosis of tumours of the ureter is very poor, largely on account of late diagnosis.

EXPOSURE AND REMOVAL OF THE KIDNEY

The kidney lies in a deep recess beneath the rigid bulwarks of the thoracic cage and, having regard to the possibility of a short renal pedicle, no single incision will meet all requirements.

The Oblique Lumbar Incision (Morris).—The patient is placed in the lateral 'kidney' position (fig. 1371), and is held in this position by a broad band of adhesive strapping, which is fixed to one side of the table and passed over the pelvis to the other side. The arm-rest also steadies the patient and relieves the chest of the weight of the arm, which would otherwise embarrass respiration. Morris's classical incision commences 1·25 cm below and lateral to the angle made by the twelfth rib with the outer border of the sacrospinalis, and it passes downwards and outwards towards the anterior superior iliac spine (fig. 1372) and all muscular layers are incised until the extraperitoneal fat is reached. An alternative and more favoured approach is to make the incision over the 12th rib and then either to resect the rib or, by displacing the upper half of the periosteum, to 'drop' it into the lower part of the wound. Care must be taken when removing periosteum not to open the pleural cavity. The pleura will be seen lying on the diaphragm after removing the rib. The extraperitoneal fat has a free posterior border and this is displaced forwards to display the perinephric fascia, which is then incised to reveal the perinephric fat surrounding the kidney. To perform nephrectomy the kidney is lifted from the posterior leaf of the perinephric fat, the fingers of one hand being inserted behind the

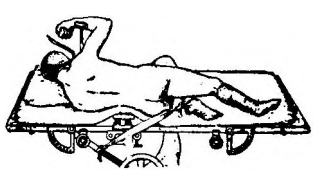


FIG. 1371.—Position on the operating table for exposure of the kidney by Morris's incision.

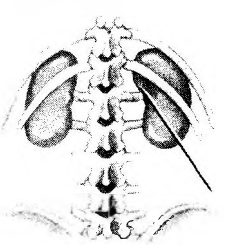


FIG. 1372.—Morris's incision.

Sir Henry Morris, 1844–1926. Surgeon, Middlesex Hospital, London.

pelvis and the vessels. Fat and fascia are dissected away from these structures as they lie on the operator's fingers. The vessels are ligated individually and the ureter is divided at a convenient site. The wound is closed in layers with a corrugated drain placed beside the pedicle.

If the pleura has been breached, the anæsthetist should expand the lung fully before the pleura is closed, at the end of the operation. An X-ray should be taken to check that the lung is re-expanded. Any residual pneumothorax may need to be aspirated.

Transabdominal nephrectomy is frequently employed for the removal of renal tumours, as it gives an unrivalled approach to the renal vessels which must be tied before the tumour is handled, in order to prevent cells being displaced into the circulation by the manipulations. A transverse or paramedian incision is used.

Subcapsular Nephrectomy.—In order to prevent damage to densely adherent structures such as the duodenum, colon, and spleen, the kidney may be removed by incising the capsule along the convex border and dissecting it free to the hilum, where it is again incised to allow access to the renal pedicle.

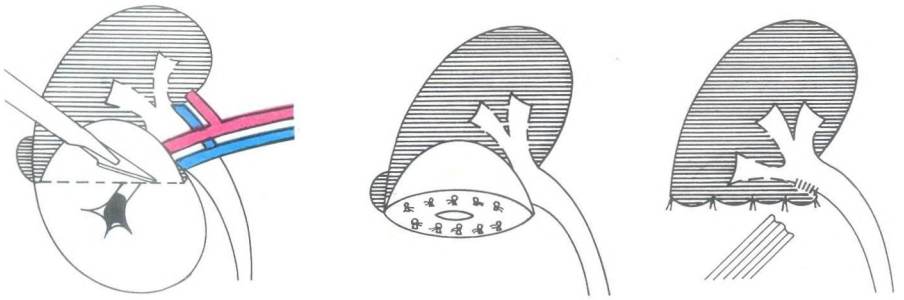


FIG. 1373.—(A) Capsule preserved and reflected; clean cut across parenchyma. Isolation of the affected calyx. (B) Hæmostasis by suture of individual vessels. (C) No attempt to approximate the cut surface of the renal parenchyma, just cover the surface with the capsule.

Partial Nephrectomy (Semb).—A wide lumbar exposure with resection of the twelfth rib is required. Bleeding is controlled by compression of the appropriate branches of the renal vessels. The stages of the excision are shown in fig. 1373.

Carl Boye Semb, 1895–1971. (*See footnote, p. 748*).

CHAPTER 57

THE URINARY BLADDER

SURGICAL ANATOMY AND PHYSIOLOGY

Surgical Anatomy.—Possessing an anterior, superior and posterior surface and being lined by transitional epithelium, the average bladder is capable of holding between 400 and 500 ml. of urine without over-distension. The bladder wall is made up of a muscular coat of unstriated muscle, known as the **detrusor muscle**, the fibres of which are arranged in three more or less distinct strata. *The internal layer* is thin, and its fasciculi have a reticular[1] arrangement. *The middle layer* is thicker than the others, and consists of densely interlaced fasciculi running mainly in a circular direction. When hypertrophy of the musculature of the bladder occurs it is the fasciculi of this layer that stand out and give rise to the characteristic trabeculation of the bladder wall (fig. 1430). While this layer is virtually absent within the limits of the trigone, its fibres are so well marked below that structure as to warrant their being accorded the dignity of the term **sphincter vesicæ**. The **trigonal muscle** is a separate entity derived from a prolongation of the longitudinal layer of each ureter. In the trigone the muscular strands from each side decussate, and eventually are inserted into the wall of the posterior urethra. *The external layer* is composed mainly of longitudinal fibres. Some of the fibres of this layer are prolonged in the pubo-prostatic ligaments (see below).

Supports of the Bladder.—Several parts of the related pelvic fascia are described as **true ligaments of the bladder.** The rectovesical portion of the pelvic fascia supports the bladder posteriorly. At the base of the bladder this fascia is united closely to the muscular tunic, but it thins out rapidly as it is reflected upwards. The two lateral true ligaments of the bladder are indistinct, but the anterior, or **pubo-prostatic ligaments**, are well defined, and are of great surgical importance. Each stretches from the front of the prostate and adjacent portion of the bladder to the lower part of the periosteum of the pubis.

The urachus and obliterated hypogastric arteries, together with the folds of peritoneum overlying these structures, are called the **false (median and lateral umbilical) ligaments of the bladder.** Condensation of fascia around the blood-vessels passing to the bladder is known as the **superior and inferior vascular pedicles.**

Arteries.—The superior and inferior vesical arteries are derived from the anterior trunk of the internal iliac artery. Branches from the obturator and inferior gluteal arteries, and in the female from the uterine and vaginal arteries, also help to nourish the bladder.

Veins form a plexus on the antero-inferior surface of the bladder; in the male the prostatic plexus (fig. 1428) is continuous with the vesical plexus, which drains into the internal iliac vein.

Lymphatics accompany the veins, and drain into the lymph nodes along the internal iliac vessels. Some lymphatics from the fundus pass to lymph nodes situated at the promontory of the sacrum.

Physiology.—Micturition is partly a reflex and partly a voluntary act. The nerves concerned in micturition are:

The sympathetic fibres, which arise in segments eleventh dorsal to second lumbar. These fibres pass via the presacral hypogastric nerve and the sympathetic chains to the inferior hypogastric plexus, and thence to the bladder.

The parasympathetic innervation, which is derived from the anterior primary divisions of the second, third and fourth sacral segments. These fibres pass through

[1] Reticular (Lat. *reticularis*) = resembling a net.

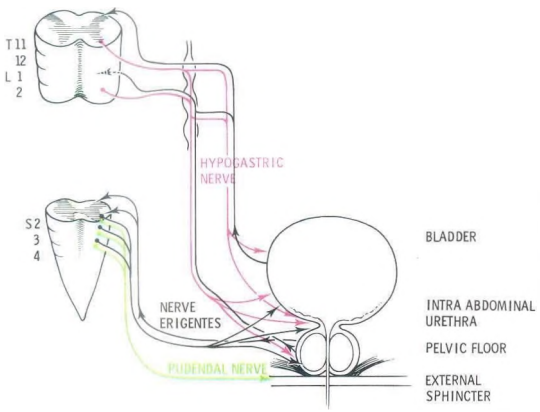


FIG. 1374.—The nervous control of the bladder.

the pelvic splanchnic nerves to the inferior hypogastric plexus, from which they are distributed to the bladder.

The somatic innervation also comes from the second, third and fourth sacral segments as the pudendal nerves which pass, not to the bladder itself, but to the sphincter urethræ (striated muscle).

While the sympathetic nerves convey afferent painful stimuli of over-distension *from* the bladder to the brain, it is unlikely that either set of autonomic nerves convey *to* the bladder any cortical impulses, such impulses being transmitted via the pudendal nerves to the sphincter urethræ, relaxing the latter. At the same time stretching of the muscle fibres by distension of the bladder initiates a reflex which is mediated through the parasympathetic nerves, and the detrusor muscle contracts. The neurogenic theory therefore ascribes the act of micturition to a stretch reflex which, if the time or place are not propitious, can be inhibited by cerebral control (fig. 1374).

ECTOPIA VESICÆ (*syn.* EXSTROPHY OF THE BLADDER)

Ætiology.—Incomplete development of the infra-umbilical part of the anterior abdominal wall, associated with incomplete development of the anterior wall of the bladder, is the embryological origin of the condition.

Clinical Features.—Ectopia vesicæ occurs once in 50,000 births, and may be complete or incomplete. In the more common complete variety, because of the pressure of the viscera behind it, the deep-red posterior bladder wall protrudes through the defect (fig. 1375). If the exposed mucous membrane, which bleeds readily, is drawn gently upwards, the paler, wet trigone is displayed, and effluxes of urine from the ureteric orifices can be seen. There is a well-defined line of demarcation between the protruding mucous membrane and the adjacent skin, and, especially after reducing the extruded bladder wall beneath the mucocutaneous junction, the firm edge of the hernial ring can be felt. Usually the umbilicus is absent. There may be umbilical and inguinal herniæ. The rectal sphincter is often lax. The sex ratio is about four males to one female.

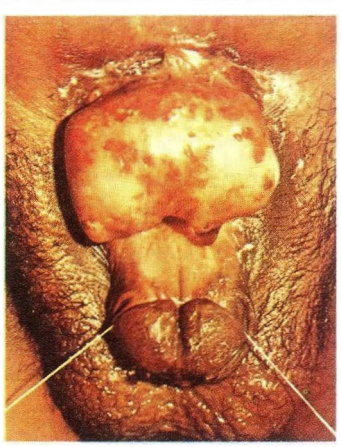


FIG. 1375.—Ectopia vesicæ in a male. A drop of urine is seen at the left ureteric orifice, the corona glandis being retracted by threads. (*G. D. Adhia, F.R.C.S., Bombay.*)

In the male, the completely epispadiac penis is broader and shorter than normal, and not infrequently bilateral inguinal herniæ are present; the prostate and seminal vesicles are rudimentary or absent, whereas the testes are normal and descend more often than not. *In the female*, the clitoris is cleft and the labia minora are separated anteriorly, exposing the vaginal orifice. In both sexes there is separation of the pubic bones

(fig. 1376), which are connected by a strong ligament, and, except for the fact that the patient has a waddling gait, this bony defect causes no disability; indeed, in those female sufferers who become pregnant, it facilitates delivery. The linea alba is as wide as the separation of the pubic bones. In the rare incomplete form the pubes are united and the external genitalia are more normal.

The lot of a patient with ectopia vesicæ is particularly unfortunate. A portable urinal seldom keeps him dry for long, and the odour of urine always accompanies him. Frequently the everted vesical mucous membrane becomes ulcerated and painful. It undergoes metaplastic changes, and sometimes becomes the seat of an adenocarcinoma. Attempts to prevent recurrent attacks of ascending pyelonephritis by chemotherapeutic and antibiotic agents are only partially successful; 50 per cent. of those afflicted die of renal failure before the age of thirty.

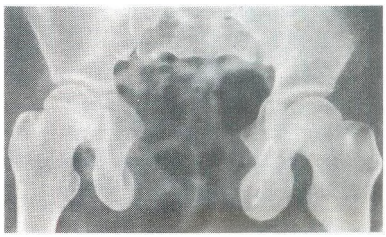


FIG. 1376.—Showing the separation of the pubes in a case of ectopia vesicæ. (*The late Professor Grey Turner, London.*)

Treatment

(*a*) *Diversion of urine* (p. 1258), into the colon, followed some months later by cystectomy. The operation is undertaken between four and six years of age.

(*b*) The best long term results would seem to follow diversion into an *ileal conduit* (p. 1260), *with excision of the bladder*, at the age mentioned. Renal function is likely to be better maintained than following uretero-colic anastomosis, and there are no problems of rectal control (see clinical features).

(*c*) *Iliac Osteotomy and closure of abdominal wall.*—In recent years, attempts to reconstruct the bladder and sphincters by operation within the first year of life have shown that it is possible to obtain a sound mid-line abdominal scar by commencing the operation with osteotomy of both iliac bones just lateral to the sacro-iliac joints. The bladder can be closed, and the reconstructed urethra displaced behind the pubis, which now meets in the mid-line. However, the ultimate results in respect of continence, freedom from urinary infection, and stone formation are disappointing, though in males the procedure may be a useful method of keeping the patient dry with an appliance. It is better, if this is the intention, to make no attempt to produce continence at the bladder neck.

Results.—Comparatively few who have undergone successful uretero-sigmoidal implantation for ectopia vesicæ live for twenty years. The causes of the untimely demise are (1) stricture at the site of anastomosis and bilateral hydronephroses, (2) recurrent pyelonephritis and (3) hyperchloræmic acidosis (p. 1261). Many different methods of implanting the ureters have been devised (fig. 1424); all aim at reducing the risks of stenosis and reflux.

RUPTURE OF THE BLADDER < **Intraperitoneal 20 per cent.** **Extraperitoneal 80 per cent.**

As the bladder has intraperitoneal and extraperitoneal relations it follows that rupture or perforation results in either intraperitoneal or extraperitoneal extravasation of urine (figs. 1377 and 1378). Intraperitoneal rupture may be due to a blow, kick or fall on to a fully distended bladder and it is more common in the male than in the female. Extraperitoneal rupture is usually caused by a fractured pelvis, occasioned by the all too common road traffic accidents of today. Other causes of rupture or perforation of either type include gunshot wounds, stab wounds, the passage of instruments, endoscopic resection, or diathermy coagulation of bladder tumours.

Intraperitoneal Rupture.—There is sudden, agonising pain in the hypogastrium, often accompanied by severe shock and perhaps syncope. However, especially if the patient was 'drunk and distended', in a few minutes

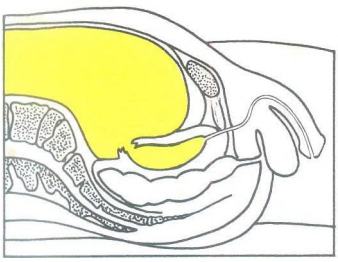


FIG. 1377.—Intraperitoneal extravasation of urine.

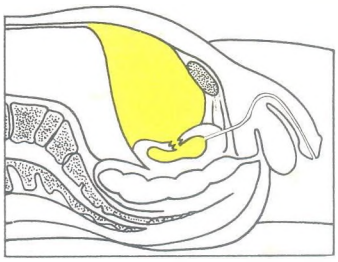


FIG. 1378.—Extraperitoneal extravasation of urine.

the shock subsides and the pain lessens, so that the patient may continue with his orgies, but the abdomen commences to distend. Usually the patient has no desire to micturate. On examination varying degrees of abdominal rigidity and, after a few hours, abdominal distension are present. In spite of the fact that the patient has not passed urine since the accident, there is no dullness above the pubes corresponding to a distended bladder. Usually there is tenderness in the hypogastrium. If the amount of urine in the peritoneal cavity is considerable, shifting dullness can be elicited. Rectal examination often reveals a bulging in the rectovesical pouch. When the urine is sterile, symptoms and signs of peritonitis are delayed for hours.

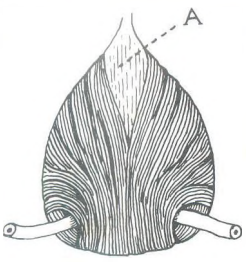


FIG. 1379.—Intraperitoneal rupture occurs within the area A. The tear is nearly always longitudinal.

Extraperitoneal Rupture.—As in many cases this cannot be differentiated from a rupture of the posterior urethra and as the treatment is the same, this injury is dealt with on p. 1289.

Confirming a Suspected Diagnosis.—A plain X-ray taken in the erect position may show the ground glass appearance of fluid in the lower abdomen. Intravenous pyelography may confirm a leak from the bladder. A peritoneal 'tap' may be of value. If doubt still exists and if there is no sign of fracture then retrograde cystography can be performed safely. With careful asepsis a small (14F) catheter is passed. Usually some blood-stained urine will drain. A solution made from 60 ml. of 35% Hypaque or Conray with 120 ml. of normal saline for intravenous use is injected into the bladder and radiographs taken.

Treatment.—When there are reasonable grounds for suspecting rupture of the bladder, operation should be undertaken without delay.

Intraperitoneal Rupture.—Lower laparotomy is performed. Urine is removed by suction, after which the patient is placed in Trendelenburg's position. The edges of the rent, which is usually situated in the posterior part of the dome of the bladder (fig. 1379), are trimmed and sutured by two layers of interrupted catgut stitches, and the operation completed by stitching a large Malécot catheter into an extraperitoneal suprapubic incision in the bladder. The latter ensures intravesical tension being kept at a minimum during

Friedrich Trendelenburg, 1844–1924. Professor of Surgery, Leipzig, Germany.
In 1839 Dr. Walther, a General Practitioner of Pittsburg, successfully sutured an intraperitoneal rupture of the bladder of a blacksmith.

healing of the sutured tear. Unless obvious infection is present the peritoneum can be closed without drainage. The abdominal wall is repaired around the Malécot catheter, not omitting drainage of the prevesical space. Following the operation, antibiotic therapy is given until the urine is proved sterile after removal of the catheter in 8–10 days.

Prognosis.—When operation is performed within twelve hours the mortality is approximately 11%; when operation is delayed to twenty-four hours the mortality rises to 55%. As in the days of ancient Greece, when the condition was regarded as inevitably fatal, without operation the mortality is 100%.

WOUNDING OF THE BLADDER DURING OPERATION

Operations in which the bladder is liable to be injured are (1) inguinal or femoral herniotomy (especially during the performance of the low operation for strangulated femoral hernia); (2) hysterectomy (particularly panhysterectomy by either the abdominal or vaginal route) and (3) excision of the rectum. In all these operations, to minimise this accident the bladder must be emptied *after* the patient has been anæsthetised. If the injury is recognised at the time of its infliction, the bladder must be repaired in two layers and urethral catheter drainage maintained for seven days. If it is not so recognised, the treatment is similar to that of rupture of the bladder.

When accidental perforation of the bladder occurs during endoscopic resection of a bladder tumour, or the prostatic capsule is perforated during transurethral prostatectomy, for practical purposes the perforation is always extraperitoneal. When the accident is recognised at the time, drainage of the bladder with a large urethral catheter, and the administration of antibiotics, usually suffice.

RETENTION OF URINE

Retention of the urine is either acute or chronic, the latter leading ultimately to retention-with-overflow.

Acute retention

Ætiology.—The most frequent causes are:

In the male	*In the female*	*In the male child*
Prostatic enlargement.	Retroverted gravid uterus.	Meatal ulcer with scabbing.
Urethral stricture.	Disseminated sclerosis.	
Post-operative retention.	Hysteria.	

Other causes:

Following spinal anæsthesia.
Blood-clot in the bladder.
Rupture of the urethra (p. 1288).
Neurogenic (injury or disease of the spinal cord).
Fæcal impaction in the rectum.
Acute urethritis or prostatitis.
Urethral calculus (p. 1303).
Phimosis
Certain drugs (p. 1230).
Muscular atony from advanced age.

Clinical Features.—The patient has not passed urine for some hours, and is unable to do so. The swelling caused by a full bladder often can be

Achille-Etienne Malécot, 1852–?. He invented his catheter while Interne des Hôpitaux de Paris.

seen in a thin person; it is somewhat tender to palpation, and dull to percussion above the symphysis pubis. Spasms of acute pain occur periodically as the muscle of the bladder contracts. An attempt is made to elicit the cause of the retention; this is most frequently due to vascular engorgement of an already enlarged prostate (p. 1266), but the floor of the urethra should be palpated for the induration characteristic of a stricture, and the reflexes in the lower limb and perianal sensation, tested.

Treatment.—It is of paramount importance never to relieve, or attempt to relieve, acute retention and forthwith send the patient home. He must always be admitted and kept under observation for at least twenty-four hours. Once the patient is in suitable surroundings, the effect of administering a dose of Omnopon to relieve anxiety, and a hot bath to diminish internal congestion, is tried. In many cases the patient is able to pass urine into the bath. If this fails, catheterisation is attempted. This should always be performed with full aseptic ritual (p. 1268). A small (12F) pre-sterilised plastic catheter (Jaques), is employed; if this is unsuccessful, a Tieman's catheter (fig. 1380) should be tried. If the catheter is to be left indwelling, a Gibbon catheter (fig. 1386), or a small Foley catheter (fig. 1381) should be

FIG. 1380.—Tieman's catheter.

FIG. 1381.—Foley's catheter.

used. Failure of the catheter to find its way past an enlarged prostate may be due to a middle lobe projection. Catheterisation may be accomplished then by using a coudé[1] or bi-coudé catheter (18F), or by inserting a curved introducer down a Foley catheter. The bladder is then emptied completely and the catheter removed.

All catheters (and gum-elastic bougies) are calibrated in the French scale (F.),[2] and as the narrowest part of the urethra is the external urinary meatus, unless there is reason to believe that the urethra is strictured, it is usual to choose a catheter with a diameter a little less than that of the orifice, viz.—→

If, after a reasonable attempt with catheters, the bladder has not been entered, one of four courses may be adopted according to circumstances.

1. **Suprapubic Puncture.**—Suprapubic puncture with a lumbar puncture needle or an 'Abbocath' (p. 96) is a useful method of relieving acute retention when catheterisation has failed. If the bladder is allowed to refill after it has been punctured, leakage into the prevesical space may follow.

2. **Suprapubic Catheterisation** (Riches' Technique).—A catheter is inserted into the bladder suprapubically through a 1·25 cm. incision made under local anæsthesia 2·5 cm. below the level at which the anterior surface of the bladder curves upwards and backwards to form the dome. The fibres of the linea alba are divided in the

[1] coudé (adjective) = bent; coude (noun) = elbow.

[2] The French scale is also known as Charrière's scale, so called after Joseph Charrière (1803–1876), a Paris surgical-instrument maker.

James Archibald Jaques, 1815–1878. Works Manager, William Warne & Co. Ltd. India Rubber Mills, Barking, Essex, improved and patented the soft rubber catheter.
George Tieman & Co., Instrument Manufacturers, New York.
Frederic Eugene Baisal Foley, 1891–1966. Urologist, Miller and Ancker Hospitals, St. Paul, U.S.A.
Sir Eric William Riches, Contemporary. Consulting Surgeon and Urologist, The Middlesex Hospital, London.

length of the incision. After the special catheter has been mounted on its introducer (fig. 1382 (*a*), (*b*)) it is passed through the incision until its sharp point touches the surface of the bladder, and the instrument is passed into the bladder with a short, sharp thrust directed backwards and downwards. The introducer is removed and the advancer (fig. 1382 (*c*)) is passed down the lumen of the catheter and its expander is opened. Thus the catheter is carried towards the trigone, and the advancer is removed. A stitch secures the catheter to the skin, and this is reinforced with a strip of adhesive plaster.

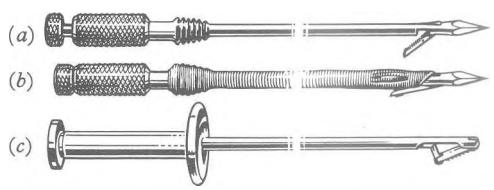


FIG. 1382.—(*a*) Riches' suprapubic catheter introducer; (*b*) the catheter stretched on the introducer; (*c*) the advancer.

3. **Immediate Prostatectomy.**—Possibly immediate prostatectomy in cases of benign enlargement of the prostate in fit patients.

4. **Urethral instrumentation** (p. 1298), in the case of a urethral stricture.

Chronic retention

In such cases the distension of the bladder is painless. The blood urea must be estimated before any attempt is made to relieve the retention. If it is below 70 mg% (12 mmol/l), treatment is as outlined for acute retention. Above this figure slow decompression is advisable (p. 1268).

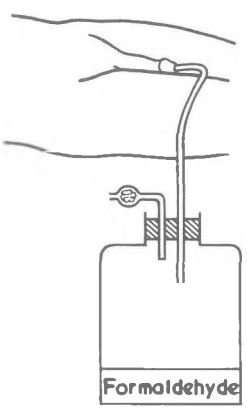


FIG. 1383.—The principle of continuous bladder drainage. The indwelling catheter has been introduced, using aseptic and antiseptic precautions (p. 1268). The sterile connecting tubing leads to the collecting bottle. This bottle should contain a suitable disinfectant, e.g. formaldehyde to keep the contents sterile. In common use today are closed sterile plastic containers fitted with a non-return valve (fig. 1384).

Retention with overflow

In this condition the patient has no control of his urine, small amounts passing involuntarily from time to time overflowing from a bladder which is distended. It may follow a neglected acute retention, or chronic retention.

Retention with overflow is referred to also under 'incontinence' (p. 1232) and 'prostatic enlargement' (p. 1267). The general principles which govern the treatment of this condition are similar to those of acute retention, but decompression of the bladder must be carried out very slowly (p. 1269).

THE INDWELLING CATHETER AND THE CLOSED SYSTEM OF CATHETER DRAINAGE

The incidence of ascending infection is nearly halved by connecting the catheter (urethral, suprapubic or perineal) to sterile tubing conducted to a sterile collecting bottle (fig. 1383), and employing irrigations only if clot retention occurs. When a catheter has been *in situ* for five or more days, some degree of urethritis is likely to supervene, though this has been much reduced by the use of catheters made from various non-irritant plastic substitutes for rubber. To change the catheter in the presence of urethritis entails a grave risk of infection from the anterior to the posterior urethra, and thence to other parts of the urogenital system (fig. 1385).

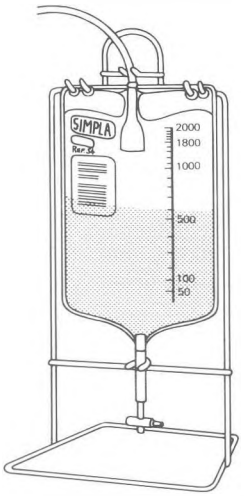


FIG. 1384.—Modern 'Simpla' bag[1] used for continuous bladder drainage. (The nurse who is emptying the bag should be wearing a disposable glove to avoid contaminating her hands with organisms). A disadvantage is that the tube may be too stiff to 'milk' and that formalin does not go with plastic.

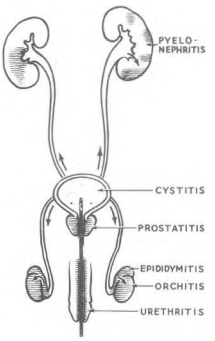


FIG. 1385.—Complications liable to follow changing a urethral catheter in the presence of urethritis. (*After C. G. Scorer, F.R.C.S., London.*)

SPECIAL FORMS OF RETENTION OF URINE

Post-operative Retention of Urine

Retention of urine can be encountered after any operation. It is common after operations on the anal canal and perineal region, and is due to reflex spasm of the urinary sphincters. After operations on the pelvic viscera, retention of urine is so common that it is usual to forestall it by inserting a catheter before or at the conclusion of the operation.

When the patient is an elderly male, prostatic obstruction, hitherto latent, should be suspected. Many patients cannot urinate while lying or sitting in bed.

Treatment.—First of all, reassure the patient. Provide privacy. If the male patient, while supported, is permitted to sit on the edge of his bed, he is often able to empty his bladder. The sound of running water is often helpful. When circumstances permit (e.g. after hæmorrhoidectomy) a warm bath is often helpful. If after a reasonable trial the patient cannot pass urine, he or she must be catheterised.

Acute Retention due to Drugs.—A number of drugs are prone to induce or precipitate retention of urine. Propantheline bromide, antihistamine drugs, antihypertensive drugs, anticholinergic drugs and I.N.A.H. compounds (chemotherapeutic agents for tuberculosis) have all been reponsible for producing acute retention of urine. Indeed, 'drug retention' has become a clinical entity.

Management of the Bladder in Spinal Injuries (p. 483)

Immediately after a spinal cord injury *spinal shock*, which may last for days, weeks or even months, occurs and in this state the detrusor is paralysed, the

[1] Simpla Plastics Ltd., Cardiff, Wales, U.K.

bladder distends and overflow incontinence will occur. This will lead to *atony*, infection and ultimately renal failure.

1. The bladder must be kept empty either by intermittent catheterisation with an aseptic technique performed two or three times daily or by the use of an indwelling Gibbon catheter (fig. 1386) on continuous drainage together with sulphamethizole (Urolucosil) and a high urinary output (3 litres daily) to combat infection.

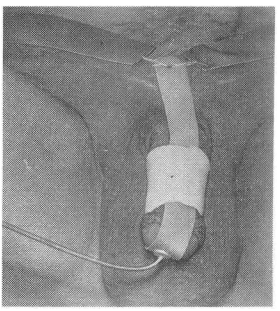


FIG. 1386.—Portex Gibbon catheter in place.

2. The upper level of the neurological lesion must be assessed by the level of sensory and motor loss. Ischæmic necrosis of the cord may extend a variable distance below the upper level of injury, but will indicate whether any section of the nerve supply to the bladder is likely to be involved. The sacral roots S2, 3, 4 will be at or about vertebræ level T12, L1.

Where sensory loss below the level is total, recovery is unlikely. Incomplete lesions on the other hand may recover somatic and particularly bladder function.

3. Demonstration of intact bulbo-cavernosus and anal reflexes indicates an intact sacral cord and ultimately a *reflex or automatic bladder* will result. If these reflexes are absent and there is total loss of perineal sensation recovery is unlikely and the bladder will behave in an *autonomous* fashion.

In cauda equina lesions there may be sensory, motor or mixed loss.

4. Full assessment of the bladder lesion should be undertaken about three months after the injury.

With the help of *urodynamic techniques*, including electromyography where possible, much more accurate assessment of the detrusor and sphincter activity and the sensory side of the reflexes controlling micturition can be obtained. N. K. Gibbon of Liverpool has pleaded for the assessment of the three functions, *storage*, *evacuation* and *control of urine*, carefully related to the neurological damage which at this time is stabilised. Many types of bladder dysfunction can occur in the absence of detectable neurological abnormality (fig. 1389).

The urethral pressure profile (U.P.P.) is helpful in demonstrating resistance to outflow at the bladder neck, external sphincter and along the urethra. Evidence is accumulating that the smooth muscle elements of the posterior urethra and external sphincter may play a larger part than previously supposed in the increased peripheral resistance which is so often the main factor in poor bladder emptying. Block of the alpha adrenergic receptors (with i.v. phentolamine 10 mg) and repeating the U.P.P. will demonstrate this. In addition to this effect which is common to all complete cord lesions, Gibbon and his associates have suggested that damage to the sympathetic outflow can lead to a hypersensitivity of the smooth muscle of the posterior urethra to circulating catecholamines—thus patients have increased difficulty in emptying the bladder in the erect posture.

The results of these studies should enable decisions to be made as to the further management of the bladder, the aim being to produce good emptying and to avoid infection.

Lesions above cord segment D10.—Upper motor neurone bladder with all reflexes intact but separated from higher control and inhibition.

Emptying: Detrusor contractions are normal. The bladder neck should

Norman Otway Knight Gibbon, Contemporary. Surgeon, Sefton Hospital, Liverpool, England.

open with detrusor contraction. The external sphincter is often spastic and division of this in the midline at 12 o'clock perurethrally may be needed. (This will not impair erections.) 'Alpha blockers' may be needed if there is excessive smooth muscle activity. Sometimes a perurethral resection (P.U.R.) of the bladder neck may be needed if fibrosis has occurred.

Capacity: This tends to reduce as the years pass by because trabeculation (fir-tree bladder on X-ray) seems to be progressive.

Control: Continence may be incomplete owing to the uninhibited detrusor contractions and in men a condom appliance may have to be worn.

A small percentage of patients can remain continent by regulating fluid intake and by provoking a reflex bladder response by deep tapping over the bladder in anticipation of the spontaneous event.

Lesions involving the sympathetic outflow, D11, 12, L1, L2.—Essentially an upper motor neurone bladder but with the additional loss of the sympathetic afferents and sensory afferents from the bladder. There is less smooth muscle element in the urethral resistance as shown by phentolamine block but a greater response to posture and these patients may have difficulty in emptying in the erect posture. 'Alpha-blockers' may nevertheless be a help in cutting out the effect of circulating catecholamines in the erect posture.

Damage to the sacral centre S2, 3, 4 and Cauda Equina lesions.—*Emptying:* The detrusor is paralysed (parasympathetic injury). Abdominal straining and pressure on the bladder through the abdominal wall can produce reasonable emptying. Sensation through the hypogastric nerves if D11, 12 is intact may enable the patient to know when the bladder is getting full.

Storage: Quite a good capacity.

Control: Reasonable in the early stages if there is no infection but liable to leak on straining. The bladder neck is usually open. The external sphincter may be paralysed but if the motor nerves are intact incoordination may be present and sphincterotomy is needed to enable emptying to occur. Reflux is more common with the lower motor neurone bladder.

Persistent Retention of Urine following Excision of the Rectum.—In 10 per cent. of cases retention of urine persists for three months or more. Probably, owing to the absence of cystometric substantiation of damage to the parasympathetic nerve supply to the bladder, the retention is due to mechanical factors, notably sagging of the bladder into the space formerly occupied by the rectum. In most cases the retention can be cured by transurethral resection of the prostate. Usually the prostate is not enlarged, and consequently the resection can be carried out easily and quickly. Therefore, if after two weeks the patient is unable to empty his bladder and his general condition is satisfactory, transurethral prostatic resection should not be postponed. Thus the patient is spared what would probably be a long period of invalidism and the risk of urinary infection.

INCONTINENCE OF URINE

Incontinence of urine — the patient is either unaware that urine is passing, or if aware of it, is unable to prevent the leakage of urine from the urethra. The history is most important and in many cases will indicate the cause.

Enuresis — incontinence of urine due to involuntary acts of micturition in children without detectable neurological or urological disease. Usually the condition presents as failure to acquire normal control but in about 15 per cent. of cases it may commence after control has been acquired. In such instances there is usually an illness or psychological factor which reveals the condition. In both the underlying state is a persistence of the infantile pattern of bladder activity—sudden forceful contraction and emptying in response to a certain distension, an impairment of the sensory awareness of bladder content with lack of inhibitory control from higher centres.

Nocturnal enuresis is much more frequent than diurnal. Diagnosis may be made confidently if the history is typical; there is no dribbling between acts, there is no abnormality on clinical examination and the urine is completely normal. It is important to remember that retention and overflow (p. 1267), always due to obstruction at the bladder neck or in the urethra, may present at *any age* as bedwetting. Control by day may be adequate but there will be frequency and urgency.

Treatment.—Drugs to inhibit bladder irritability (belladonna) and to tighten the sphincter (ephedrine) may help. Diurnal enuretics should be encouraged to pass urine by the clock and gradually lengthen the duration between acts. At night the child should be wakened regularly. In cases of ' onset ' enuresis after a dry period, the social and psychological aspects of the family must be investigated. Older children may respond to an alarm which is activated by urine passing on to a pad.

Stress Incontinence — leaking of urine on coughing or sneezing or lifting heavy weights. It is due to weakness of urinary sphincters. Common in women after childbearing, it may also follow operations on the bladder neck and prostate, to relieve obstruction.

Operative Treatment.—In patients with considerable bulging of the anterior vaginal wall (cystocele), anterior colporrhaphy frequently improves continence. In the absence of a cystocele an operation to rectify sagging of the bladder neck can be undertaken in cases in which there is little or no improvement after a course of exercises.

Everard Williams' operation for stress incontinence is simple, and gives as good results as more complicated procedures. A Foley's catheter having been introduced into the bladder, the bladder neck and adjoining portion of the urethra are exposed via the retropubic route. The lower part of the bladder and the intrapelvic portion of the urethra are mobilised, but there is no necessity to separate the urethra from the vagina. Three chromic catgut sutures are introduced on either side, each traversing the wall of the urethra or that of the bladder neck, being careful not to penetrate the mucous membrane. The sutures are then passed through the fascia covering the deep surface of the pubic bones near the symphysis. When the sutures are tied they suspend the bladder neck and the proximal part of the urethra.

Dribbling of Urine and Normal Micturition.—This occurs when there is a ureteric fistula (p. 1242) or an aberrant ureter opening into the urethra beyond the sphincters (in male), or into the vagina in females. The history is diagnostic, and intravenous pyelography may reveal the absent upper calyces on one side. These segments are very liable to infection. Treatment is by excision of the aberrant ureter and portion of kidney which feeds it.

Harry George Everard Williams, 1892–1970. Obstetrician and Gynæcologist, Charing Cross Hospital, London.

Total Incontinence:

Here there is complete loss of control. The urine dribbles away all the time and the bladder is usually empty. Excluding all fistulæ (p. 1242) the causes of this condition are:

Congenital.—Severe epispadias.

Traumatic.—Damage to pelvic muscles and nerves, as in fractured pelvis or after prostatectomy.

Infective.—Acute exfoliative membranous cystitis as in chemical irrigation. Chronic tuberculosis.

Neoplastic.—Fixation of sphincters: Carcinoma of Prostate and Cervix.

Neurological.—Meningo-myelocoeles, Multiple Sclerosis (usually retention and overflow).

Management of Incontinence.—The aim is to keep the patient dry, free of odour, to lessen the incidence of skin excoriation, and to protect the kidneys from the effects of infection and back pressure.

Appliances in women are usually unsatisfactory. An indwelling catheter drained constantly into a leg urinal is usually a satisfactory solution although in some instances diversion via an ileal conduit is necessary (p. 1262).

In men, a condom urinal, or a Cunningham penile clamp (fig. 1387) may be satisfactory, and they avoid an indwelling catheter. Regular pyelographic checks to confirm the absence of retention and back pressure effects are essential.

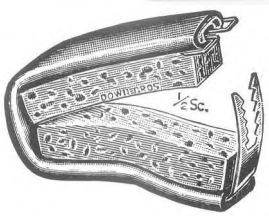


FIG. 1387. — Cunninghams' penile clamp.

Some of the conditions listed above may be amenable to specific surgical treatment. Thus a gracilis muscle sling may be attempted for epispadias or a post prostatectomy incontinence (fig. 1388). This usually needs to be activated by an electrical stimulator for complete success. Severe contraction of the bladder if sphincters are normal can be helped by a cystoplasty using ileum to enlarge it. Mechanical devices implanted around the urethra are having some success.

In neurological disease it is usually necessary to divert the urine into an ileal loop (p. 1262) if there is evidence of increasing dilation of the upper tract, particularly in the presence of recurrent infections. The bladder need not be removed but should be washed out after the operation and 1 g neomycin instilled. It should also be washed out two or three times in the post-operative period; after this it rarely produces any symptoms.

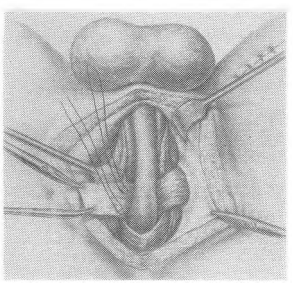


FIG. 1388.—Transplantation of the gracilis muscle to make a new sphincter urethræ.

Uretero-ileostomy (p. 1262).—The collection of urine from an artificial bladder into an ileostomy bag especially if the ileostomy has been given a spout of 2·5 cm long, is so much more water-tight than a portable urinal worn over the genitalia. The incidence of pyelonephritis is so much less, that the substitution of an artificial for a normal non-functioning bladder is a

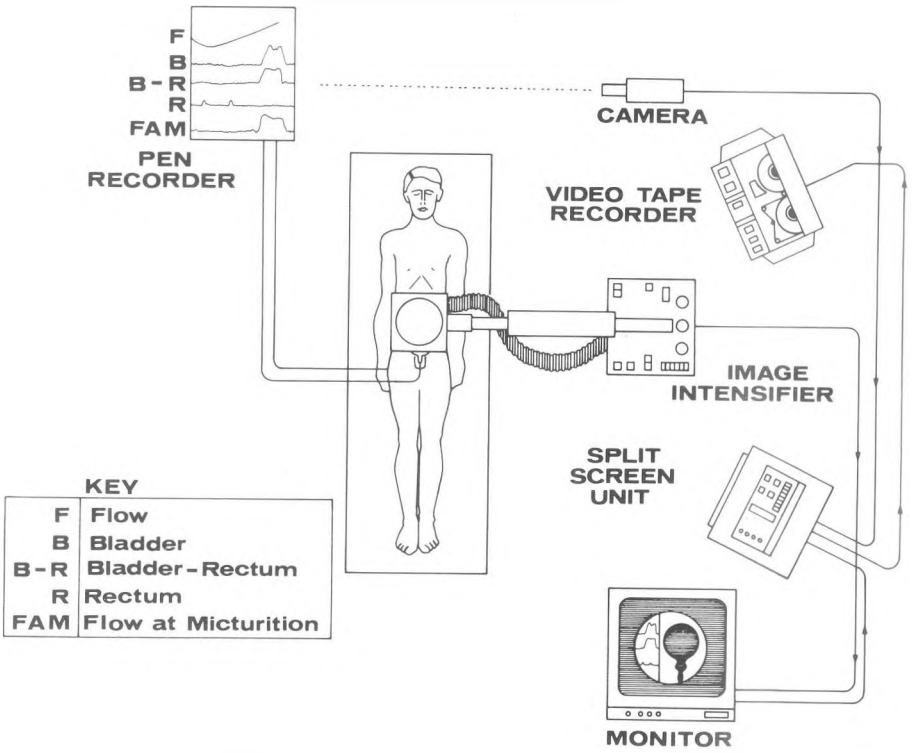


FIG. 1389.—Urodynamic study (p. 1231).

change for the better, not only for the patient but for those with whom he or she lives (the uriniferous odour is reduced, sometimes to vanishing point). The operation is particularly successful in cases of incontinence due to spina bifida (Wells).

Urodynamic study (fig. 1389) has thrown a great deal of light on cases of urgency and incontinence. The principle is to record the pressure within the bladder during filling, and emptying, and at the same time to record the patient's subjective symptoms, and observe the activity of the bladder with an image intensifier. A catheter is passed along the urethra into the bladder together with a small fine pressure tube. A similar pressure tube is placed in the rectum to record intra-abdominal pressure. Via transducers the pressures in the bladder and abdomen are recorded as waves on heat sensitive paper. A third pen records true intravesical pressure by a subtraction performed electronically.

Using a television camera to ' observe ' the pressure traces it is possible to display this on the screen of the image intensifier alongside the bladder and also to record all action and a commentary on video-tape.

VESICAL CALCULUS

Definition.—*A primary vesical calculus* is one that develops in sterile urine; it often, but not necessarily, originates in a kidney and passes down the ureter to the bladder, where it enlarges.

A secondary vesical calculus occurs in the presence of infection.

A vesical calculus can occur also by the deposition of urinary salts upon a foreign body in the bladder.

Incidence.—Until the twentieth century vesical calculus was one of the most prevalent disorders among the poorer classes, and the incidence was especially high

Charles Alexander Wells, Contemporary. Professor Emeritus of Surgery, Liverpool, England.

in childhood and adolescence. Owing to improved diet, especially an increased protein-carbohydrate ratio, primary vesical calculus is comparatively rarely encountered. The most remarkable fall in incidence is among children.

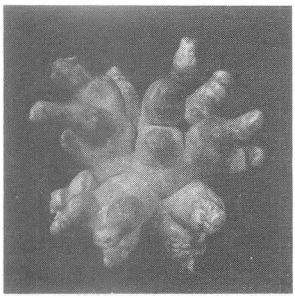


FIG. 1390.—'Jack-stone'[1] calculus. This type of vesical calculus is typically found in a post-prostatic pouch.

Composition and Cystoscopic Appearance.—Most vesical calculi are composite, but have one component in excess, and assume the appearance of that variety (see also p. 1190).

1. *Oxalate calculus* is a primary calculus that grows slowly. Usually it is of moderate size, and is solitary. Its surface is uneven (mulberry type; sometimes it bristles with spines (fig. 1390). Although calcium oxalate is white, the stone is usually dark brown or black because of the incorporation of blood pigment into it.

2. *Uric acid and urate calculi* are round or oval, fairly smooth, and vary in colour from pale yellow to light brown: they may be single or multiple. These stones also are primary: they are not opaque to X-rays.

3. *Cystine calculus* occurs only in the presence of cystinuria (p. 1191), and is radio-opaque due to its high sulphur content.

4. *Triple Phosphate calculus* is composed of ammonium, magnesium and calcium phosphates and occurs in urine infected with urea-splitting organisms. It tends to grow rapidly. In some instances it occurs on a nucleus of one of the foregoing types of calculus; much more rarely on a foreign body. In others the nucleus is composed of desquamated epithelium and bacteria. It is dirty white in colour, and of chalky consistency.

A vesical calculus is usually free to move in the bladder. It gravitates to the lowest part of the organ, which, when the patient is erect or sitting, is the bladder outlet. In the recumbent position (as also at cystoscopy) the stone occupies a position behind the inter-ureteric ridge. Less commonly the stone is wholly or partially in a diverticulum or a post-prostatic pouch, and in either case the stone may be partially or completely hidden from view.

Clinical Features.—Males are eight times more often affected than females.

(*a*) *Latent.*—When a stone is situated in a post-prostatic pouch or a diverticulum of the bladder, it is usually discovered unexpectedly at cystoscopy or on X-ray examination.

(*b*) *Typical.*—*Frequency* is the earliest symptom. Unlike other forms of frequent micturition, it is not much in evidence during the night. After micturition the patient does not feel satisfied that the bladder is empty.

Pain is most in evidence in cases of spiculated oxalate calculus. It occurs at the end of micturition, and is usually referred to the tip of the penis or to

[1] 'Jack-stone' = the kernel of the fruit of the jack tree. These fruit stones are used for a game played by children in the U.S.A.

the labia majora; more rarely to the perineum or suprapubic region. Pain and discomfort are much in evidence during exertion, and are aggravated by jolting movements (e.g. riding in a vehicle). If the patient lies down, the symptoms tend to pass off because the stone falls away from the sensitive portion of the bladder, the trigone. Thus he usually sleeps peacefully through the night. In young boys, screaming and pulling at the penis with the hand at the end of micturition are indicative of vesical calculus.

Hæmaturia is characterised by the passage of a few drops of bright red blood at the end of micturition, and is due to the stone abrading the vascular trigone—a fact that also accounts for the pain.

Interruption of the urinary stream by the stone blocking the internal meatus, occurs occasionally, and may be remedied by a change of posture.

Acute retention of urine from a vesical calculus is extremely uncommon in adults, but not so in children.

(*c*) *Masked.*—The symptoms of a concomitant persistent cystitis may overshadow those that might be occasioned by the stone.

Rectal or vaginal examination, when accompanied by abdominal palpation, occasionally enables a vesical calculus to be felt. Unless the stone is large, rectal examination is negative in the adult male, but in a female or child a calculus of moderate size may be palpable.

Examination of the urine will reveal blood, and possibly pus or crystals typical of the calculus, e.g. envelope-like in the case of an oxalate stone, or hexagonal plates with cystine calculi.

Radiography.—In at least 92 per cent. of cases vesical calculus can be demonstrated on an X-ray film (fig. 1391). If the stone is not opaque, a filling defect may be visualised. Radiographs of the whole of the urinary tract should be taken, after which excretory pyelography is carried out; the former will reveal or disprove the presence of opaque renal or ureteric calculi, while the latter will help to determine the normality or otherwise of the kidneys.

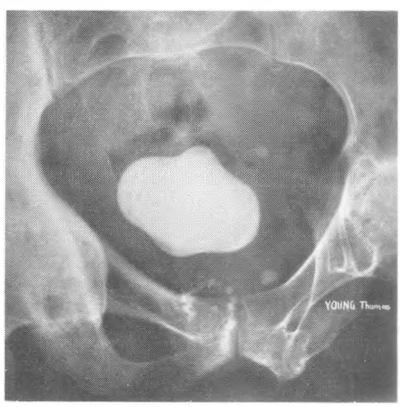


FIG. 1391. — Radiograph showing a vesical calculus.

Cystoscopy is essential for evaluation of the case. Frequently, on introducing the sheath of the cystoscope, a significant 'click' will be felt when a free-lying stone comes in contact with the instrument. As described already, cystoscopy usually determines the composition of the calculus. Stones non-opaque to X-rays, e.g. uric acid, can be seen.

In all cases the whole of the bladder wall is inspected: in primary calculus aseptic cystitis is basal; in secondary calculus bacterial cystitis is universal. In appropriate cases the exit of the bladder is examined for prostatic enlargement or contracture of the bladder neck.

Treatment:

Litholapaxy.—In most cases crushing the stone with a lithotrite is highly satisfactory. However, the contraindications to the method are as follows:

Contra-indications to Litholapaxy

- **Urethra**
  - Prostatic obstruction.
  - A urethral stricture that cannot be dilated sufficiently.
  - When the patient is below ten years of age.
- **Bladder**
  - Vesical diverticulum.
  - Cystitis that fails to respond to treatment.
  - Contracted bladder.
  - Other conditions requiring operation, e.g. enlarged prostate, new growth.
- **Stone**
  - A very large stone.
  - A very small stone, which should pass naturally.
  - Too hard to crush—an oxalate calculus.
  - Too soft to crush—a cystine calculus.
  - A stone encrusting a foreign body.

Technique.—For several days before the operation a suitable drug is administered to reduce infection. While blind litholapaxy is preferred by those who have had much experience with the solid lithotrite, its only advantage is that, by reason of its solidity and greater strength, harder stones can be crushed than is the case with an instrument that contains a light and telescope incorporated in its shaft. A cystoscopic lithotrite (Canny Ryall, Riches) enables the stone, and such fragments as are necessary, to be seized under vision. The instrument, with its obturator in place, is introduced into the bladder, and rotated so that its closed jaws point downwards. The obturator is removed and the bladder is irrigated with water or glycine solution by means of two 200-ml. syringes until the lotion is returned clear. The bladder is then filled with not more than 300 ml. of the solution, and after inserting the telescope, the stone is seen. The screw on the handle of the instrument is turned, and the jaws thereby opened. The distal blade is hooked over the centre of the stone (fig. 1392) and by rotating the screw handle the proximal movable blade is advanced so that the stone is grasped firmly. The ocular end of the lithotrite is depressed, thereby lifting the calculus away from the bladder wall. After withdrawing the telescope slightly, to prevent damage to the light bulb, the screw is turned slowly, breaking the stone. Large fragments are crushed into small ones by repeating the manœuvre. With the jaws closed, the lithotrite is rotated so that the jaws point upwards, and after removing the telescope and allowing the lotion to escape, the instrument is withdrawn.

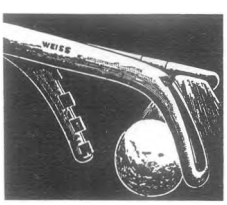


FIG. 1392.—A cystoscopic lithotrite.

FIG. 1393.—An evacuating cannula.

FIG. 1394.—Modified Bigelow's evacuator.

Evacuation of the Fragments.—An evacuating cannula (fig. 1393), the largest that the urethra will take, is passed, and 200 ml. of lotion are introduced into the bladder. The evacuator (fig. 1394), filled with lotion and with its tap closed, is fitted on to the cannula. The tap is opened and the bulb is elevated so as to depress the beak of the cannula towards the base of the bladder, after which the bulb is compressed slowly.

Edward Canny Ryall, 1865–1934. Surgeon, All Saint's Hospital, London.

The bulb is then permitted to expand, and the returning lotion carries with it sand and fragments of the stone which, being heavier than the lotion, drop into the glass receptacle. Compression of the bulb and aspiration is continued until no further fragments fall. The beak of the cannula is turned to the left and to the right, and suction is applied in these situations. To save time in emptying and refilling two evacuators can be used. If at any time the bulb fails to expand, this may be due to blocking of the eye by bladder mucosa, in which event release can be effected by further compression of the bulb and slight rotation of the instrument. Blockage of the cannula by fragments of the stone can sometimes be remedied in the same way, but more often they must be dislodged by detaching the evacuator and passing the obturator of the cannula. When no more fragments can be aspirated, the evacuator is detached and the bladder is irrigated until the lotion is returned clear. The cystoscopic lithotrite is reintroduced, and if no fragments remain, a Foley's catheter is passed. After the patient has been returned to bed the catheter is connected to a sterile bottle. Usually the catheter can be removed in twenty-four hours. As soon as it is assured that the patient is passing urine without difficulty, he can return home. Suitable drugs should be given until the urine is sterile.

Suprapubic Lithotomy.—The alternative to litholapaxy is removal of the stone through a suprapubic incision, after which the bladder is closed and drained by a urethral catheter.

Lithotomy in Special Circumstances.—A stone associated with an enlarged prostate can be removed in the course of a suprapubic or retropubic prostatectomy. When the stone is associated with contracture of the bladder neck, it can be removed in the course of an open operation for that condition.

A very small stone sometimes can be removed by means of an evacuator after passing the largest-sized cannula commensurate with the calibre of the urethra. For stones too large to pass through the cannula but small enough to pass through the urethra, removal by seizing the stone in the jaws of a cystoscopic rongeur (fig. 1395) is ideal.

FOREIGN BODIES IN THE BLADDER

A piece of catheter or bougie may become broken and remain in the bladder. The variety of foreign bodies which have been removed from the bladder is astonishing, e.g. manicure sticks, hair-clasps (fig. 1396), hairpins and candle-grease. The presence of such objects in the bladder is usually accounted for by urethral masturbation, or attempts to produce a miscarriage. Occasionally a foreign body enters through the wall of the bladder, e.g. a piece of rubber tubing after cystostomy; unabsorbable sutures used in an extravesical pelvic operation. The diagnosis rests on cystoscopy, and in the case of radio-opaque foreign bodies on radiography.

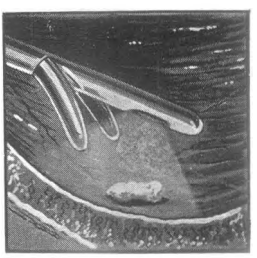


FIG. 1395.—Young's cystoscopic rongeur.

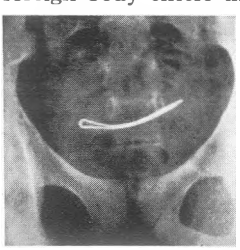


FIG. 1396.—A hair-clasp in the bladder.

Complications of a Foreign Body in the Bladder:

1. Cystitis, which is the most common complication.
2. Perforation of the bladder wall.
3. Vesical calculus.

Treatment.—A small foreign body can be removed per urethram by means of an operating cystoscope or Young's cystoscopic rongeur. When the foreign body is heavily encrusted, penetrating the bladder wall, or accompanied by severe cystitis, the suprapubic route should be chosen. Paraffin wax can be dissolved by the introduction of equal parts of xylol and water into the bladder for half an hour.

Frère Jacques de Beaulieu, 1651–1714, was the most famous Journeyman Lithotomist; he mainly practised the perineal operation using a bread-knife.

Hugh Hampton Young, 1870–1945. Director, James Buchanan Brady Urological Institute, Baltimore, Maryland, U.S.A.

DIVERTICULUM OF THE BLADDER

Definition.—It is most important to distinguish a saccule from a diverticulum of the bladder. The normal intravesical pressure at the commencement of micturition is about 35 cm of water (25·7 mmHg or 3·4 kPa). Pressures as great as 100 cm (73·5 mmHg or 9·8 kPa) are reached by a hypertrophied (trabeculated) bladder (fig. 1430) endeavouring to force urine past an obstruction. This pressure causes the mucous lining between the inner layer of hypertrophied muscle bundles to protrude, so forming multiple saccules. If one or more, but usually one, saccule is forced through the whole thickness of the musculature of the bladder wall it becomes a diverticulum.

Ætiology

(*a*) **Congenital diverticulum** is rare. It may be situated in the middle line anteriosuperiorly, and represent the unobliterated vesical end of the urachus. It empties with the bladder, and is symptomless. Others in the usual situation on the base of the bladder can occur without obstruction and may require excision because of the risk of chronic infection or stone formation in a young adult.

(*b*) **Pulsion Diverticulum.**—The causative obstructive lesion, in order of frequency, is contracture of the bladder neck, benign enlargement of the prostate, fibrous prostate, urethral stricture and congenital valves of the posterior urethra.

Pathology.—Usually the mouth of the diverticulum is situated above and to the outer side of one ureteric orifice. Exceptionally, it is near the middle line behind the interureteric ridge. The size varies from 2–5 cm, but may be larger. It is lined by bladder mucosa and the wall is composed of fibrous tissue only (compare traction diverticulum p. 1242). A diverticulum enlarges in a downward direction and sometimes may obstruct a ureter.

Complications

1. **Recurrent Cystitis.**—As the pouch cannot empty itself there remains a stagnant pool of urine within it. Once infected, the infection persists and continues to reinfect the bladder. In long-standing cases peridiverticulitis causes dense adhesions between the diverticulum and surrounding structures. Squamous cell metaplasia and leukoplakia occur in 15–20% of cases.

2. **Vesical calculus** due to stagnation and infection is present in 20% of cases, most often in the bladder, sometimes in the diverticulum as well, less often in the diverticulum only. On rare occasions a dumb-bell calculus fills the diverticulum and projects into the bladder.

3. **Hydronephrosis and hydroureter,** consequent upon compression of the lower end of the corresponding ureter, are liable to be followed by pyelonephritis and pyonephrosis.

4. **Neoplasm** arising in a diverticulum is an uncommon complication (7–8%). Unless the diagnosis is made at a very early stage, the prognosis is poor, because extravesical invasion through the thin wall of the sac occurs so readily.

Clinical Features.—An uninfected diverticulum of the bladder may cause no symptoms whatsoever. The patient is nearly always a male (95 per cent. of cases), usually over fifty years of age.

There are no pathognomonic symptoms of a vesical diverticulum; they are those of lower urinary tract obstruction, recurrent cystitis, and

pyelonephritis. Hæmaturia (due to cystitis, vesical calculus, or, rarely, a neoplasm) is a leading symptom in one-third of cases. In a few cases micturition occurs twice in rapid succession (the second act may follow a change of posture), and when the first specimen is clear and the second cloudy, diverticulum of the bladder should be strongly suspected. In cases of chronic retention of urine, two swellings may be recognised rising out of the pelvis, one being the bladder and the other the diverticulum.

Cystoscopy is the usual means of discovering the diverticulum. Most often its orifice is seen as a clear-cut hole about the diameter of a lead pencil, the depths of which are black and unilluminated (fig. 1397). With inadequate distension of the bladder, sometimes the mouth of the diverticulum is seen closed, when the mucous membrane around the potential orifice is thrown into radiating pleats (fig. 1398); therefore, when searching for a diverticulum, it is important to have the bladder fully distended. In heavily infected cases much irrigation is necessary before a clear view of the bladder wall can be obtained. The differential diagnosis of a diverticulum from sacculation of a trabeculated bladder is not difficult, for the orifice of the saccule is relatively large, and the shallow interior can be seen. It is sometimes possible to pass an endoscope into a diverticulum and examine its interior.

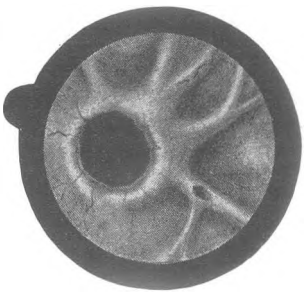


FIG. 1397.—Cystoscopic appearance of the orifice of a diverticulum and trabeculation of the bladder.

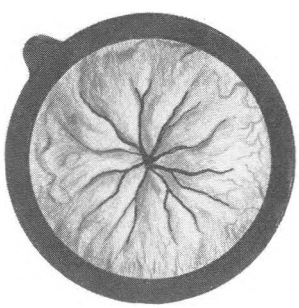


FIG. 1398.—Occasional appearance with inadequate distension of the bladder.

Excretory pyelography will not only exclude or reveal implication of the upper urinary tract, but in many instances the accompanying cystogram will give information regarding the size of the diverticulum.

Retrograde cystography is employed only when the former fails to show the pouch clearly. The bladder is emptied of urine, and filled with a non-irritating radio-opaque medium. Radiographs taken before and after micturition show the dimensions and position of the diverticulum clearly (fig. 1399).

Indications for Operation. — A vesical diverticulum gradually increases in size, and once infected the infection cannot be permanently eradicated. Therefore, unless the pouch is small and uninfected, it should be removed, usually when the associated lower urinary tract obstructive lesion is treated.

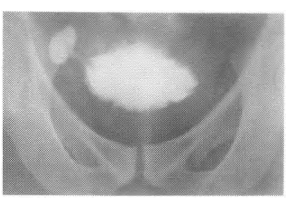


FIG. 1399.—Diverticulum of the bladder shown by cystography.

Pre-operative Treatment.—When the urine is infected, suitable pre-operative antibiotic treatment

is given. In the presence of gross sepsis and retention of urine, it is necessary to resort to an indwelling urethral catheter, and give bladder washes.

Combined intravesical and extravesical diverticulectomy is the operation that is practised most frequently. Cystoscopy is performed, and a large ureteric catheter is passed up the ureter on the affected side, and left in place, 240 ml. of lotion remaining in the bladder. With the patient in the Trendelenburg position, the anterior bladder wall is exposed through a suprapubic incision. The peritoneum is dissected upwards, and that side of the bladder bearing the diverticulum is cleared from surrounding structures with the fingers until some part of the pouch is brought into view. The bladder is then incised in the middle line near its dome, and emptied by suction. The interior of the diverticulum is packed with a strip of gauze, and with the fingers of one hand in the bladder and the recti muscles retracted widely, the diverticulum is freed from surrounding structures by gauze and sharp dissection. Usually the neck of the diverticulum can be separated from the ureter, and when the pouch is free it is severed from its attachment to the bladder with a diathermy knife. The resulting defect is closed in two layers. The cystostomy incision is sutured around a Malécot catheter, and the abdominal wall is closed, leaving a drainage tube in the extravesical space which housed the sac.

An alternative method, if the sac is densely adherent, is to carry the incision in the bladder down to the rim of the diverticular orifice, then to detach the diverticulum, together with its fibrous rim. The incision in the bladder is closed and the diverticulum left in position with a corrugated drain into it for two to three days. The track fibroses rapidly after removal of the drain.

TRACTION DIVERTICULUM OF THE BLADDER (*syn.* HERNIA OF THE BLADDER)

A portion of the bladder protruding through the inguinal or femoral hernial orifice occurs in 1·5 per cent. of such herniæ treated by operation (Sir Cecil Wakeley). The condition is relatively frequent in femoral and direct inguinal herniæ. The disposition of the diverticulum in the hernia varies, and in order of frequency is:

(*a*) (*b*) (*c*)

FIG. 1400.—(*a*) Intraperitoneal (*b*) paraperitoneal (see fig. 1271) (*c*) extraperitoneal hernia of the bladder.

Clinical Features.—In a few cases, in order to empty the bladder completely, the patient finds it necessary to exert pressure on the hernia. Suspicion of a bladder hernia can be confirmed by cystoscopy or cystography.

Accidental Wounding of the Bladder during an Operation for Hernia.—When recognised at the time, the wound in the bladder should be closed by a double layer of catgut and an indwelling catheter is retained for a week or so. If the accident is not recognised at the time and unless early operation is undertaken to repair the rent and drain the prevesical space or peritoneal cavity, as the case may be, together with suprapubic cystostomy, the outlook is grave.

URINARY FISTULÆ

A urinary fistula is an abnormal communication between any part of the urinary system and the skin or some internal hollow viscus. The persistence of a fistula on the skin implies the presence of distal obstruction or the presence of chronic infection such as tuberculosis or a foreign body, e.g. stone, or non-absorbable ligature (p. 68).

Sir Cecil Pembrey Grey Wakeley, 1892–1980. Surgeon, King's College Hospital, London.

Congenital Urinary Fistula.—(1) *Ectopia vesicæ* (p. 1224). (2) *From patent urachus.*—If present at birth or commencing soon after, the possibility of obstruction at the bladder neck or urethra must be investigated. In later life infection in a urachal cyst may produce a fistula. Carcinoma in such instances is not uncommon. Treatment in each case is excision of the urachal tract and closure of the bladder once distal obstruction has been dealt with or excluded.

(3) *In association with imperforate anus* (p. 1079).

Traumatic Urinary Fistula.—Perforating or penetrating wounds, damage not recognised during surgery, or poor healing and avascular necrosis following a combination of radiotherapy and surgery may lead to leakage of urine from ureter or bladder through the wound. Also a clot retention occurring after a transvesical prostatectomy or diverticulectomy may lead to dehiscence of the bladder wound and a temporary fistula, which will heal quickly, provided the bladder is kept empty with an indwelling catheter.

Vesico-vaginal fistula "is the greatest misfortune that can happen to a woman, the more so because she is condemned to live with it, and cannot hope to die from it" (Dieffenbach).

Ætiology: 1. *Obstetrical Causes.*—From time immemorial the usual cause has been protracted or neglected labour.

2. *Gynæcological Causes.*—The operations chiefly concerned with this complication are total hysterectomy and anterior colporrhaphy.

3. *Radiotherapeutic Causes.*—First and foremost is the radium treatment of carcinoma of the cervix uteri; to a lesser extent irradiation of the pelvic viscera for other reasons is responsible.

4. *Direct Neoplastic Infiltration.*—Exceptionally carcinoma of the cervix uteri ulcerates through the anterior fornix to implicate the bladder.

When a wound of the bladder is recognised and repaired at once, leakage is uncommon, but escape of urine will quickly follow if such damage is passed unnoticed. However, most vesico-vaginal fistulæ are the result of ischæmic necrosis of the bladder wall due to prolonged pressure of the fœtal head in obstetric cases. In gynæcological cases the ischæmia is brought about by grasping the bladder wall in a hæmostat, including the bladder wall in a suture, or perhaps even by local œdema or hæmatoma. Leakage due to necrosis of tissue seldom manifests itself before seven days after the operation.

An intractable fistula following radium treatment of carcinoma of the cervix uteri may arise from avascular necrosis years after the apparent cure of the original lesion.

Clinical Features.—There is leakage of urine from the vagina, and, as a consequence, excoriation of the vulva. Digital examination of the vagina reveals a localised thickening on its anterior wall. On inserting a vaginal speculum, urine will be seen escaping from an opening in the anterior vaginal wall. It is usually possible to pass a bent probe from the vagina into the bladder. Cystoscopy is often difficult, owing to the contraction of the bladder from cystitis and the escape of urine from the fistula; however, usually the tip of the probe that has been passed can be seen emerging through an area of granulation tissue.

Differential Diagnosis between a Uretero-vaginal and Vesico-vaginal Fistula.—If a swab is placed in the vagina and a solution of methylene blue is injected through the urethra, the vaginal swab becomes coloured blue. In the case of a uretero-vaginal fistula the vaginal swab is not so coloured. Uretero-vaginal fistula is discussed on p. 1180.

Treatment.—Operation must be postponed for several months to enable viable

Johann Friedrich Dieffenbach, 1792–1847. Director of the Surgical Division of the Charité Krankenhaus, Berlin. James Marion Sims, 1813–1883, while a country Practitioner at Montgomery, Alabama, was the first to succeed in closing a vesico-vaginal fistula.

tissue in the neighbourhood to recover fully, and for scar tissue to consolidate. The fistula is exposed via the vagina; the edges are freshened and a longitudinal repair of tantalum wire used to traverse the bladder wall (not mucosa) and the whole thickness of the vagina. Parallel vaginal incisions may be used to relieve tension on the suture lines. The bladder is drained by No. 4 polythene tubing (Chassar Moir) for fourteen days, unless the urethra is primarily affected, when a suprapubic cystotomy is performed.

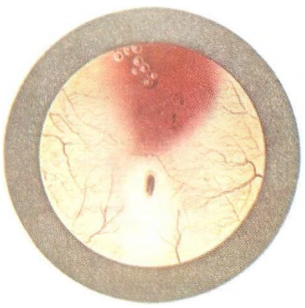


FIG. 1401.—Cystoscopic view of a vesico-intestinal fistula. Bubbles of gas can be seen issuing from the orifice of the fistula.

Fistula from Renal Pelvis to Skin or Gut.—Tuberculosis of a kidney may result in destruction of the kidney by caseation, and a chronic sinus leading to duodenum or colon and/or the skin, usually in the iliac fossa, or in the lumbar triangle of Petit. Similarly pyonephrosis and stone may spontaneously discharge into the gut or by a fistula to the skin. Cases of duodenal ulcer involving the pelvis of the right kidney are also on record.

Fistulæ Arising as a Result of Infection.—The commonest cause is diverticulitis of the colon. It may also follow Crohn's disease, appendix abscess, or pelvic sepsis in association with acute salpingitis, or as a result of surgery and radiotherapy within the pelvis.

The onset may not be dramatic, and may well be treated as acute cystitis. The diagnosis can be difficult to make because the acute signs subside, but on cystoscopy a patch of œdema on the left side of the vault is suggestive, and a cystogram may reveal the fistula. However, as the tract is not always patent a negative result may be obtained. The passage of gas per urethram in a patient who has cystitis is most suggestive (providing diabetes is excluded).

Treatment.—A defunctioning colostomy is made above the fistula as the first step. Inflammation is allowed to subside over two to three months. At laparotomy the communication is separated, the hole in the bladder being closed and patched with omentum, and the segment of diseased bowel resected. The bladder is drained by a urethral catheter. The colostomy is closed about 3 weeks later provided a barium enema reveals no leaks.

Cases due to Carcinoma.—By the time a fistula between the bowel and the bladder has developed, as a rule, the growth is inoperable or requires pelvic evisceration for its attempted eradication. As so often these patients are unfitted for this major procedure, it is best to leave the patient with the defunctioning colostomy performed in the first instance. Exceptions do occur, and occasionally it is warranted to undertake segmental resection of the colon and a partial or complete cystectomy.

Urethral Fistulæ occur as the result of infection above a stricture producing a peri-urethral abscess which rupturing into the urethra allows extravasation to occur suddenly into the scrotum and perineum. Urine and infection extend into upper 2·5 cm. of thigh and lower abdominal wall (p. 1292). Widespread cellulitis and tissue necrosis will occur unless diversion of urine is achieved by suprapubic cystotomy, and the tissue planes are freely drained by inguinal and scrotal incisions.

Neoplastic Fistulæ.—Primary bladder tumours may fungate through the abdominal wall when neglected. Only palliative treatment is possible in most of such cases. Involvement of the bladder by growths of cervix, uterus and colon and rectum do produce fistulæ, as may lymphosarcoma of small gut. Carcinoma of the prostate rarely produces a rectal fistula. Treatment in most such cases is difficult and prolonged and in most only palliative relief can be given. It is rarely in the patient's interest to divert the ureters (p. 1258).

CYSTITIS

Until after middle age, cystitis is very much commoner in women than in men. Cystitis occurs in some healthy women after intercourse, without any demonstrable abnormality of the urinary tract. Repeated attacks of cystitis

John Chassar Moir, 1900–1977. Nuffield Professor of Obstetrics and Gynæcology, Oxford, England.
Burrill Bernard Crohn, Contemporary. Gastro-enterologist, Mount Sinai Hospital, New York, U.S.A.

in women, or a single attack in a man or a child of either sex, should always be followed by investigation to discover and treat the predisposing cause; sometimes, however, no cause can be found.

Predisposing causes

1. Incomplete emptying of the bladder such as in prostatic obstruction, urethral stricture, stenosis of the external urinary meatus, diverticulum of the bladder, pregnancy or puerperium, also injuries or diseases of the spinal cord.

2. The presence in the bladder of a calculus, foreign body or neoplasm. Cystitis is sometimes associated with vesico-ureteral reflux causing attacks of pyelonephritis. It is especially important to detect and treat this in childhood and so prevent serious permanent damage to the kidney.

3. Lowered general resistance from intercurrent disease and avitaminosis.

Avenues of Infection

Ascending infection from the urethra is much the commonest. The organisms, especially *E. coli*, originate in the bowel, contaminate the vulva and reach the bladder because of the shortness of the female urethra. The passage of urethral instruments may cause cystitis in either sex, more especially when the bladder contains residual urine. This may happen even when the instrument is sterile because it carries organisms from the urethra into the bladder.

Other routes e.g. *descending* from the kidney, *hæmatogenous*, *lymphogenous*, and from *adjoining structures* (Fallopian tube, vagina, or intestine, especially fistulas) are less common.

Bacteriology.—*E. coli* is the commonest organism, followed by *Proteus mirabilis* and (in young women), *Staphylococcus saprophyticus* (formerly called ' Micrococcus '). Other organisms, sometimes mixed, and found especially in cystitis complicating underlying lesions of the urinary tract, include Pseudomonas, klebsiellæ, *Staph. aureus*, *Staph. albus* and various streptococci. Tuberculous cystitis is considered under ' special forms of cystitis ' (p. 1246).

The presence of pus cells without organisms calls for repeated examination for *M. tuberculosis* and the gonococcus (very rare cause of cystitis). Having eliminated these possibilities, the condition may be abacterial cystitis (p. 1246), or a bladder tumour.

Clinical Features.—The severity of the symptoms varies greatly; those of acute cystitis are usually the more distressing.

Frequency is the outstanding symptom, both during the day and at night. The desire to urinate occurs from every hour to every few minutes, and often it is so urgent that if the bladder cannot be emptied forthwith, incontinence results. In severe cases, because of infection and loss of sleep, the patient looks drawn and haggard.

Pain varies from mild to agonising.[1] When the inflammation is situated in the dome of the bladder, pain is referred to the suprapubic region, while when, as is often the case, the inflammation involves the trigone, pain is referred to the tip of the penis, the labia majora, and the perineum.

Hæmaturia.—The passage of a few drops of blood-stained urine or blood-

[1] An Irish surgeon, long since dead, always finished his evening prayers,—" Please God, when you take me, may it not be through the bladder".

stained debris at the end of micturition is a frequent accompaniment. Less often the whole specimen is blood-stained, but more so at the end.

Pyuria is always present, except in some cases of trigonitis and interstitial cystitis (p. 1248). If urine is passed into two glasses, the second is the more cloudy.

On examination there is tenderness over the bladder suprapubically, per rectum, or per vaginam.

Initial and midstream urine specimens should be collected in a male as prostatitis may be present, in which case threads will be seen clearly in the initial specimen (p. 1281). In a female, and when the initial urine in the male does not contain threads, a midstream specimen must be subjected to microscopy and culture, and the sensitivity of any organisms assessed.

Treatment should be commenced forthwith, and modified if necessary when the bacteriological report is to hand. The patient is urged to drink plentifully, and while awaiting sensitivity results, one should prescribe either a sulphonamide or nitrofurantoin (100 mg q.d.s.) with Mist. pot. cit et hyocyamus 10 ml. t.d.s. as a bladder sedative. Failure to respond, or early recurrence of infection, indicates the necessity for further investigation to exclude predisposing factors.

Cystoscopy to confirm the diagnosis may be performed in women during the acute phase. The appearances may vary from hyperæmia, sometimes with mucosal hæmorrhages, to severe sloughing and ulceration of the mucosa. In men, cystoscopy to exclude predisposing causes should be delayed until the infection has been controlled.

SPECIAL FORMS OF CYSTITIS

Acute Abacterial Cystitis (*syn.* Acute Hæmorrhagic Cystitis).—The patient, a young man or woman, presents with symptoms of severe cystitis. Pus is present in the urine, but no organism can be cultured therefrom. The condition, which sometimes is associated with abacterial urethritis, commonly follows recent venereal exposure. While tuberculous cystitis always must be ruled out by cultural or biological tests, the fact that excretory pyelography shows normal excretion on both sides strongly favours acute abacterial cystitis. In 60 per cent. of cases, by special cultural methods the pleuro-pneumonia-like organism, which frequently accompanies acute abacterial urethritis, is found.

Although this is a self-limiting disease its course can be shortened by the antibiotic therapy recommended for abacterial urethritis (p. 1295). When cystoscopy is performed after the acute symptoms have abated, abundant mucosal hæmorrhages are seen, and presumably at its height the infection causes a hæmorrhagic cystitis.

Urethro-trigonitis is a common form of recurrent lower urinary tract infection in women. Cystoscopy shows increased vascularity of the trigone, œdema of its mucous membrane (most marked towards the apex) and, in severe cases, there is a pseudo-membrane limited to the trigone. Inflammatory polypi may be present at the bladder neck (fig. 1483). Later the urethra becomes narrowed. A cystocele is frequently present.

A primary focus of infection must be sought; the most common being (*a*) the cervix uteri (chronic cervicitis or a cervical erosion) or (*b*) the intestinal tract, notably chronic colonic diverticulitis. No examination is complete

without examining the cervix with a speculum, and if the cervix is healthy a barium enema is necessary for evidence of diverticulitis.

Treatment.—In addition to the usual measures, urethral polypi should be destroyed by light fulguration and the urethra dilated, if necessary. Cervicitis or a cervical erosion is treated by excising the glandular tissue of the cervix with a diathermy loop. Diverticulitis is discussed on p. 999.

Tuberculous cystitis is always secondary to renal tuberculosis, and unless treated early is a particularly relentless form of chronic cystitis.

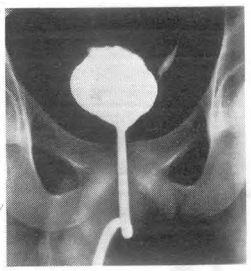


FIG. 1402.—Retrograde cystograph showing exceedingly contracted ('thimble') bladder in a case of tuberculous cystitis.

Cystoscopy shows that early tuberculosis of the bladder commences around one ureteric orifice or in the neighbourhood of the trigone, the earliest evidence being pallor of the mucosa, due to submucous œdema. Subsequently tubercles may be seen, and in long-standing cases there is much fibrosis, and the capacity of the bladder is so much reduced that it has earned the name of 'thimble' bladder. This feature can be well shown by cystography (fig. 1402).

Treatment.—Tuberculous cystitis usually responds rapidly to antituberculous drugs (p. 37), but occasionally, in cases with advanced renal changes, may not subside until the involved kidney and ureter have been removed. Local treatment of persistent tuberculous cystitis consisst of instillations of a substance inimical to *M. tuberculosis*. Among the best is B53, a soap derived from a branched fatty acid, having a high inhibitory index for the *M. tuberculosis*. A correctly buffered 0·5 per cent. solution containing local anæsthetic is instilled and retained for one hour. A fortnight's course can be repeated after an interval of a week.

When the bladder is considerably contracted, but free from ulceration, remarkable relief from the distressing frequency can be achieved by ileocystoplasty (fig. 1403), which is an extremely efficacious method of increasing the capacity of a contracted bladder. When, in addition, tuberculous contracture threatens to implicate the remaining kidney by back pressure, as shown by dilatation of the ureter on excretory pyelography, the ureter is implanted into the newly constructed pouch of intestine (p. 1261) with every hope of conserving renal function as well as rendering the patient symptom-free.

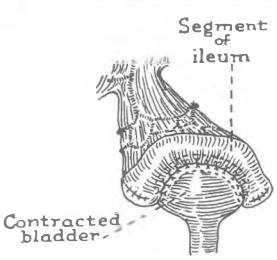


FIG. 1403. — Ileocystoplasty using a 6-inch segment of ileum. (*After D. O. Ferris.*)

Ileocystoplasty.—After pre-operative sterilisation of the intestines (p. 1022) a 15-cm segment of ileum with an ample blood supply as demonstrated by transillumination is disconnected, leaving its mesentery intact, and the continuity of the intestine is restored by anastomosis. After closing the ends of the donor segment and opening it longitudinally it is anastomosed to the dome of the contracted bladder (fig. 1403).

B53 (Hinconstarch) is produced in the laboratories of the Irish Medical Research Council.

Interstitial Cystitis (*syn.* **Hunner's Ulcer;** Elusive Ulcer).—For practical purposes Hunner's ulcer can be said to be a condition peculiar to women. The symptoms commence in the early forties. It can cause more pain, mental anguish and associated neurosis than does carcinoma of the bladder; as a consequence, the incessant painful micturition it occasions sometimes leads to drug addiction and even suicide.

Ætiology is still as obscure as it was when Guy Hunner first described the condition in 1914. Certainly it does not commence as an ordinary pyogenic infection of the mucous membrane, but rather as an infection of the paravesical tissues secondary, it is suggested, to infection of the adnexae, or even to infection from a more distant focus such as the nasopharynx. Some believe the condition is due to an attenuated *M. tuberculosis* which so far has defied isolation. In a number of instances the symptoms have commenced three or four months after a pelvic operation.

Pathology.—As a result of the paracystitis, fibrosis of the vesical musculature ensues, leading to contracture of the bladder and areas of avascular atrophy of the mucous membrane. Finally, ulceration of the mucosa occurs in the least vascular portion of the bladder, namely the fundus. Often the capacity of the bladder is reduced to 30 to 60 ml. The characteristic linear bleeding ulcer is a crack due to splitting of the mucous membrane when the bladder is distended under anæsthesia for cystoscopy.

Microscopically, inflammation of all coats of the bladder is present with granulation tissue in the submucosa underlying the ulcer. The muscularis is hypertrophied and the peritoneum in proximity to the area of maximum disease is thickened.

Clinical Features.—Bladder capacity becoming much reduced, increased frequency, eventually every hour both day and night, is the leading symptom. Pain, relieved by micturition and aggravated by jarring and overdistension of the bladder is the second and most characteristic symptom. In early cases the urine is crystal clear and sterile; in later cases it contains ordinary pyogenic bacteria in under half the specimens examined; in the remainder the urine remains sterile. In 60 per cent. of cases hæmaturia is a leading symptom.

Cystoscopy.—The lesion appears in the roof of the bladder as a star-shaped area of intense congestion in which a fissure can be seen when the bladder is distended. The ulcer, if ulcer it can be called, bleeds readily.

Treatment is difficult and unsatisfactory. Local hydrocortisone and irrigation of the bladder with various sedatives have proved valuable. Hydrostatic dilatation under anæsthesia may give relief for some months. Light diathermy fulguration of the ulcer may help. Complete relief may be obtained by urinary diversion, while ileo-cystoplasty after excision of the affected bladder wall, though frequently very satisfactory, is not always permanently successful in relieving all symptoms.

Alkaline encrusting cystitis is due to urea-splitting organisms causing phosphatic encrustations on the bladder mucosa of elderly women. There are symptoms of chronic cystitis and a plain X-ray shows the bladder outline. The encrustations may be curetted and removed via a cystoscope or a suprapubic cystotomy, or controlled by tidal lavage using a dissolvent solution such as 0·5 per cent. acetic acid (see also p. 1196).

Cystitis Cystica.—Glands are not found in the normal vesical mucosa. Under the influence of chronic inflammation, the surface epithelium sends down buds, resulting in minute cysts filled with clear fluid, most abundant on the trigone. Most of the cases are seen in women, and the urine usually contains pus, and commonly *E. coli*. An adenocarcinoma may originate in one of these cysts.

BILHARZIASIS OF THE BLADDER

Geographical Distribution.—The disease is endemic in the greater part of Africa, throughout Israël, Syria, Arabia, Iran and Iraq. It is met with frequently in Portugal and in Greece, and in the islands of Madagascar, Mauritius and Cyprus. Dwellers along the valley of the Nile have suffered from time immemorial. The condition also occurs along the shores of some of China's great lakes. Marshes or slow-running fresh water provide a favourable habitat for the particular fresh-water snail (Bullinus contortus), which is the intermediate host.

Mode of Infestation.—The disease is acquired while bathing or standing in infected water. The free-swimming bifid-tailed embryos (cercariæ) of the trematode

Guy Leroy Hunner, 1868–1951. Gynæcological Surgeon, Johns Hopkins University, Baltimore, U.S.A.
Theodor Maximilian Bilharz, 1825–1862. Professor of Zoology, Cairo.

Schistosoma hæmatobium penetrate the skin by their motile and erosive secretory powers. Shedding their tails, which enabled them to swim, they enter blood-vessels and are swept by the blood-stream into all parts of the body. All perish save those that are carried to the liver. Once within the liver the survivors flourish by sustaining themselves on erythrocytes, and they develop into male and female worms. The female is long, smooth and slender and is furnished with two weak suckers anteriorly. The male is broader, shorter (11 mm. in length), bosselated and provided with a strong sucker at either end. Sexual maturity having been attained, the nematodes leave the hepatic nursery and enter the portal vein. Here the male bends into the shape of a canoe and in so doing a gutter (the gynæcophoric canal) is formed along its length. Into the groove a female nestles and, thus conjugating, the pair (fig. 1404) paddle their way against the stream towards the inferior mesenteric vein. The long journey is accomplished in short stages, the male's suckers serving as anchors to the vein wall during periods of rest. *Schistosoma hæmatobium* worms have an affinity for the vesical venous plexus, and to reach it they must pass along the portal-systemic anastomotic channels, the most important in this respect being the venules in relation to the lumbar lymph nodes (Makar); other communications being venules in relation to the lower part of the ureters.

FIG. 1404.—Conjugating male and female *Schistosoma hæmatobium* trematodes. (*After A. Looss.*)

Having reached one of the smaller radicles of the vesical plexus the pair can go no further coupled, so the female, now pregnant but still comparatively slender, parts from the male and moves forward until she enters a submucous venule so small that she completely blocks it. She now proceeds to lay about twenty ova in a chain, and after each is deposited, by slightly withdrawing herself, the venule contracts upon the ovum. Each ovum is provided with a terminal spine (fig. 1405) which penetrates the vessel wall. Aided by muscular contraction of the bladder, and perhaps by secreting lytic fluid, some of the ova reach the lumen of the bladder; others die incarcerated in the mucous membrane. A heavily infected subject passes many hundreds of ova a day. If the ova reach fresh water, the low osmotic pressure of the new environment causes their envelope to burst, and thus emerges the miracidium, which is ciliated. To survive, the miracidium must reach and penetrate the intermediate host within thirty-six hours. Within the snail's liver the miracidium enlarges and gives rise to myriads of daughter cysts, broods of which are set free on the death of the snail. A single miracidium begets thousands of cercariæ to complete the life-cycle.

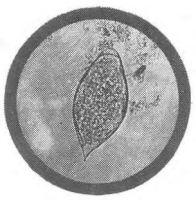


FIG. 1405.—A bilharzia hæmatobium ovum.

Clinical Features.—After penetration of the skin there may arise urticaria, which lasts for about five days and sometimes recurs (swimmer's itch). After an incubation period ranging from four to twelve weeks, high evening temperature, sweating, and asthma, together with leucocytosis and eosinophilia of over 10 per cent., sometimes last several weeks. Usually an asymptomatic period of several months supervenes before the ova laid in the bladder wall find egress and cause the typical early sign and symptom of intermittent, painless, terminal hæmaturia. Men are three times more often affected than women. Native patients of the peasant class rarely consult a doctor for this hæmaturia *per se*, so that many late cases are encountered.

Examination of the Urine.—The last few millilitres of an early-morning specimen of urine are collected and centrifuged. It is essential that all receptacles be absolutely dry. The ovum is recognised without staining under the low-powered microscope. Examination on several consecutive days may be required. Even so, a negative result does not exclude bilharziasis, especially in patients no longer resident in bilharzial districts.

Cystoscopy.—Dependent on the length of time the disease has remained untreated, cystoscopy will reveal one or more of the following:

1. *Bilharzial pseudo-tubercles* are the earliest specific appearance. The pseudo-

Napoleon's troops who served in his march to the Pyramids suffered from hæmaturia.

Naguib Makar, Contemporary. Emeritus Professor of Urology, Cairo University, Cairo, Egypt.

tubercles are larger, more prominent, more numerous, more yellow and more distinctly grouped (fig. 1406) than those of tuberculosis.

2. *Bilharzial nodules* (fig. 1407) are due to the fusion of tubercles in the presence of secondary infection. They are larger and greyer than the foregoing.

3. '*Sandy patches*' are the result of calcified dead ova with degeneration of the overlying epithelium. They occur in the first instance around one or both

FIGS. 1406.—Bilharzial tubercles; 1407 Bilharzial nodules; 1408 'Sandy patches'. (*After N. Makar.*)

ureteric orifices (fig. 1408). Considerable calcification of this nature is visible on the radiograph.

4. *Ulceration* is the result of sloughing of mucous membrane containing dead ova, or what is even more common, sloughing of a bilharzial papilloma. The ulcer is shallow (fig. 1409), bleeds readily, and its common position is the posterior wall of the bladder.

5. *Fibrosis* is mainly the result of secondary infection. The capacity of the bladder becomes much reduced. Contracture of the bladder neck is also a common result of bilharzial fibrosis.

FIG. 1409.—Bilharzial ulcer. (*After N. Makar.*)

6. *Granulomas*.—Bilharzial masses are due to an aggregration of nodules. They are sessile, soft and bleed readily when touched.

7. *Papillomas* are distinguished from the foregoing by being more pedunculated (fig. 1410). They vary in size from that of a pea to that of a walnut, and they may be single or multiple.

8. *Carcinoma* is a common end-result in grossly infected bilharziasis of the bladder which has been neglected for years. It usually commences, not in a papilloma, but in an ulcer, and is therefore a squamous-celled carcinoma.

FIG. 1410.—Bilharzial papilloma. (*After N. Makar.*)

Treatment[1].—Lesions 1 to 6 inclusive can be expected to heal under general treatment by antimony and other preparations (see p. 1103). It takes many months for dead ova to be expelled, and even after repeated courses and healing of the bladder lesion, living bilharzial worms have been found at necropsy in the portal system. In addition to general treatment, healing of bilharzial ulcers and granulomas is expedited by light diathermy coagulation. Bilharzial papillomas and, of course, carcinoma do not respond to general treatment:

[1] John Brian Christopherson, 1868–1955. Introduced the treatment of bilharziasis by the intravenous injection of tartar emetic in 1918 whilst he was Director of Civil Hospitals at Khartoum and Omdurman, Sudan.

these lesions require the same surgical measures as non-bilharzial papillomas and carcinomas, in addition to general treatment.

Other Complications and their Treatment:

(*a*) *Secondary bacterial cystitis* is commonly present in cases of some standing and the treatment of it must be thorough and prolonged.

(*b*) *Urinary calculi*, especially vesical and ureteric, occur more frequently when bilharzial lesions of the bladder are present. Litholapaxy is contraindicated in cases where there is ulceration of the bladder wall.

(*c*) *Stricture of the ureters* affects the last inch of the ureters. These strictures often respond to dilatation, but sometimes transplantation of the affected ureter into another part of the bladder is necessary.

(*d*) *Prostato-seminal vesiculitis*, like tuberculosis of these structures, is made worse by prostatic massage, and general treatment alone must be employed.

FIG. 1411.—Bilharzial contracture of the bladder, with ureteric reflux.
(*H. Talib, F.R.C.S., Baghdad.*)

(*e*) *Contracture of the bladder, and contracture of the bladder neck* (fig. 1411), must be treated in the same ways as similar contractures of non-bilharzial origin.

(*f*) *Bilharzial urethral strictures* are often accompanied by fistulæ, and can be cured only by excision of the fistulous tracks and urethroplasty (p. 1299).

NEOPLASMS OF THE BLADDER

More than 95 per cent. of primary tumours of the bladder originate in its mucous membrane; the remainder are connective-tissue growths—angioma (the least uncommon), myoma, fibroma, and sarcoma. These are too rare to merit further description, as also is a phæochromocytoma arising in the bladder wall from accessory adrenal tissue.

Another uncommon benign bladder tumour is an **endometrioma**, characterised by a localised, smooth, vascular projection on the bladder wall, sometimes containing chocolate-coloured cysts; and at other times translucent cysts of a bluish hue. The tumour enlarges and bleeds during menstruation. Treatment is partial cystectomy.

Secondary involvement of the bladder can occur from extension of a malignant neoplasm of a neighbouring organ, particularly the large intestine (sigmoid and rectum), the uterus or an ovary.

On rare occasions the first symptoms of a carcinoma of the pelvic colon are those of cystitis. Cystoscopy reveals a circumscribed area of intense inflammation, usually on the left side of the fundus. If the diagnosis is not made and treatment carried out at this stage, a vesico-intestinal fistula results (p. 1244).

Ætiology: The first suspicion of a chemical cause for bladder tumours was raised by Rehn in 1894 when he recorded a series of tumours in workers in aniline dye factories. Subsequent investigation has exonerated aniline itself—it was the impurity of beta-naphthylamine. It is now recognised that benzidine, alpha-naphthylamine, xylenamine, auramine and magenta are all carcinogenic. The particular industries involved are the dyeing industry, gas workers, the rubber and cable industries and the printing industry. Tumours occurring in workers in these industries are accepted for compensation.[1]

[1] Workers exposed to this hazard should have a sample of urine centrifuged and examined for red cells and neoplastic cells at three-monthly intervals.

Ludwig Rehn, 1849–1930. Surgeon, Frankfurt on Main, Germany.

Bilharzia, active or inactive, and bladder cancer are commonly found together and probably the former produces chronic irritation and incomplete emptying which produce metaplasia and squamous carcinoma.[1] Fibrosis reduces lymphatic metastases, but produces ureteric obstruction and renal damage. Leukoplakia of the bladder must also be regarded as a precancerous lesion. Despite these facts in the majority of cases the cause of a bladder cancer cannot be determined.

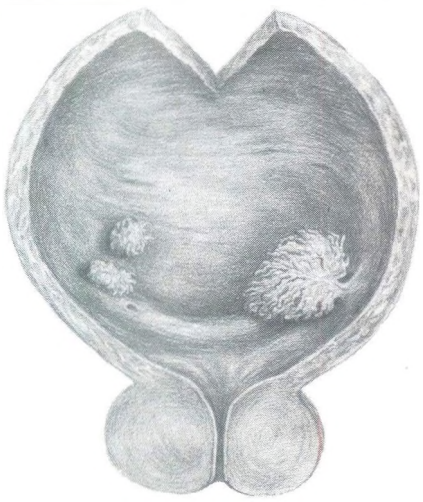


FIG. 1412.—Papillary tumour with daughter implantations (' kiss ' cancer).

Pathology

Benign Villous Tumours. — A papilloma of the bladder commences as a single frond with a central vascular core—later it becomes tufted. From these tufts spring villi—long, finger-like projections composed of three or four layers of transitional epithelium surrounding a capillary vessel with a minimum amount of supporting fibrous tissue. Thus the fully developed papilloma appears like a red sea anemone with delicate tentacles eddying to and fro with each and every movement of the bladder contents.

Diffuse papillomatosis is a term used to signify a condition in which multiple small growths are disseminated over a relatively wide area of the bladder. Pedicle formation is not so pronounced as in the solitary variety. To justify a diagnosis of diffuse papillomatosis, a stage must have been reached when multiplicity has outstripped the ability to keep the bladder clear by endoscopic destruction of the neoplasms.

Malignant Villous Tumours.—Low-grade transitional cell carcinomas have exactly the same macroscopic appearance as the benign villous growths. As the degree of differentiation decreases, the appearance of the growth alters: (*a*) the villi are stunted, closely packed, and swollen, resembling a cauliflower; (*b*) the growth is sessile (90 per cent. of villous tumours are pedunculated); (*c*) the bladder wall immediately adjacent is more vascular and œdematous; (*d*) the surface of the growth may ulcerate, show areas of necrosis, or become encrusted with urinary salts; (*e*) a bladder tumour accompanied by cystitis is nearly always malignant; (*f*) submucosal lymphatic nodules appear around the growth; (*g*) the depth of invasion of the bladder wall increases, and ureteric obstruction may occur.

Multiplicity of the growth is not necessarily a sign of malignancy, provided the daughter growths are limited to two or three and the rest of the bladder

[1] It has been suggested that nitrosamines excreted in the urine of agricultural workers may be an essential factor in the development of carcinoma in those bladders which are affected by bilharzia.

mucous membrane is normal. When this number is exceeded and/or a patch of œdematous vascular mucosa is seen the growths must be deemed malignant. Unablated, this neoplasm, like other forms of vesical carcinoma, invades the bladder wall (fig. 1416) and gives rise to regional and distant metastases.

Solid tumours are always malignant and grow rapidly through the bladder wall. The surface, instead of being tufted, is bald from the outset. The tumour, which is sessile, becomes lobulated (fig. 1413), is deep red, and bleeds on being touched. It is liable to become covered with a powder of phosphates. Later the surface becomes ulcerated in places. Lymphatic metastases occur earlier than is the case in malignant villous tumours. Solid carcinoma is the second most common form of carcinoma of the bladder.

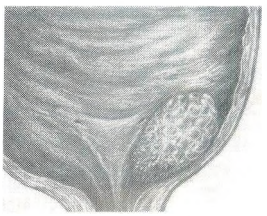


FIG. 1413.—Nodular carcinoma of the bladder, sometimes known as the 'bun-shaped' tumour.

A **carcinomatous ulcer** is similar in appearance to a carcinomatous ulcer elsewhere, and occasionally it arises in a patch of leukoplakia. This type of growth occurs in the base of the bladder and on the trigone. This is the most malignant carcinoma of the bladder. The frequency of carcinoma at various sites is shown (fig. 1414).

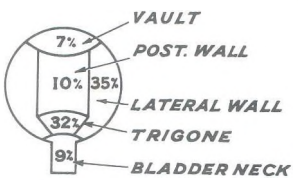


FIG. 1414.—Sites of carcinoma of the bladder. (*American Urological Association Statistics.*)

Primary adenocarcinoma of the bladder is rare (1 per cent. of cases). It is a highly malignant tumour commencing in the fundus of the bladder of relatively young patients, and is believed to arise in the remnants of the urachus. Secondary adenocarcinoma of the bladder from a primary neoplasm in the stomach, colon, lung, rectum, or prostate is more common than one originating in the bladder.

Metastases.—When a carcinoma has invaded the muscle coat (but no further) it is doubtful if metastasis occurs, though local recurrence is common. On the whole, vesical neoplasms are slow to metastasise and the incidence of distant metastases is relatively low, irrespective of the histological characters of the tumour. On the other hand, once the neoplasm has penetrated the musculature and has reached the prevesical fat, it disseminates by the lymphatics, to reach the lymph nodes along the external iliac and common iliac vessels, thence to the para-aortic and mediastinal lymph nodes. By the blood-stream they can be swept to the lungs, liver, bones or the brain.

Clinical Features.—*Papilloma.*—Painless, profuse, paroxysmal hæmaturia is the only symptom for a long period. The hæmaturia may last for a few hours or a few days, and then it ceases. After an interval of weeks or months, bleeding recurs; as time progresses the intervals become shorter and the hæmaturia more severe and of longer duration. Eventually the resulting anæmia may be so severe as to necessitate blood transfusion. Clot retention sometimes occurs, as also dysuria if the growth obstructs the internal urinary meatus. Bimanual rectal or vaginal examination is usually negative.

In 80 per cent. of cases painless hæmaturia remains the only symptom and sign.

Malignant Neoplasm.—In 95 per cent. of cases the initial symptom is intermittent hæmaturia, which eventually becomes continuous and associated with dysuria. It is a sad fact that in spite of this ominous sign more often than not twelve or more months elapse before the patient presents for treatment, and this is not always the patient's fault. Occasionally (in carcinomatous ulcer) the first symptoms are those of severe cystitis. In all cases sooner or later cystitis supervenes, and painful, frequent, blood-stained micturitions are the dominating symptoms in established cases. As time goes on, strangury occurs at the end of each act of micturition. Loin (renal) pain from an obstructed ureter is not uncommon. A late manifestation, due to nerve involvement, is pain referred to the suprapubic region, the groins, perineum, the anus, or down the thighs.

Investigation:

Examination of the Urine.—Culture is essential. Cytology of a 3-hour specimen of urine is proving accurate in diagnosing tumours and also in assessing the histological grade.

Excretory Pyelography.—Occasionally the preliminary plain film shows a faint shadow of an encrusted neoplasm of the bladder. As a rule in cases of papilloma, excretory pyelography displays normal kidney function. When the neoplasm is obstructing a ureteric orifice, dilatation of that ureter will be apparent. In the case of a large neoplasm a filling defect of the bladder (fig. 1415) is likely to be revealed.

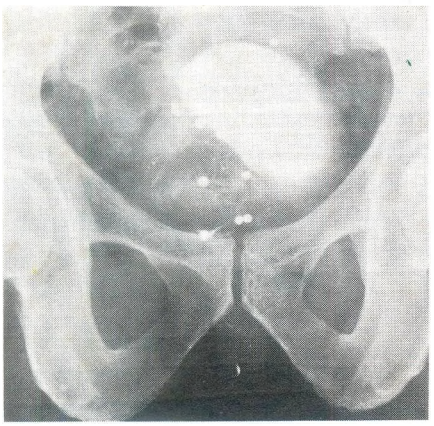


FIG. 1415.—Cystograph in a case of malignant tumour of the right side of the bladder.

Cystoscopy is the mainstay of diagnosis. It should be performed in every case of hæmaturia. The main cystoscopic appearances of bladder tumours have been described under 'Pathology' (p. 1252).

Following cystoscopy, but before any transurethral operation or biopsy is undertaken, the following important examination is made.

Bimanual palpation under general anæsthesia (recto-abdominally in the male and vagino-abdominally in the female), with the patient's abdominal musculature completely relaxed and the bladder absolutely empty, is of salient importance in determining the stage of progression of the neoplasm and the potentialities of a cure by any given method. When a tumour is impalpable, it is most unlikely that it has invaded the bladder wall. While a very large papilloma sometimes can be felt, the tumour is mobile within the bladder (fig. 1416 (T1)). When a localised but

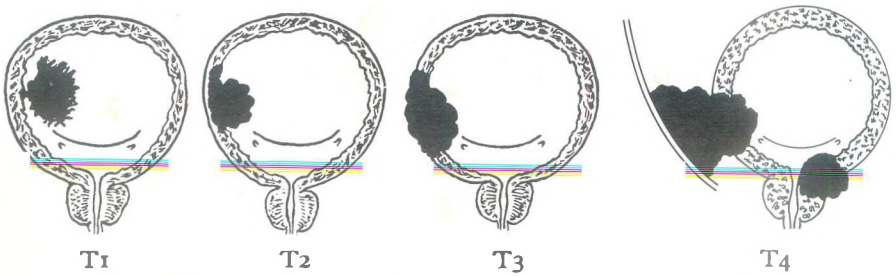


FIG. 1416.—Clinical assessment of the stage of progression of a bladder tumour by bimanual palpation. (*After D. M. Wallace.*)

Staging by the **TNM** classification (cf. p. 53). *Stage T1*—involving subepithelial connective tissue. *Stage T2*—infiltration of muscle superficially. *Stage T3*—full thickness of muscle and possibly perivesical tissues affected, but mobile. *Stage T4*—fixed to adjoining organs or involving prostate.

definite thickening can be felt, but which is as movable as one would expect the bladder to be, it is probable that the bladder wall is infiltrated, but not penetrated (fig. 1416 (T2)). On the other hand, if in addition the mass is larger than would be expected from the cystoscopic appearance but is not fixed, extra vesical spread (fig. 1416 (T3)) has certainly occurred. When a mass is easily palpable, fixed to the bony pelvis (fig. 1416 (T4)) and firm to stony-hard in consistency, palliative measures alone are advisable. The degree of infiltration, as judged by bimanual palpation under anæsthesia, is a more valuable criterion of potential curability than the degree of malignancy revealed by histological studies.

Biopsy.—The removal of a tiny piece of tissue is useless in most instances—the biopsy must be adequate, should include the underlying vesical wall itself, and is best taken with a resectoscope. Alternatively, special biopsy forceps (Riches or Lowsleys), or cystoscopic rongeur forceps (fig. 1395) may be used.

Treatment:

While the treatment of a solitary pedunculated papilloma is standardised except for comparatively minor details in technique, the same being true if a few seedlings are present, as we proceed from the probably malignant to the undoubtedly malignant bladder tumour, so the gulf of controversy as how best to treat the neoplasm widens. At the present time vacillation between ultra-radical operations and wholly conservative measures is to some extent influenced, on the one hand, by the appearance of new isotopes or new methods of applying them, and, on the other, by better methods of diverting the urinary stream after removal of the bladder.

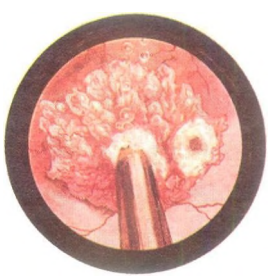


FIG. 1417.—Cystodiathermy of a papilloma of the bladder.

Cystodiathermy.—The widely employed method of destroying benign papillomas is coagulation diathermy applied by an electrode through an irrigating cystoscope. A small solitary papilloma can be treated satisfactorily employing local anæsthesia of the urethra, the patient being ambulatory. For larger growths a general or spinal anæsthetic

is given which permits more massive fulguration, but necessitates in-patient treatment. If daughter growths are present, they are fulgurated first. It is of great importance to attempt eradication of the growth at the first treatment.

The patient is cystoscoped again in twelve weeks by which time necrotic tissue should have sloughed off, and the area epithelialised. Further treatment is carried out if necessary. Larger papillomas can be destroyed more quickly by Kidd's diathermy cystoscope, which has a relatively large ball electrode incorporated on the beak of the instrument, viz.→

Excision with a resectoscope is indicated when the tumour is too large to be destroyed at one session of cystoscopic electro-coagulation, and especially for small papillary carcinomas with little or no infiltration. After the main part of the tumour has been sheared away, electro-excision of the base flush with the bladder wall is accomplished. Cautiously a slight gutter is then made beneath the base of the tumour, into the muscle. Avoidance of downward pressure minimises the risk of perforation of the bladder—the leading danger of this excellent method.

Transvesical Excision of a Large Villous Growth.—The bladder is distended with 1 : 2,000 silver nitrate solution and the viscus is exposed by a suprapubic incision. The contents of the bladder having been evacuated by a trocar and cannula (fig. 1418), which permits suction of the fluid contents so that none is spilled, the edges of the abdominal wall are covered with packs soaked in silver nitrate solution or 60 per cent. alcohol, as a precaution against contamination of the wound by detached tumour cells. The bladder is opened, and the surface of the tumour is lightly coagulated (with the object of preventing detachment of cells). It is then picked up with Fergusson's cupped forceps which surround the tumour. An innocent tumour can be elevated so that there is a substantial pedicle of normal mucous membrane. This is transected with the diathermy needle. After coagulating bleeding-points, the area from which the papilloma sprang is electro-coagulated. In the case of a sessile neoplasm, a diathermy loop electrode (fig. 1419) allows excision of the tumour including a fraction of the muscle layer. This accomplished, the cut surface is lightly fulgurated. The bladder can then be closed with indwelling urethral catheter drainage. Drainage of the prevesical space is necessary.

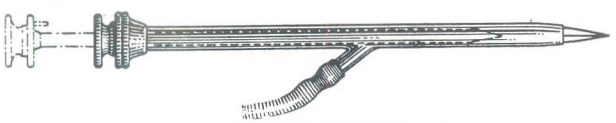


FIG. 1418.

Follow-up.—After the papilloma has been destroyed, endoscopically or by open operation, it is essential that the patient be examined cystoscopically, commencing at three-monthly intervals and, if there is no recurrence, gradually lengthening the interval to one year. In 30 per cent. of cases there is no recurrence. In the remainder there are recurrences, either at the site of the original lesion or in another part of the bladder, but as a rule they can be kept under control with regular cystodiathermy.

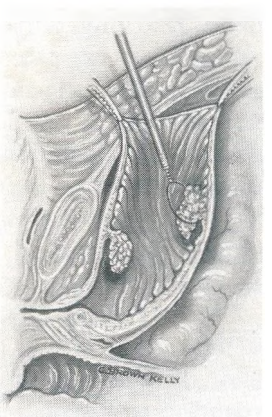


FIG. 1419.—Transvesical excision of a bladder tumour with diathermy loop electrode. *(After A. R. C. Higham.)*

Local Radiotherapy.—When the lesion is not anaplastic, is 4 cm. or less in diameter with evidence of local invasion either on the pyelogram (hold-up or dilatation of one ureter), or on bimanual examination, it is wise to

Francis Seymour Kidd, 1878–1934. Surgeon, The London Hospital.

add local irradiation to the open diathermy excision described above. This may be achieved with radioactive gold grains or tantalum wire, inserted after the initial breach in the mucosa has been closed with 3/0 chromic catgut sutures.

(*a*) **Implantation of Radioactive Gold Grains** (^{198}**Au**).—The gold grains are inserted by means of a repeater gun, which is loaded with a cartridge containing fourteen grains. This isotope has a half life of two and a half days. The grains are implanted according to physical rules depending on the area to be irradiated. Stereoscopic films taken subsequently enable the physicist to determine the dosage the tumour will receive.

(*b*) **Radio active tantalum wire** (^{192}**Ta**) (Wallace) (fig. 1420) has a long half-life (four months), and it therefore can be stored in the hospital ready for use when required, instead of having to be obtained from an atomic pile for each patient. The wire is bent hair-pin fashion with a loop provided at the blunt end. Through the opened bladder the sharp ends of the wire are drawn beneath the base of the tumour by means of twin, hollow needles, mounted on a boomerang needle introducer which enters and emerges some millimetres from the periphery of the neoplasm. The wire having been inserted, a plastic or rubber catheter is passed from the external urinary meatus and its tip is attached to the loop of wire by a stitch. The bladder is then closed. Again, stereo-radiographs enable the physicist to determine how long the wires should be allowed to remain in position. They are removed by withdrawing the catheter.

Deep X-ray Therapy.—The advent of penetrating radiation such as Cobalt €o, and the linear accelerator which enables a high depth dose to be obtained easily, has greatly improved this method of treatment. It is now possible to irradiate the bladder to a high dose level and largely spare the rest of the pelvis, particularly the skin. Undifferentiated carcinomas are best treated by this method and the local results are now good. It is important, however that the tumour should be accurately localised, employing cystography.

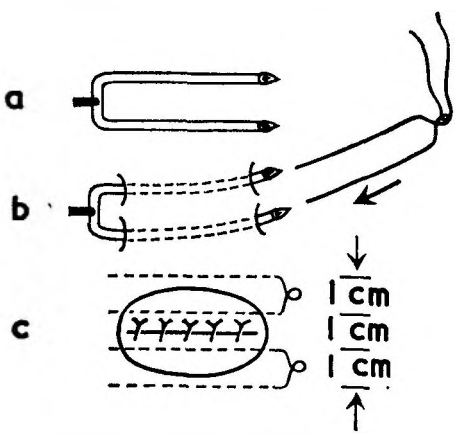


FIG. 1420.—Insertion of radioactive tantalum wire. (a) The curved twin hollow needles. (b) Insertion of the wire loop via the curved needles. (c) Position of wires relative to site of tumour pedicle.

Excision of a Part or the Whole of the Bladder:

Partial Cystectomy (*syn.* Segmental Resection).—Its disadvantage is that many malignant tumours of the bladder are situated near the trigone, where the operation is inapplicable. That part of the bladder containing the growth, together with a surrounding inch of the whole thickness of healthy bladder wall, is excised. When the neoplasm is situated near a ureteric orifice, the ureter should be divided before the excision is carried out, and after the excision has been completed, reimplantation of the ureter into the bladder is performed. The operation is of most value in small lesions involving muscle in the vault of the bladder.

Subtotal Cystectomy.—Like total cystectomy, the operation is conducted intraperitoneally. The whole of the bladder is mobilised to the entrance of the ureters into the bladder wall posteriorly and to within a finger's-breadth of the internal urinary meatus anteriorly. A new bladder is then constructed, utilising a segment of ileum with its blood-supply intact, the graft being split along its length. This operation has a very limited application; usually a growth extensive enough to necessitate it has encroached on the trigone.

Total Cystectomy.—The indications for total cystectomy are somewhat controversial. Prior to this operation it is wise to assess the para-aortic lymph nodes by lymphography. Spread of tumour to these nodes implies that total cystectomy can only be palliative. Provided the patient is fit to withstand this operation it would be justifiable to undertake it in the following circumstances: (1) Profuse papillomatosis

David Mitchell Wallace, Contemporary. Professor of Urology, University of Riyadh, Saudi Arabia.

uncontrolled by other methods; (2) Recurrence or residual growth after adequate radiotherapy; (3) Carcinoma involving the bladder neck in either sex; (4) Recurrences in a small contracted bladder that has resulted from oft-repeated conservative operations; (5) Recurrence after partial cystectomy.

Operation.—With the bladder empty, a lower left paramedian incision is made, and the incision is carried down far enough to expose the symphysis pubis. The peritoneum having been opened, the iliac and para-aortic lymph nodes and the liver and the kidneys are palpated. Having decided to proceed, the operating table is tilted and the ureters are isolated and divided as low as possible in the same way as described on p. 1261. A suitable catheter is passed along the lumen of each ureter, and the urine is allowed to drip into a bottle placed away from the operation area. Hæmorrhage is reduced by ligating the anterior division of the internal iliac arteries.

The bladder and its overlying peritoneum, together with the prostate and all the fascia and fat surrounding these structures (including the uterus in the female), and the urethra, are removed *en bloc*.

One of the most important steps of the operation is to find the correct plane of cleavage between the prostate and the rectum. This is accomplished by following the vas deferens proximally. *En route* this will lead to the superior and inferior vesical vascular pedicles, which are ligated and divided.

For total cystectomy to be truly radical, the lymph vessels and nodes of the pelvis at least up to the bifurcation of the common iliac artery should be included, but of necessity this increases the operating time.

Diversion of Urine.—For methods of diversion see below.

URINARY DIVERSION

This chapter closes with an account of the principles of this important subject, and includes indications, the methods employed and their attendant problems, and some operative details.

Indications.—Diversion of the urine may be either (*a*) a temporary expedient to relieve distal obstruction, or (*b*) a permanent procedure when (1) the bladder has been removed, (2) the sphincters of the bladder have been damaged or have lost their normal neurological control, (3) there is an incurable vesico-vaginal fistula, (4) there is an irremovable obstruction, and (5) in cases of ectopia vesicæ (p. 1224).

Methods of Urinary Diversion (fig. 1422).

The diversion may be achieved by any of the following methods, but the choice in each case will be decided largely by the primary disease.

Pyelostomy or nephrostomy (p. 1173), or urethrostomy (with indwelling catheters).

Cutaneous ureterostomy.

Suprapubic cystostomy (with in-dwelling catheter).

Cutaneous vesicostomy (cystostomy).

Suprapubic displacement of the urethra.

Uretero-sigmoidostomy (*a*) in continuity, (*b*) making a rectal bladder and colostomy.

Trigono-colostomy (*a*) in continuity, (*b*) into an isolated conduit.

Ureteric transplantation into an isolated ileal or colonic conduit.

Diversion of urine immediately raises the following problems:—1. the collection of the urine, 2. stricture-formation at any anastomosis, and 3. reflux and reabsorption of urinary solutes. The problems of infection are intimately related to all three.

Collection of the Urine

(*a*) **Catheters.**—In the past, indwelling catheters have been used for permanent diversion. They invariably result in infection and they often become blocked by phosphatic encrustation. There is now *no place for permanent catheter drainage* of the kidneys or ureters. For temporary drainage the tubes should be of latex or soft silastic (polyvinyl plastics are irritant and predispose to a stricture).

Bladder Drainage.—In elderly patients unfit for prostatectomy and in some terminal cases of carcinoma of the prostate, an indwelling Foley catheter (fig. 1381), changed every three weeks, is a satisfactory method of drainage. Alternatively, a St. Peter's Hospital suprapubic drainage apparatus (fig. 1421) incorporating a straight catheter which the patient or nurse changes daily is acceptable.

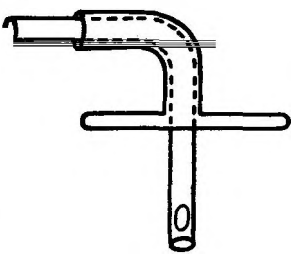


FIG. 1421.—St. Peter' Hospital suprapubic drainage apparatus.

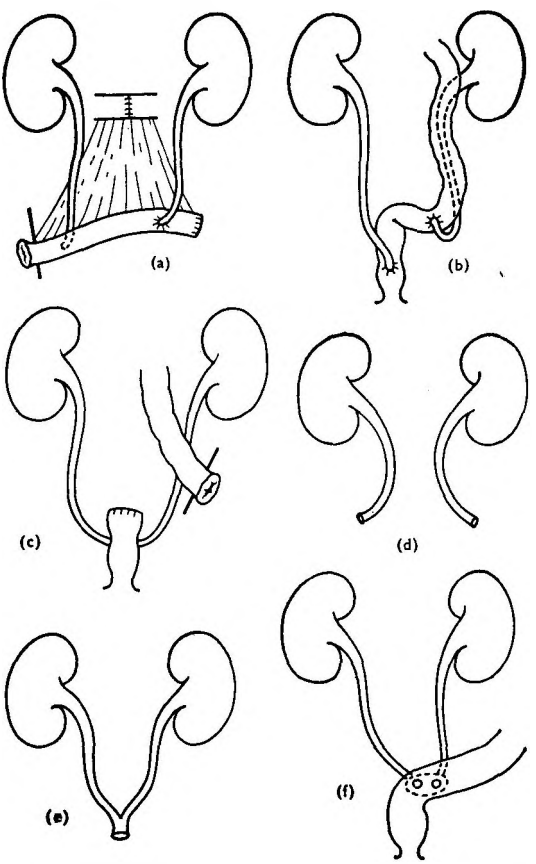


FIG. 1422.—Diversion of urine. Favoured methods: (a) Ileal conduit, (b) Uretero-sigmoidostomy, (c) Rectal bladder with terminal colostomy, (d) Bilateral cutaneous ureterostomies, (e) Joined ureters—cutaneous opening, (f) Trigono-colostomy.

(*b*) **Cutaneous Stomas:** *Suprapubic Vesicostomy or Urethrostomy.* — Collection from a formal suprapubic vesicostomy (cystostomy), or from the urethra displaced on to the abdominal wall (e.g. for cases of multiple sclerosis in a woman), may be unsatisfactory because the local incisions result in creases which make it difficult to apply a water-tight collecting appliance.

Cutaneous ureterostomies are very liable to stricture formation. In addition two openings (fig. 1422 (d)) and appliances add to the patient's burden. Mobilisation of the ureters and the making of a central abdominal stoma (fig. 1422 (e)) can be successful, but such a degree of mobilisation is attended by the high risk of sloughing of the distal ureters due to impairment of blood-supply.

St. Peter's Hospital for Stone, London, founded in 1860.

Ileal or Colonic Conduit.—At present the most generally useful form of diversion is to implant each ureter with as little mobilisation as possible into an isolated segment of gut (ileum or colon), which conducts the urine onwards to a cutaneous stoma (fig. 1422 (a)). Urine is then collected in an ileostomy bag. This form of diversion limits infection and avoids the problems of reabsorption of urine as contact-time with the mucosa is minimal. In some cases in which the pelvis has been subjected to radiation the lower ureters may be unhealthy. A high division with insertion of the ureters into an ileal loop above the roof of the mesentery may be wiser (fig. 1423), (Wallace).

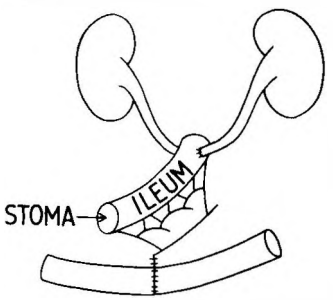


FIG. 1423. (*After D. Wallace, F.R.C.S.*)

Siting of Stoma.—The site for the stoma must be chosen before operation. The ileostomy ring and bag are applied to the abdominal wall, and by trial and error the point at which the belt can support the ring and bag at all times and in all positions is found. Having checked this by sticking on a bag full of water and asking the patient to perform the type of movements which his work will entail, the site of the future stoma is marked indelibly on the skin.

(*c*) **Colon and Rectum.**—The advantage of diverting urine into the colon is that no collecting apparatus is necessary (fig. 1422 (b) and (f)). Clearly, however, the rectal sphincter must be competent. Before any uretero-sigmoidostomy is undertaken, the patient must prove that he can control at least 200 ml. of fluid in the rectum. The disadvantage of the operation is that the renal tract is exposed continuously to infection from the fæces. This can be avoided by establishing a terminal left iliac colostomy, and closing the upper rectum to make a rectal bladder (fig. 1422 (c)). It prevents the urine refluxing retrogradely round the colon to the cæcum, diminishes reabsorption (see below), and protects renal function.

Stricture Formation

Uretero-sigmoidostomy was first used by Chaput (1894). Subsequent modifications included those made by Coffey and Grey Turner. In these methods the ureters were cut obliquely and pulled into the gut by a stitch—the ends were not stitched to the gut wall. Stenosis was not uncommon. Nesbit, Cordonnier, and Leadbetter all recognised that these strictures could be prevented by anastomosing mucosa to mucosa (fig. 1424).

Reflux of Urine and Reabsorption of Urinary Solutes

Reflux of Urine.—Pressure within the colon can interfere with drainage, and so force air, fæces, and organisms up to the renal pelvis. Daniel has demonstrated that a high intra-colonic pressure (over 24 cm water) results in all these complications being more likely. For many years it had been noted that the left kidney was much more likely to cease functioning after uretero-sigmoidostomy than the right kidney. As the left ureter is implanted into the sigmoid and the right often in the upper rectum, the former is more likely to be subjected to higher intra-colonic pressures. A sigmoid myotomy performed from the upper rectum to the splenic flexure may help to overcome this problem.

Henri Chaput, 1857–1919. French Surgeon. *Robert Calvin Coffey, 1869–1933. American Surgeon.*
George Grey Turner, 1877–1951. Professor of Surgery, Postgraduate Medical School, London.
Reed M. Nesbit. Formerly Professor of Surgery, University of Michigan Medical School, Ann Arbor, U.S.A.
Justin Joseph Cordonnier, Contemporary. Professor of Urology, Washington University School of Medicine, St. Louis, Missouri, U.S.A.
Wyland F. Leadbetter, Contemporary. Clinical Professor of Surgery, Harvard Medical School, Boston, U.S.A.
Owen Daniel, Contemporary. Consultant Surgeon, Royal Alexandra Hospital, Rhyl, Wales.

Reabsorption of Urinary solutes depends upon two factors, (1) reflux of urine round the colon and even into the terminal ileum (fig. 1422 (b) and (f)), (2) because the colon is acting as a reservoir, the urine is in contact with gut wall for a longer period. Biochemical disturbances are rare if a rectal bladder is made (see above).

The biochemical changes associated with transplantation into the colon are due to a combination of reabsorption of chlorides and urea, and progressively diminishing tubular function as a result of chronic pyelo-nephritis. Diarrhœa with loss of potassium-containing mucus may exacerbate the loss of potassium.

The typical changes are a hyperchloræmic acidosis with potassium depletion, and they occur in every patient with a uretero-sigmoid diversion. When severe, the patient develops loss of appetite, weakness, thirst, and diarrhœa. He becomes listless, and respirations are rapid, deep, and unremittent. Coma may ensue. Mild acidosis, unrecognised over a long period, produces osteomalacia from calcium reabsorption. Bone pain and even pathological fractures occur.

Renal impairment from pyelonephritis and reabsorption from the mucosa are infrequent complications if the diversion is into an ileal or colonic conduit.

Treatment.—(*a*) *Prevention.*—Patients should be instructed to empty the rectum two-hourly by day, and in cases where acidosis is present, a rectal tube should be inserted at night to drain the urine continuously. The patient should avoid added salt in the diet, and he should take a mixture of potassium citrate and sodium bicarbonate t.d.s. (two grams of each either as crystals or tablets). Regular biochemical analyses, including calcium, are required. The patient should be told to report if he loses his appetite or develops weakness.

(*b*) *In established hyperchloræmic acidosis*, the patient should be given suitable doses of sodium bicarbonate (8·4% = 1 mEq/ml = molar) intravenously to correct the pH (p. 94), with suitable amounts of potassium, also intravenously, if necessary. Furadantin, ampicillin, or the appropriate antibiotic should be given to correct the inevitable pyelonephritis which will be present. Later, conversion to an ileal conduit should be considered.

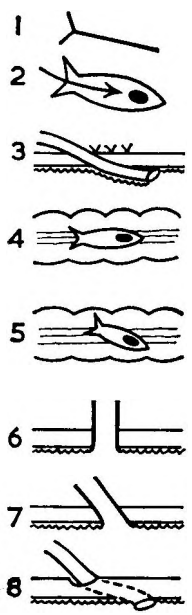


FIG. 1424.—Methods of uretero-colic anastomosis. 1. Incision in the sero-muscular layer, which, on dissection, exposes an area of submucosa and mucosa which looks rather like a fish. 2. The ureter is laid along the 'fish' and inserted through the opening into the lumen made in the 'fish's head'. 3. Lateral view of completed anastomosis. 4. Coffey type. 5. Grey Turner type. 6. Nesbit, 7. Cordonnier, and 8. Leadbetter types entail a careful mucosa to mucosa anastomosis. The last named includes a submucosal tunnel, and is probably the best anastomosis for preventing stricture formation and reflux.

Operative Details

Uretero-colic Anastomosis.—The preparation of the colon is the same as that described on p. 1022. The abdomen is opened by a right lower paramedian incision. The patient is then placed in the Trendelenburg position and the right ureter is sought as it crosses above the bifurcation of the common iliac artery. An incision is made through the peritoneum overlying the medial side of the ureter, thus avoiding damage to the ureteric vessels which always approach the duct from the outer side (Daniel), and the ureter is identified and dissected from its bed towards its entry into the bladder. The ureter is then divided and its distal stump ligated. The proximal end is trimmed obliquely and split upwards for one centimetre. An incision 3 cm. long is made in the anterior wall of the colon and the peritoneal and muscular coats are divided, but not the mucous membrane (fig. 1424(3)). An incision is made into the extreme lower end of the exposed mucous membrane and the full thickness of the ureteric wall is joined by interrupted 4/0 chromic catgut sutures to the mucosal opening. The incision in the outer coats of the bowel is approximated over the ureter (Leadbetter anastomosis). Suturing the peritoneal incision around the

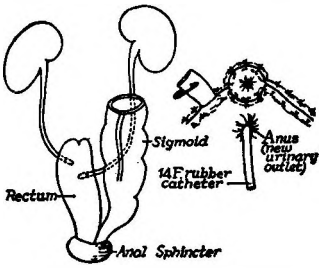


FIG. 1425.—Lowsley's operation.

ureteric implant extraperitonealises the site, and a drain is left down to the area. The left ureter is implanted into the colon above in a similar manner. A full-sized Foley's catheter (fig. 1381) is inserted through the anus no farther than the rectal ampulla, the balloon then being inflated. This permits all urine to be measured until the third day, when the catheter is removed. There must be a full fluid intake. If oliguria or anuria supervene, treatment is as described on p. 1168.

Uretero-ileostomy (Ileal Loop Conduit).—A coil of ileum, approximately 15–25 cm long, and one foot from the ileo-cæcal valve, with its blood supply intact, is isolated. The left ureter is brought through the pelvic mesocolon, and after the ureter has been cut obliquely, end-to-side anastomosis is performed between the ureter and the intestine. The right ureter is also cut obliquely, and it is joined to an elliptical opening made in the side of the coil. In each case the anastomosis may be effected by the method of Nesbit (fig. 1424(6)). The coil of intestine is tacked lightly to the peritoneum of the anterior abdominal wall at the level of the pelvic brim. The distal end of the coil is brought out through a stab incision in the right lower abdomen, and, because of the anchoring of the intestine to the peritoneum, the opening is made a little lower than is the case when ileostomy is performed for ulcerative colitis. The method of anchoring the ileostomy opening to the abdominal wall, and the after-care of the orifice so formed, in no way differs from that described on p. 1007.

Lowsley's Operation.—The rectosigmoid junction is divided completely and its lower end closed. The ureters or the trigone are implanted into the excluded lower segment, so that the rectum functions as a bladder. The sigmoid colon is mobilised sufficiently for it to be drawn through the perineum immediately anterior to the anus. Thus both the original anal canal and the new anal canal are encompassed by the external sphincter ani muscle (fig. 1425).

Oswald S. Lowsley, 1884–1955. Director of Urology, James Buchanan Brady Foundation, New York Hospital.

CHAPTER 58

THE PROSTATE AND SEMINAL VESICLES

Embryology.—The prostate arises from the primitive urethra as a series of solid buds; in a matter of weeks these become canalised. The surrounding mesenchyme becomes the muscular and connective tissue of the gland. The buds are arranged into five groups—anterior, middle, posterior, and two lateral. These are the forerunners of the lobes of the prostate.

Skene's tubules opening on either side of the female urethral orifice are the homologues of the prostate.

Surgical Anatomy.—The prostatic glands (fig. 1426) lie in the fibromuscular stroma and their ducts open into the posterolateral grooves on either side of the verumontanum. The epithelium is columnar. Commencing peripherally, and passing centrally, beneath the anatomical capsule lie the long branched **prostatic glands proper.** This region is named the carcinomatous zone. Beneath this envelope, and separated from it by an indefinite capsule, lies another mass of secreting elements, also branched. These are the **submucosal glands**—and the zone that they occupy is known as the adenomatous zone. Still nearer the urethra are the unbranched **urethral glands** whose mouths open directly into the urethra.

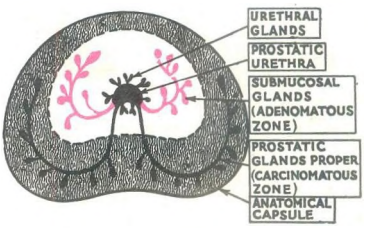


FIG. 1426.—Distribution of the normal glands of the prostate, transverse section. *(After J. C. B. Grant.)*

Into the prostatic urethra, therefore, open the prostatic ducts proper, the ducts of the submucosal and mucosal glands, as well as the common ejaculatory ducts and the prostatic utricle. No wonder that chronic infection of the prostatic urethra is difficult to eradicate!

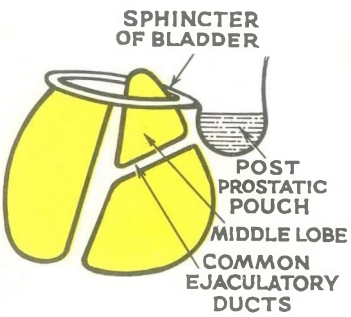


FIG. 1427.—The surgical subdivisions and relationships of the prostate.

Certain relationships and divisions of the prostate as seen in sagittal section are set out in fig. 1427. The middle lobe is that part of the prostate included between the common ejaculatory ducts and the prostatic urethra: it contains more secretory glandular element (subcervical glands, as opposed to muscle and fibrous tissue) than the lateral lobes.

An enlarged prostate is invested with three capsules: (1) The compressed outer zone = *the false capsule*; (2) The anatomical capsule = *the true capsule*; and external to this (3) *The prostatic sheath of pelvic fascia.* Between the anatomical capsule and the prostatic sheath lies the prostatic venous plexus (fig. 1428). The prostatic sheath is contiguous with the strong fascia of Denonvilliers that separates the prostate and its coverings from the rectum.

Physiology.—That the prostate is purely a genital organ is evinced by the fact that in such animals as manifest a seasonal sexual life, the organ is rudimentary except during the rutting season. That normal adult prostatic epithelium undergoes atrophy after castration was known to John Hunter.

Hormonal Influences.—The prostate is governed by two testicular hormones, one male (androgenic) and the other female (œstrogenic). Normally the preponderant

Alexander Johnston Chalmers Skene, 1838–1900.. Professor of Gynæcology, Long Island College Hospital, New York, U.S.A.
Charles Pierre Denonvilliers, 1808–1872. Professor of Anatomy, and later of Surgery, Paris.
John Hunter, 1728–1793. Surgeon, St. George's Hospital, London.

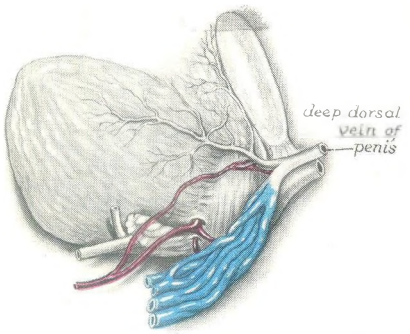


FIG. 1428.—The prostatic venous plexus.

testicular hormone is androgen, which is supplemented by androgens secreted by the adrenal glands (fig. 1429).

Œstrogens cause retrogressive changes in the testes and prostate gland. For example, most capons are produced by implanting œstrogen pellets into the neck of the bird rather than by castration.

Elaboration and Secretion of Acid Phosphatases.—Enzymes that split organic phosphates and are most active about pH5, are present in many human tissues, but their concentration in the adult prostate is several hundred times greater than in any other organ or tissue. This high level is not achieved until after puberty. While the function of this enzyme is speculative, it is highly suggestive that the acidity which is most favourable for its action corresponds to that of the vagina at the time of ovulation. Most of the acid phosphatase secreted by the prostate drains along the prostatic ducts into the urethra, so that the blood level of this enzyme remains low. When the cells producing this enzyme cannot discharge their products externally, the serum acid phosphatase rises.

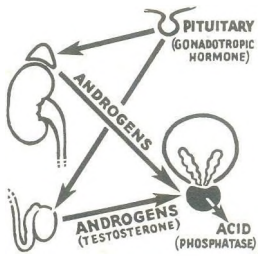


FIG. 1429. — Preponderant hormonal control of the prostate. *(After L. N. Pyrah, F.R.C.S., Leeds.)*

Acid phosphatase estimations are made on plasma or serum, free from hæmolysis. The blood sample must be taken in the morning before breakfast, and on no account after a heavy meal, since lipoid-rich serum gives inaccurate estimations. Digital rectal examination has now been shown not to affect the acid phosphatase level in the blood. The normal range of findings is 0–5 King-Armstrong units per 100 ml. of serum.

The serum acid phosphatase is usually raised in carcinoma of the prostate *with metastases*, but is seldom above normal if the growth is confined to the gland, and almost never in benign prostatic hyperplasia. On the other hand, slightly increased values are not uncommon (1) in acute prostatitis, (2) Paget's disease of bone, (3) hepatic cirrhosis.

BENIGN ENLARGEMENT OF THE PROSTATE

"*When the hair becomes grey and scanty, when specks of earthy matter begin to be deposited in the tunics of the artery, and when a white zone is formed at the margin of the cornea, at this same period the prostate gland usually—I might perhaps say invariably—becomes increased in size.*" (SIR BENJAMIN BRODIE.)

Benign enlargement of the prostate usually occurs in men[1] over fifty years of age; most often between sixty and seventy. In Indians prostatic enlargement is less frequent, and occurs more often in a younger age-group; in Negroes it is rare. The reason for these discrepancies is unknown.

Theories of causation of benign enlargement of the prostate:

The Hormonic Theory.—As age advances the male hormone diminishes while the quantity of the œstrogenic hormone is not decreased equally. According to this

[1] It is interesting to note that the ageing dog is prone to prostatic hyperplasia but, unlike his master, does not often develop retention of urine. Due, however, to the bulging upwards of the enlarged prostate the dog's rectum is compressed. This engenders a feeling of continuous fullness in the rectum, and induces painful straining; symptoms are relieved by stilbœstrol. The prostates of eunuchs are small and completely under-developed, and benign enlargement of the prostate is unknown.

Earl J. King, 1901–1962. Professor of Chemical Pathology, University of London (Postgraduate Medical School) and Arthur Riley Armstrong, Contemporary. Director of Laboratories, Hamilton Health Association, Ontario, formerly co-workers at the Department of Medical Research, Banting Institute, University of Toronto.
Sir James Paget, 1814–1899. Surgeon, St. Bartholomew's Hospital, London.
Sir Benjamin Collins Brodie, 1783–1862. Surgeon, St. George's Hospital, London.

theory the prostate enlarges because of predominance of the œstrogenic hormone. Prostatic enlargement can be regarded as involutionary hyperplasia akin to fibro-adenosis of the breast, due to a disturbance of the ratio and quantity of the circulating androgens and œstrogens.

The Neoplastic Theory postulates that the enlargement is a benign neoplasm. As the prostate is composed essentially of fibrous tissue, muscle tissue, and glandular tissue, the neoplasm is a *fibro-myo-adenoma.*

Pathology.—Hyperplasia affects the glandular elements and connective tissue, but in variable degrees. In this respect the changes are very similar to those occurring in the breast in 'chronic mastitis', where adenosis, epitheliosis, and stromal proliferation are seen in differing proportions. Benign adenomatous hyperplasia affects the submucous group of glands, forming a nodular enlargement—the typical 'lateral' lobes—which compresses the external group of glands into a false capsule (fig. 1426).

As the prostate enlarges extravesically, it tends to displace the seminal vesicles, so that instead of lying on the base of the bladder, these structures become a direct posterior relation of the upper limit of the prostate. When the hyperplasia affects the subcervical glands a 'middle' lobe develops which projects up into the bladder within the internal sphincter (fig. 1427). Sometimes both lateral lobes also project into the bladder, so that when viewed from within, the sides and back of the internal urinary meatus are surrounded by an intravesical prostatic collar.

SECONDARY EFFECTS OF PROSTATIC ENLARGEMENT

Urethra.—That portion of the urethra lying above the verumontanum becomes elongated, sometimes to as much as twice its normal length. The canal is compressed laterally, so that it tends to become an antero-posterior slit. The normal posterior curve may be so exaggerated that it requires a coudé, bicoudé, or even a fully curved metal catheter to negotiate it. When one lateral lobe is enlarged predominantly, lateral distortion of the prostatic urethra occurs.

Bladder.—The musculature of the bladder hypertrophies to overcome the obstruction. When viewed from within, bands of muscle fibres can be seen—*trabeculation* (fig. 1430). Between these hypertrophied bundles there are shallow depressions, i.e. *sacculations.* Sometimes one of the saccules (rarely two or more) continues to enlarge, and forms a *diverticulum.*

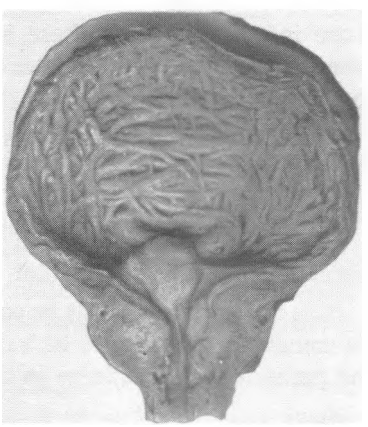


FIG. 1430.—Trabeculation of the bladder from prostatic obstruction. (*The late Professor K. A. L. Aschoff, Freiburg.*)

When the middle lobe projects upwards into the bladder it acts as a dam to the last ounce or more of urine, which remains in the post-prostatic pouch. Calculi are prone to form in this stagnant pool of urine.

The enlarged prostate may compress the prostatic venous plexus; the resulting congested veins (vesical ' piles ') at the base of the bladder are apt to cause hæmaturia.

Unless the obstruction is relieved, a time is reached when bladder hypertrophy gives place to atony, the tired muscle making no attempt to overcome the obstruction.

Ureters and Kidneys.—Increasing intravesical pressure, or in some cases direct pressure of the intravesical portion of the prostate on the ureteric orifices, causes gradual dilatation of the ureters, followed by some degree of bilateral hydronephrosis. When bladder hypertrophy wanes, the sphincter mechanism around the ureteric orifices ceases to function, permitting reflux of urine from the bladder into the dilated ureters, with increasing damage to the renal parenchyma. As a result of ascending infection, or more rarely from the blood-stream, acute or chronic pyelonephritis supervenes.

Sexual Organs.—In the early stages of prostatic enlargement there is increased libido. Later, impotence is the rule.

Clinical features are variable and depend upon the lobes affected. *Frequency* is the earliest symptom. At first it is nocturnal, the patient being obliged to get up to micturate twice or more during the night, usually commencing at 2 or 3 a.m. Frequent micturition at this stage is probably due to vesical introversion of the sensitive prostatic mucous membrane by the intravesical enlargement of the prostate. The frequency becomes progressive, and is then present both by night and by day. When the vesical sphincter becomes stretched, a little urine escapes into the normally empty prostatic urethra, causing an intense reflex desire to void, and *urgency* is added to the frequency. Later, as residual urine increases, frequency becomes more and more in evidence, and there is terminal dribbling. Finally, in neglected cases, frequency is further encouraged by cystitis, and polyuria due to renal insufficiency.

Difficulty in Micturition.—The patient notices that he must wait patiently for urination to start; it is useless to strain (cf. urethral stricture and fibrous prostate—pp. 1296 and 1273).

The stream is variable, often weak, tending to stop and start, and dribble towards the end of micturition.

Pain occurs with cystitis or acute retention of urine. When hydronephrosis commences there may be a dull pain in the loins. A feeling of weight in the perineum, or a fullness in the rectum, are occasional complaints.

Acute retention of urine is sometimes the first symptom to compel the patient to seek relief because of the intense pain it produces. The postponement of micturition is a common precipitating cause (e.g. a non-corridor train), as also is indulgence in alcoholic liquors, particularly when he goes out of doors on a cold night, as congestion of internal organs then tends to occur. Confinement to bed on account of some intercurrent illness or operation is another cause of acute retention of urine (p. 1227).

Retention with Overflow.—The patient complains that urine constantly dribbles away. It is exceptional for him to have noticed the swelling caused by the distended bladder and he experiences no pain (fig. 1431).

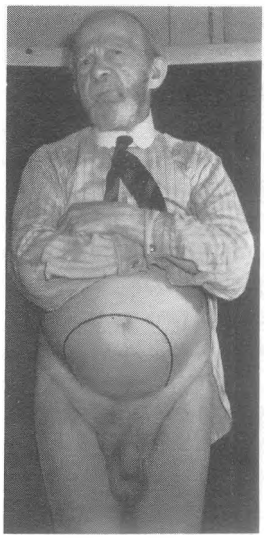


FIG. 1431.—Retention with overflow. The patient's only complaint was that he 'wet his trousers'. Note the distended bladder.

Hæmaturia.—A drop of blood at the beginning or end of micturition is not unusual. Occasionally alarming hæmaturia occurs from a ruptured prostatic vein (p. 1266), or from an erosion on the enlarged prostate itself. Incrimination of an enlarged prostate as the source of hæmaturia before excluding other causes has resulted in the term 'decoy' prostate.

Renal Insufficiency.—The patient presents himself with signs of renal failure (p. 1169).

Examination.—The patient lies on a couch and the abdomen is examined. In patients with a long history, varying degrees of chronic retention of urine will be found on palpation, percussion, and sometimes on inspection. The renal areas are palpated for tenderness and possible enlargement of the kidneys. The state of the tongue is noted; a dry brown tongue and a urine of low specific gravity indicate a considerable degree of renal insufficiency. The external urinary meatus is examined to exclude stenosis, and the epididymes are palpated for signs of inflammation.

Rectal examination is carried out and, in the absence of a full bladder, bimanually in the dorsal position. In benign enlargement of the lateral lobes, increase in their size is evident. They are smooth, convex, and typically elastic, but the fibrous element may give the prostate a firm consistency. The rectal mucosa can be made to move over the prostate. On bimanual palpation an intravesical lobe can sometimes be felt. By exerting pressure on the apex of the prostate by the finger in the rectum, it will be found that a gland which is the seat of benign enlargement possesses a definite degree of mobility. Residual urine may be felt as a fluctuating swelling above the prostate. It should be noted that if there is a considerable amount of residual urine present, it pushes the prostate downwards, making it appear larger than it is.

When possible, the act of micturition should be watched. Loss of projectile power is significant. The urine is passed into two glasses; mere inspection of it is often helpful. It is later examined chemically, and a mid-stream specimen may be sent for bacteriological examination.

The nervous system is examined to eliminate a neurogenic lesion. Tabes and disseminated sclerosis may give symptoms similar to those of prostatic obstruction.

The Mictiograph.—A graphic recording of a patient's stream rate and volume of urine can be obtained and is most helpful in determining the degree of outflow obstruction.

Examination of the Blood.—A blood urea estimation, a blood count, and a Wassermann reaction are all important, the first two being essential.

Excretory pyelography is omitted only if the patient shows clinical signs of renal failure, or if the blood urea estimation is high. It affords a great deal of information without resorting to instrumentation and it is an excellent test of renal function. The preliminary radiograph may show a renal calculus, or a stone in the bladder, or in the diverticulum.

The pyelogram will exclude or confirm the presence of hydroureters and hydronephroses; a vesical diverticulum may be shown and if a film is taken after micturition, residual urine will be revealed. Sometimes an intravesical enlargement of the prostate is outlined by the medium. Nevertheless, complete reliance must not be placed on radiological evidence of residual urine as the patient may have difficulty in passing urine in the radiological department, especially if a female radiographer is nearby!

Cysto-urethroscopy.—Cysto-urethroscopy is an essential preliminary to prostatectomy, especially when the operation is not transvesical, to exclude the presence of a growth, a diverticulum, or non-opaque stones. It may also be necessary in order to make a diagnosis when the gland is small, and sometimes to distinguish between a urethral stricture and a bladder-neck obstruction.

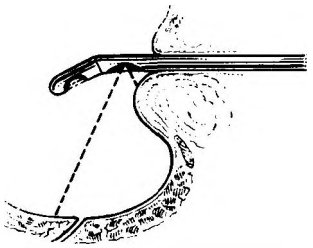


FIG. 1432.—Marion's sign.

The patient must empty his bladder just prior to the examination, so that the amount of residual urine present in the bladder can be measured. After irrigation, if necessary, the bladder is distended and examined. Trabeculation and sacculation are the significant findings. The presence of cystitis will have been anticipated by the examination of the urine. Normally the posterior edge of the internal urinary meatus and a ureteric orifice cannot both be seen in the same cystoscopic field. In cases of moderate enlargement of the middle lobe this becomes possible (Marion's sign, fig. 1432). Should the intravesical projection be considerable, the ureteric orifices and the inter-ureteric bar are hidden completely beneath the prostatic shelf.

The Management of Retention

Acute retention initially is very painful. Chronic retention is painless. It is safe to decompress an acute retention rapidly. It may not be safe to decompress a chronic retention rapidly. If the kidneys are hydronephrotic, reactionary hyperæmia may lead to bleeding from the kidney, impaired blood flow to the tubules, and an acute tubular necrosis. In chronic retention an urgent blood urea estimation is essential, and if above 70 mg. per cent. slow decompression should be the rule. Below this level it is better to avoid instrumentation until the cystoscopy immediately prior to prostatectomy.

Catheterisation.—Where there is a possibility that the catheter may be left indwelling, wash and shave the pubic, perineal, and scrotal regions. Anæsthetise the urethra with 1 per cent. Xylocaine combined with 0·5 per cent.

August von Wassermann, 1866–1925. Director of the Kaiser Wilhelm Institute for Experimental Therapeutics, Berlin.
Jean Baptiste Camille Georges Marion, 1869–1960. Professor of Urology, Faculty of Medicine, Paris.

chlorhexidine in a methyl-cellulose base. Allow three minutes for the anæsthetic and the antiseptic effects to take place. With strict aseptic precautions pass a size 8, 10, or 12 F. plastic catheter by a non-touch technique (Gibbon-type if it is to be left indwelling, p. 1231). Collect a specimen of urine for culture.

Slow decompression can be obtained by joining such a catheter to a bottle and arranging the end of the inlet into the bottle at such a height, 18 to 20 cm, above the pubis so that urine flows over only when the patient coughs or moves, thus raising his intra-abdominal pressure. The bottle can be lowered 2·5 cm every four hours. Alternatively a 'gate' clip can be applied to the drainage tube to permit a rapid drip into the bottle. The clip is released every hour or so. If the urine flow increases, the clip is re-applied, and if no urine flows it is removed, for the bladder is now empty and must not be allowed to refill because this encourages the growth of organisms in the bladder.

In cases of acute retention, especially where there is a history of overholding the urine, as on a long journey or after drinking alcohol, the bladder should be emptied and the catheter removed. Micturition may then be re-established. Failure to do so confirms the need for prostatectomy.

Suprapubic Puncture.—If there is a long history of prostatism, and early prostatectomy is decided upon, relief of pain can be achieved by suprapubic needle puncture, running in a length of polythene tube with multiple side holes and connecting this to a sterile drainage bottle or bag.

All catheters should be connected by straight connections to sterile tubes and drainage bottles, which should contain 120 ml. of 40 per cent. formalin solution. Air vents should be protected by formalin tablets (fig. 1383).

Indications for Operation

(1) *Prostatism.*—Increasing difficulty in micturition, with considerable frequency day and night, delay in starting, and a poor stream—such are the usual symptoms for which prostatectomy is advised. It must be stressed that *frequency alone* is never an indication for prostatectomy, and also that the natural progression of benign prostatic enlargement is variable and rarely gets worse after ten years. A patient with symptoms for nine years is unlikely to get much more severe symptoms, while a similar degree of symptoms reached in three years is a greater indication for surgery. This fact may be helpful when considering the correct advice to give to elderly patients possibly suffering from other disease.

(2) *Acute retention which is unrelieved* by passing a catheter, emptying the bladder, and immediately removing the catheter.[1]

(3) *Chronic Retention.*—A residual urine of 200 ml. or more. A raised blood urea, hydroureter, or hydronephrosis demonstrated on urography, and uraemic manifestations.

[1] Wilson Hey recommended immediate prostatectomy as the treatment of choice in such patients who are usually fit, thus avoiding the dangers of infection by instrumentation and the increased liability to venous thrombosis if catheter drainage and bed-rest are instituted.

Wilson Harold Hey, 1882–1956. Surgeon, Royal Infirmary, Manchester, England.

(4) *Complications.*—Stone, infection, and diverticulum formation, which are often present in varying combinations.

(5) *Hæmorrhage.*—Occasionally venous bleeding from a ruptured vein overlying the prostate will not stop with catheter drainage, so prostatectomy must be performed as an emergency measure.

Operative Treatment

Until the commencement of the present century the accepted treatment of prostatic obstruction was a catheter-life. The patient passed the instrument himself, often lubricating it with saliva. The more affluent sufferers carried their catheters in their top hats. Nearly 20 per cent. of all sufferers died within three months. Some of the remainder acquired partial immunity to recurring bouts of infection.

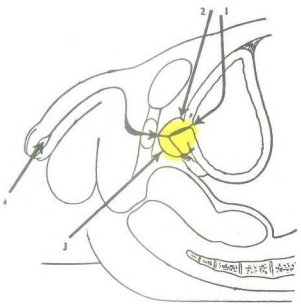


FIG. 1433.—The surgical approaches to the prostate.

Nowadays a permanent catheter, either urethral or suprapubic, is a rarity. Prostatectomy, or more correctly the removal of the adenomatous hyperplasia, by one of the four routes, is practicable in the great majority of cases.

The prostate can be approached (1) through the bladder (transvesical), (2) retropubically, (3) from the perineum, or (4) perurethrally (fig. 1433).

Preliminary Vasectomy.—In all the operations about to be described, bilateral vasectomy (dividing the vasa deferentia) is advisable. It prevents infection from the prostatic bed spreading to the epididymes. This minor procedure can be undertaken as a preliminary step, or at the conclusion of prostatectomy. *Technique.*—Through a small incision over each superficial abdominal ring the vas deferens is isolated from the spermatic cord and a small piece resected, the ends being ligated with catgut.

Transvesical Prostatectomy.—The bladder is opened, the prostate enucleated by putting a finger into the urethra, pushing forwards towards the pubes to separate the lateral lobes, and then working the finger between the adenoma and the false capsule. In **Freyer's operation** (1901) the bladder was left open widely and drained by a suprapubic tube with a 16-mm lumen, in order to allow free drainage of blood and urine. **Harris** (1934) advocated control of the prostatic arteries by lateral stitches inserted with his boomerang needle (fig. 1434 and 1435), and closure of the raw

FIG. 1434.—A boomerang needle.

FIG. 1435.—Harris's ligature carrier.

areas by stitching the mucosa of the posterior lip of the internal meatus to the urethral mucosa, and narrowing the cavity with stitches in front of the urethral catheter which was left in to drain the bladder. The bladder wall was left completely closed and the wound drained. **Wilson Hey** (1944) also advocated a transvesical prostatectomy, stressing the importance of careful asepsis in this as in any other surgery. The

Sir Peter Johnston Freyer, 1852–1921. Surgeon, St. Peter's Hospital for Stone, London.
Samuel Harry Harris, 1881–1937. Urologist, Lewisham Hospital, Sydney, Australia.

catheter for drainage was passed retrogradely to avoid carrying urethral organisms up into the raw prostatic cavity. Mass ligatures of tissue for hæmostasis was replaced by careful diathermy of bleeding points with insulated hæmostats (fig. 1436). Careful closure of the bladder enabled the urethral catheter to be removed on the fourth day.

Retropubic Prostatectomy (Millin, 1945).—Using a low, curved transverse suprapubic incision, which includes the rectus sheath, the recti are retracted to expose the bladder with its typical appearance of pale brown muscle bundles with a loose covering of fatty tissue and veins. With the patient in the Trendelenburg position the surgeon separates the bladder and the prostate from the posterior aspect of the pubis. In the space thus obtained the anterior capsule of the prostate is incised with diathermy below the bladder neck, care being taken to obtain complete control of bleeding from divided prostatic veins.

FIG. 1436.—Insulated hæmostat (Riches' pattern).

FIG. 1438.—Punch prostatectomy. The fenestrum engaging the tissue of the middle lobe.

FIG. 1437.—McCarthy's endoscopic prostatic resector.

The prostatic adenoma is exposed and enucleated with a finger. A wedge is taken out of the posterior lip of the bladder neck to prevent secondary stricture in this region. The exposure of the inside of the prostatic cavity is good, and control of hæmorrhage is achieved with diathermy and stitching before closure of the capsule over a Porges catheter (inserted per urethram) draining the bladder.

Perineal Prostatectomy (Young).—A A-shaped incision in the perineum in front of the anus. The rectum is retracted backwards to expose the posterior aspect of the prostate above the triangular ligament. This is incised and the adenoma enucleated. This operation is not popular in Britain because of the incidence of incontinence.

Perurethral prostate resection, (P.U.R. or T.U.R.) is indicated when obstruction is due to a fibrous prostate, a median bar, or a carcinoma, and occasionally when a middle lobe is associated with slight lateral lobe enlargement. It can be achieved with a McCarthy resectoscope or a cold punch (figs. 1437–1439).

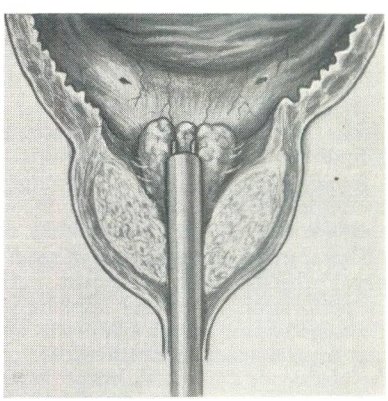


FIG. 1439.—Transurethral resection showing the loop commencing to remove a strip from the middle lobe of the prostate. *(After R. M. Nesbit.)*

Strips of tissue are cut from the lip of the bladder neck down to the verumontanum. Damage to the external sphincter will not occur if this is the lowest point of the resection. With the McCarthy resectoscope cutting is accomplished by a high-frequency diathermy current applied to the loop. In the case of the Thompson punch, a tubular knife moves across a fenestrum at the distal end of the sheath of a direct-vision cysto-urethroscope. Magnification is less than with the resectoscope, but as less necrotic tissue is left behind, secondary hæmorrhage is less frequent.

Terence John Millin, 1903–1980. Surgeon, Westminster Hospital, London.
Porges. Surgical Instrument Manufacturers, Paris, France.
Hugh Hampton Young, 1870–1945. Director, James Buchanan Brady Urological Institute, Baltimore, Maryland, U.S.A.
Joseph Francis McCarthy, 1874–1965. Formerly Professor of Urology, New York Polyclinic Medical School, U.S.A.
Gershom Joseph Thompson, Contemporary. Senior Consultant, Mayo Clinic, Rochester, U.S.A.

After Treatment

(1) 120 ml. of sterile normal saline is left in the bladder for one hour to dilute initial bleeding before continuous drainage is established.

(2) After one hour the catheter is joined via a sterile (non-tapered) connection to a sterile tube and a bottle containing 120 ml. of formalin (fig. 1383).

(3) Bladder wash-outs with full aseptic technique are only given if drainage is unsatisfactory or bleeding excessive.

(4) A high urinary output to ' wash out ' the bladder is essential.

(5) If the urine is infected, a suitable drug or antibiotic should be given pre-operatively to control bacteræmia and to reduce the danger of secondary hæmorrhage.

(6) Early ambulation has diminished the incidence of venous thrombosis and pulmonary embolism.

Complications.—*Hæmorrhage* may continue; transfusion is necessary if the blood pressure falls. *Clot retention* can occur if bleeding is severe and it is not observed that drainage is poor. Suction applied with a Riches's bladder syringe and irrigation with sterile normal saline will clear this. (No fluid must be put into the distended bladder until some has been sucked out. The use of a Bigelow's evacuator in such instances is dangerous and may lead to rupture of the bladder.)

Infection.—The routine employment of sulphonamide or appropriate antibiotic therapy before operation and during the post-operative treatment has considerably reduced complications consequent upon infection of the prostatic bed.

Epididymitis.—Division of the vasa deferentia at prostatectomy almost eliminates post-operative epididymitis, but a localised inflammation of the spermatic cord or a small abscess at the site of the division sometimes occurs.

Acute Pyelonephritis.—Ascending pyelonephritis can often be combated by antibiotics, but bilateral infection occurring in kidneys, already the seat of some degree of hydronephrosis, is always serious.

Renal Failure.—This is extremely rare. If gross renal impairment is present operation should be deferred until continuous bladder drainage has produced maximal renal recovery. Very occasionally renal dialysis before operation is justified.

Extraperitoneal perforation of the bladder is an occasional accident during transurethral prostatectomy. When the patient is under low spinal anæsthesia, immediately the perforation occurs he complains of severe suprapubic pain, often radiating towards the epigastrium. Such symptoms appearing after the patient has regained consciousness from a general anæsthetic are more difficult to interpret. When perforation of the bladder is suspected, immediate suprapubic cystostomy and drainage of the prevesical space should be carried out. If this operation is delayed more than twelve hours, the prognosis becomes grave, except in a few instances when extravasation occurs slowly with the formation of an abscess in one or other inguinal region.

Incontinence of urine occurs in retropubic prostatectomy when the membranous urethra is drawn up and divided. Damage to the external sphincter can occur in transurethral resections.

Later Complications

Osteitis pubis is rare. It is possibly due to pricking of the periosteum during operation, or to infection in the prevesical space. The symptoms do not appear until two or more weeks after the operation. There is pain and tenderness over the pubes, with inability to walk more than a few steps owing to painful spasms of the

Sir Eric William Riches, Contemporary. Consulting Surgeon, Middlesex Hospital, London.
Henry Jacob Bigelow, 1818–1890. Professor of Surgery, Harvard University, Boston, Massachusetts, U.S.A.

adductor muscles. An X-ray shows irregular rarefaction of the ischio-pubic rami (fig. 1440) and widening of the symphysis.

This exceedingly painful state continues for many weeks. Spontaneous cure with recalcification of the rarefied bone occurs after several months. Earlier recovery can be achieved by incising the posterior aspect of the symphyses and curetting out the necrotic cartilage and bone. Penicillin and streptomycin are placed in the area and the wound is closed with a small corrugated drain which is removed after forty-eight hours.

FIG. 1440.—Radiograph showing osteitis pubis following retropubic prostatectomy. (*N. M. Matheson, F.R.C.S., Ashford, Middlesex.*)

Post-operative stricture of the prostatic bed is a troublesome complication. Dilatation must be carried out regularly. More common now are strictures of the anterior urethra following the passage of a wide-bore resectoscope or punch.

CONTRACTURE OF THE BLADDER NECK

Ætiology.—The condition occurs in children of both sexes and in women as well as men. It may be due to a congenital muscular hypertrophy, or fibrosis of the tissues at the bladder neck and is then an aftermath of chronic prostatitis in men, or urethro-trigonitis in women. The former cause may give rise to symptoms at any age, usually in young children or adult life, the latter after the age of forty-five.

Clinical Syndromes:

(*a*) *Due to Muscle Hypertrophy.*—Marion described a series of cases in which muscular hypertrophy of the internal sphincter in a young person had resulted in the development of a vesical diverticulum *or* hydronephrosis (Marion's Disease, or *prostatism sans prostate*) (fig. 1441). Milder degrees of obstruction can occur without the development of these secondary effects (p. 1265). These patients have difficulty in micturition or may present with recurrent urinary infections.

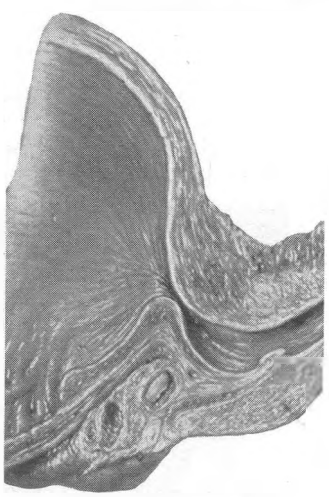


FIG. 1441.—Contracture of the bladder neck causing 'prostatic' obstruction. (*After H. H. Young.*)

A severe degree of obstruction produces symptoms at birth or in early childhood. A distended bladder may be visible or easily felt. Many cases develop urinary infection. Ureteric reflux of urine associated with high intravesical pressure during micturition leads to loin pain and severe pyelonephritis with gross scarring and contraction of the renal cortex.

(*b*) *Due to Fibrosis.*—The symptoms are similar to those of prostatic enlargement, but straining helps to expel the urine, and as a consequence an inguinal hernia may develop. There is no prostatic enlargement. Secondary effects of the obstruction are in all respects similar to those seen in prostatic enlargement. Contracture of the bladder neck is the most common cause of a diverticulum of the bladder.

Treatment:

Dilatation.—Minor degrees of the condition sometimes respond satisfactorily to intermittent dilatation by bougies and treatment of the chronic prostatitis, if it is still active. Otherwise operation is always needed.

Transurethral resection is practised widely for this condition. The immediate results are excellent, but early relapses so often follow, especially in women, that opinion is veering to open operation.

Open Operation.—The principle of open operative treatment, like that of Johanson's operation for urethral stricture (p. 1299), is to establish in the strictured portion of the canal one part of the circumference that is non-fibrotic and pliant.

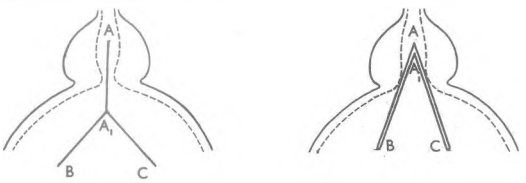


FIG. 1442.—Bonnin's operation for contracture of the bladder neck in the male.

Sphincteroplasty is conducted through a retropubic incision.

Bonnin's operation is suitable for adult male patients. A V-shaped flap in the bladder (muscle and mucosa) is introduced into the anterior aspect of the vesical outlet, and the steps of the operation are shown in fig. 1442. When severe vesical atony is present, which is usual in cases of some standing, it is desirable to resect a large portion of the bladder. This is accomplished by mobilising the antero-superior aspect of the bladder before incising it. To have the viscus well distended is most important.

Congenital Valves of the Prostatic Urethra (p. 1285).

PROSTATIC CALCULI

Prostatic calculi are of two varieties—endogenous, which are common, and exogenous, which are comparatively rare.

An *exogenous* prostatic calculus is a urinary (commonly ureteric) calculus that becomes arrested in the prostatic urethra. This is considered on p. 1303.

Endogenous prostatic calculi are usually composed of calcium phosphate combined with about 20 per cent. of organic material.

Clinical Features.—Quite often prostatic calculi are symptomless, being discovered on radiography of the pelvis, during prostatectomy, or associated with carcinoma of the prostate or chronic prostatitis. The symptoms vary in severity and are at first those of chronic prostatitis, or of prostatic obstruction. On rectal examination it is difficult to differentiate from carcinoma. On X-ray these stones often form a horse-shoe (fig. 1443) or a circle.

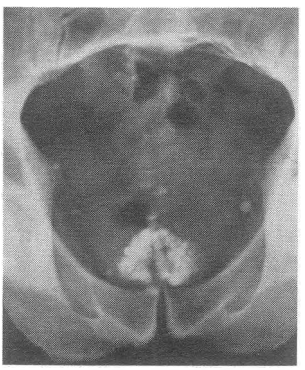


FIG. 1443. — Radiograph showing endogenous prostatic calculi.

Treatment of Prostatic Calculi:

Conservative Measures.—When the calculi are small and the symptoms are mild, the treatment of chronic prostatitis by a course of sulphonamide may keep the patient free from symptoms for months at a time.

Transurethral resection of the fibrous prostate will release small calculi as the strips of prostatic tissue are excised, and many more are removed by the irrigation inseparable from transurethral resection. Others are passed per urethram at a later date.

Retropubic Prostato-lithotomy.—After incising the capsule of the prostate, the

Noel James Bonnin, Contemporary. Surgeon, Queen Elizabeth Hospital, Adelaide, Australia.

incision is deepened until the prostatic urethra is opened. Using a sharp curette, all the calculi and as much as possible of the infected prostatic tissue is removed. The bladder neck is then exposed, and a generous wedge of the posterior lip is resected with curved scissors. The operation is concluded in a manner similar to that of retropubic prostatectomy. Total prostatectomy may be necessary in cases where there are frequent acute exacerbations of a chronic prostatitis.

Corpora amylaceæ

Corpora amylaceæ are found in the prostates of elderly men and apes, but not in the prostates of animals lower in the phylogenetic scale than anthropoids. Corpora amylaceæ occur also in the homologue of the prostate—the tubules of the bladder neck of women (Skene's tubules). These bodies consist of amorphous debris and desquamated epithelium. Often an intact epithelial wall of a prostatic duct forms their outer coat. They are always deeply pigmented, and are variously described as like poppy seeds, black pepper, or coal dust. Neither the composition nor the origin of the pigment is known. Probably corpora amylaceæ are the forerunners of endogenous prostatic calculi.

CARCINOMA OF THE PROSTATE

Carcinoma of the prostate is the commonest malignant condition in men over the age of sixty-five years. About 20 per cent. of cases of prostatic obstruction prove to be due to carcinoma, to which must be added a substantial number in whom the first and main symptoms are due to metastases. The incidence of carcinoma of the prostate has been increasing, due to an ageing population as well as to improved methods of diagnosis. Carcinoma of the prostate usually originates in the external group of glands (fig. 1426), so 'prostatectomy' for benign enlargement of the gland confers little protection from the subsequent development of carcinoma.

Latent Carcinoma of the Prostate.—Serial sections of prostates obtained at routine necropsy has revealed carcinoma in that organ in no less than 15 per cent. of men over fifty years of age. True, many of these neoplasms are tiny and (if life had continued) might have remained latent for years. In men over ninety years of age, over three-quarters of the prostates are involved (Franks). It seems, therefore, that a seedling carcinoma is often present in the prostate of an elderly man, awaiting favourable conditions to become active.

Histological Appearance.—Usually the carcinoma is spheroidal celled, with a varying degree of tubule formation, in which case the tumour is a slowly growing one. The anaplastic type is more malignant and manifests itself more aggressively than an adenocarcinoma.

Local Spread.—A growth *commencing in the posterior zone of the gland* is prevented (at least temporarily) from extending backwards by the strong fascia of Denonvilliers. Consequently it tends to grow upwards to involve the seminal vesicles. Further upward extension obstructs the lower end of one or both ureters, the latter terminating in anuria. Carcinoma *commencing in a lateral lobe* involves the prostatic urethra early: in advanced cases the base of the bladder is invaded. The rectum is involved occasionally by infiltration, and stricture of the rectal wall occurs. The mucosa does not ulcerate unless traumatised, e.g. by transrectal biopsy.

Spread by the blood-stream occurs particularly to bones; indeed, the prostate is the most common site of origin for skeletal metastases, being followed in turn by the breasts, the kidneys, the bronchial tree, and the thyroid gland.

Leonard Maurice Franks, Contemporary. Research Pathologist, Imperial Cancer Research Fund, Royal College of Surgeons of England, London.

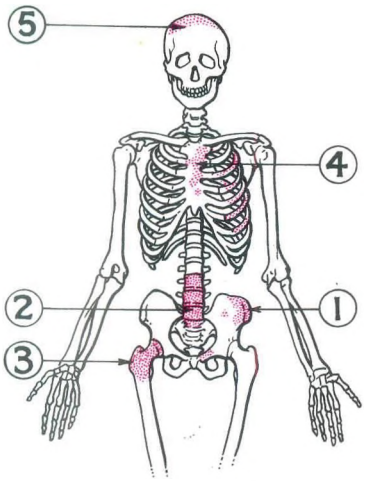


FIG. 1444.—Sites of location, in order of frequency, of metastases in bones from carcinoma of the prostate. (*After Swift Joly.*)

The bones involved most frequently by metastases of carcinoma of the prostate are shown in fig. 1444. The pelvic bones and the lower lumbar vertebræ are particularly common sites. The frequent proximity of skeletal metastases to the primary growth has been attributed to reversed flow from the vesical venous plexus to the emissary veins of the pelvic bones during coughing, sneezing, etc.

Lymphatic Spread.—(*a*) Via lymphatic vessels passing along the sides of the rectum to the lymph nodes along the internal iliac vein and in the hollow of the sacrum. (*b*) Via lymphatics which pass over the seminal vesicles and follow the vas deferens for a short distance to drain into the external iliac lymph nodes. From both these situations the retroperitoneal lymph nodes, later the mediastinal nodes, and occasionally the supraclavicular lymph nodes, become implicated.

Clinical Features.—Carcinoma of the prostate occurs in the older man. Incontinence, a short history of up to six months, and pain on micturition are suggestive features of carcinoma in a patient with a history of prostatism.

Type 1.—Discovered only on histological examination of tissue removed at prostatectomy.

Type 2.—Rectal findings of a hard nodule or extension outside the capsule; investigated by perineal biopsy (see below) (fig. 1446).

Type 3.—The primary may be tiny and occult, the patient presenting with ' rheumatism ' or ' arthritis ', with blood acid phosphatase levels often very high. Urinary symptoms are absent or slight.

Type 4.—Pain in the back, or sciatica, is the main symptom. Bilateral sciatica in an elderly man is most often due to metastases in the spine from carcinoma of the prostrate. Œdema of one or both legs, paraplegia, or a spontaneous fracture are occasionally due to metastases from a carcinoma of the prostate. Anæmia may be the presenting symptom.

On account of destruction of bone marrow, bone metastases from carcinoma of the prostate can give rise to a hæmorrhagic diathesis, and the patient suffers hæmorrhage, often severe, not necessarily from the urinary tract.

Rectal Examination.—Bimanual examination under anæsthesia, together with cystoscopy and needle biopsy are essential in order to assess the local stage of the growth. Irregular induration, characteristically stony hard in part or in the whole of the gland—with obliteration of the median sulcus suggests carcinoma. Extension beyond the capsule up into the bladder base and vesicles is diagnostic, as is deformity and projection outwards of the capsule.

T.N.M. Classification

Tumour:

T1 Local nodule | T2 Diffuse or deforming capsule | T3 Outside capsule or extension into vesicle | T4 Fixed to other tissue

Nodes: No = No evidence of involvement of regional nodes.
N1 = Involvement of one regional node.
N2 = Involvement of several regional nodes.
N3 = Fixed mass of regional nodes.
N4 = Involvement of common iliac or para-aortic nodes.

Metastases: Mo = No evidence of distant metastases.
M1 = Distant metastases.

Radiological examination to exclude or confirm the presence of prostatic calculi or pelvic and lumbar skeletal metastases is always necessary. Whereas osseous metastases from carcinomas of other organs are usually osteolytic, producing a rarefied 'moth-eaten' appearance, those in the lower lumbar vertebræ and pelvic bones from a carcinoma of the prostate are typically osteoblastic, resulting in increased density of the bone (fig. 1445), and must be distinguished from Paget's disease of bone (p. 300).

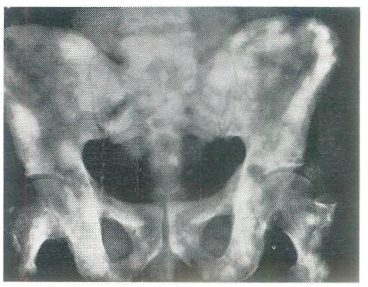


FIG. 1445.—Osseous metastases of the pelvic bones in carcinoma of the prostate. (*L. N. Pyrah, F.R.C.S., Leeds.*)

Other methods of detecting metastases:—It is an interesting fact that many apparently early cases can be shown to have distant metastases by employing:

1. *Bone Scanning* with radioactive bromine or selenium. The deposits are shown as 'hot spots'.

2. *Scalene node biopsy.*—A lymph node is removed from in front of the left scalenus anterior muscle in the neck.

3. *Bone marrow aspiration*—(sternum or ilium) reveals metastatic carcinoma cells in a surprisingly high percentage of those cases where radiological examination revealed no evidence of secondary deposits. This investigation should be carried out in every case before radical prostatectomy is even considered.

4. *Lymphangiography.*—By employing the technique described on page 186. X-rays will show whether there are deposits in the pelvic lymph nodes and any lymphatic obstruction.

Serum Acid Phosphatase. (See page 1264).—In about 40% of patients with carcinoma of the prostate the acid phosphatase is raised above normal (1 to 3 King-Armstrong units). A reading between 3 and 5 units is suspicious of carcinoma of the prostate, and above 5 units practically diagnostic. Very high levels up to 100 or more K.A. units can be found at times in patients showing extensive bone marrow involvement.

Confirmation of the Diagnosis.—Should be obtained by examination of histological material from the prostate. A true biopsy of prostatic tissue can be obtained with a Trucut or a Turkel needle passed through the perineum into any suspicious area (fig. 1446). Open perineal biopsy is still favoured in America.

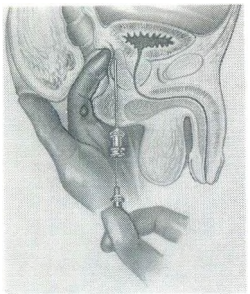


FIG. 1446.—Obtaining a specimen of prostatic tissue by means of Turkel's needle.

Cysto-urethroscopy.—When there is a history of hæmaturia, this examination is essential. There is often a grating sensation as the prostatic urethra is traversed. With the instrument in place, deep induration in the prostate is more readily appreciated by a finger in the rectum, and fixation of the gland becomes more evident. Puckering of the apex of the trigone, submucous nodules in the base of the bladder, or ulceration of an intravesical projection are late manifestations. In earlier cases there are no pathognomonic visible signs.

Transuretheral Biopsy.—Transurethral resection of the prostate has the advantage of removing the obstruction and providing large pieces of tissue, but it may not reach the posterior zone of the prostate, which is commonly the seat of early carcinoma, and it must be remembered that benign enlargement frequently co-exists.

Treatment of Carcinoma of the Prostate

In spite of every endeavour, the diagnosis of carcinoma of the prostate usually is made late in the course of the disease, as the following statistics of the condition at the time of diagnosis testify:

Neoplasm confined within the anatomical capsule of the prostate	5 per cent.
Local spread	55 per cent.
Metastases present	40 per cent.

In those patients where carcinoma is found on routine histological examination of tissue at prostatectomy then, if lymphography and a bone scan are negative and the lesion was localised and well differentiated the patient is not given any specific treatment, but is kept under regular observation. If, however, the carcinoma is diffusely infiltrating the removed tissue, or is anaplastic, then local radiotherapy should be given. Patients with positive nodes on bone scan should be treated by orchidectomy or œstrogens as well as by local radiotherapy. Radical surgery plays a very small part in the management; it may be considered for a T.1. lesion with no evidence of spread.

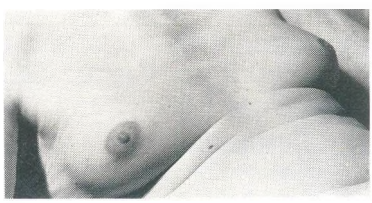


FIG. 1447.—Gynæcomazia following stilbœstrol therapy in a man 80 years of age with carcinoma of the prostate. It can to a large extent be prevented by preliminary radiation (6–800 rads) to each breast.

However, a great deal can be done to alleviate symptoms. Huggins first showed the beneficial effects of castration or stilbœstrol. **Stilbœstrol** causes improvement in 85% or more cases. It is of no value in transitional cell carcinomas arising from prostatic epithelium. Histological changes in many of the malignant cells may be apparent within forty-eight hours of commencing treatment. The cells become distended with glycogen, the nucleus becomes pyknotic, and moves to the edge of the cell, being extruded when the cell breaks up.

Symptomatic changes occur in many cases within two weeks—there is less dysuria, a better stream, less frequency, and aches and pains may disappear. The prostate becomes smaller and softer, and these changes may be detected

Eric Francis Turkel, Contemporary. Urological Surgeon, Pomona Valley Community Hospital, Pomona, California, U.S.A.
Charles Brenton Huggins, Contemporary. Professor of Surgery, Chicago, Illinois, U.S.A.

rectally in three to four weeks. Bone deposits may remain static and occasionally disappear.

The dosage of Stilbœstrol.—Probably only 3 to 5 mg a day are needed to reduce the serum testosterone from a normal of 400–1,200 ng/100 ml (14–42 nmol/l) to a satisfactory level of maintenance of 50 ng/100 ml (1·7 nmol/l). Larger doses produce a more rapid response.

Side-effects of Stilbœstrol.—There is increasing evidence that œstrogens produce an increased incidence of vascular accidents, particularly venous thrombosis and embolism. Caution should be employed in patients with a high serum cholesterol or previous history of coronary or other vascular disease. Gynæcomazia can be greatly reduced by radiotherapy, (800 rads) to each breast before starting œstrogentherapy, but pigmentation of the nipples and tenderness in the breast tissue is less affected. Nausea and gastric upsets are occasionally present, and these are unrelated to dosage. Œdema of the ankles from salt retention may occur. Excessive nasal secretion is complained of by some patients and disturbance of vision by others. Adrenal stimulation elevating the level of androgen secretion may occur.

In all such cases subcapsular orchiectomy (fig. 1448), and cessation of œstrogen therapy can be tried, especially valuable in most of the side-effects previously described. Alternatively **T.A.C.E.** (tripara-anisil-chlorethylene), which is said to stimulate the breast and adrenal less, may be tried. Dose—12 to 24 mg per day.

(A) (B)

FIG. 1448.—(A) Metastases of prostatic carcinoma in the scalp. (B) Thirteen months after orchiectomy. (*Professor C. Huggins, Chicago.*)

Honvan (phosphorylated diethylstilbœstrol) is given i.v. or orally. It is claimed that the acid phosphatase of the prostatic carcinoma cells splits the phosphate element off the preparation, thus releasing the œstrogenic hormone locally and favouring its direct action on the carcinoma cells, and the carcinoma cells only. At first 100 mg are injected (it must be injected very slowly) three times a day for ten days. The dose is then increased to 200 mg, and eventually (enfeebled patients excepted) to as much as 500 to 1,000 mg, until the twentieth day. The dosage is then gradually decreased, until one 200-mg injection is given weekly, and finally monthly. The intravenous injections, like all intravenous injections, require skilled administration. Orally, the dose of Honvan usually employed is 100 mg daily.

Radiotherapy.—Supervoltage treatment to the prostate (with a linear accelerator or cobalt source) is showing improved results, and, in the face of mounting evidence of vascular complications and malaise associated with stilbœstrol, is increasing in favour. It is most useful for localised disease and, perhaps, in anaplastic tumours. If dissemination is present, subcapsular orchidectomy should be performed.

Treatment of Retention in Carcinoma of the Prostate

Acute retention due to a carcinoma of the prostate should be treated with an indwelling Gibbon catheter (p. 1231) while definitive treatment is commenced. Usually micturition will then be resumed. If after three weeks the patient cannot pass urine normally, then the obstruction should be relieved. This is usually done by a per-urethral resection, but if the gland is also the seat of adenomas, a retropubic or transvesical removal of most of the tissue may be considered. Post-operative radiotherapy should be given after open surgery in definite cases of diffuse carcinomatous change in the gland.

Chronic Retention.—Such cases should not be catheterised, especially if the urine is sterile when first seen. Infection once established is impossible to eradicate in the presence of a growth. The residual urine may decrease gradually with treatment. Perurethral resection may be necessary if this does not occur.

Other Measures and Prognosis

Treatment of Pain and Enlarged Lymph Nodes.—Severe pain from bone secondaries, but more usually from perineural lymphatic infiltration, can be difficult to control, as is œdema of a leg from enlarged lymph nodes. Deep X-ray therapy combined with intra-arterial nitrogen mustard given into the pelvic vessels via a retrograde catheter up the femoral artery (two doses of 10 to 15 mg at weekly intervals) is most useful.

Adrenalectomy is of little value. Pituitary ablation has occasionally been found helpful.

Radioactive Phosphorus, given after the stimulation of bone secondaries by parathormone or testosterone, may be helpful in controlling pain from multiple bone secondaries.

Chemotherapy.—Various regimes are on trial, but methotrexate seems to be the most helpful.

Prognosis.—Patients who fail to respond to stilbœstrol usually die within one year. On stilbœstrol five-year survival amounts to 25 to 30 per cent. Occasional survivals up to ten years are recorded, but ultimately nearly all patients die of the disease.

PROSTATITIS

In both acute and chronic prostatitis the seminal vesicles are usually infected, and when, as is often the case, the prostatic urethra is involved also, there is present a triad of pathological conditions, to wit: posterior urethritis, prostatitis, and seminal vesiculitis. Symptoms due to infection of any one of these structures may predominate.

Acute Prostatitis

Ætiology.—Acute prostatitis is common.—The usual organism responsible is *Esch. coli*, but *Staphylococcus aureus* and *albus*, *Streptococcus fæcalis*, and the *gonococcus* may be responsible. As a rule the infection is hæmatogenous from a distant focus, notably furunculosis, infected tonsils,

carious teeth, or diverticulitis. In a minority of cases the infection ascends from the urethra or descends from the bladder or kidneys.

Clinical Features.—Infection in most cases seems to be blood-borne, as general manifestations overshadow the local: the patient feels ill, shivers, may have a rigor, has 'aches' all over, especially the back, and may easily be diagnosed as having influenza. The temperature may be up to 39° C. Pain on micturition is usual, but not invariable. The urine always contains threads in the first glass, and it is this initial specimen of urine which should be cultured. Perineal heaviness, rectal irritation and pain on defæcation can occur, and sitting may be uncomfortable. A urethral discharge is rare. Frequency occurs when the infection spreads up to the bladder. Rectal examination reveals a tender prostate, one lobe may be swollen more than the other, and the seminal vesicles may be involved. A frankly fluctuant abscess is uncommon.

Treatment.—Must be rigorous and prolonged or the infection will not be eradicated and recurrent attacks will ensue. Unless treated early, spread of the infection to the epididymes and testes is common. While awaiting the results of culture of the initial and a midstream specimen of urine, the patient is put to bed, given copious fluids, and started on a broad spectrum drug such as tetracycline. As soon as the culture result is available the most suitable drug can be chosen and continued for ten days. It should be followed by a month's course of a sulphonamide such as sulphamethizole (Urolucosil) 100 mg. 4 times daily to prevent recurrence. Bed-rest for ten to fourteen days, avoidance of alcohol and intercourse for six weeks is essential.

Prostatic Abscess.—In addition to the foregoing symptoms and signs, the advent of a prostatic abscess is heralded by the temperature rising steeply, rigors being not unusual. However, antibiotics disguise these leading features. Severe, unremitting perineal and rectal pain with occasional tenesmus often cause the condition to be confused with an ano-rectal abscess. Nevertheless, if a rectal examination is performed, the prostate will be felt to be enlarged, hot, extremely tender, and perhaps softened in one place. Retention of urine is likely to occur.

Treatment.—The abscess should be drained without delay. It is true that if a catheter is passed to relieve acute retention of urine, sometimes the abscess is ruptured into the prostatic urethra. However, in most cases such drainage is insufficient, and is followed by intractable chronic prostatitis or a residual abscess.

(*a*) The abscess can be drained by perurethral resection—unroofing the whole cavity.

(*b*) The perineal route is preferable when there is marked peri-prostatic spread.

Chronic Prostatitis

Ætiology.—This is a sequel of inadequately treated acute prostatitis. While pus is present in the prostatic secretion, often the responsible organism is difficult to find. Smears show bacteria in about 40 per cent. and cultures are positive in 70 per cent. of cases. The predominant organisms are E. coli, staphylococci, streptococci, and diphtheroids, in that order. Recently trichomonas has been found to be a cause of chronic prostatitis and may be common to both husband and wife.

Pathology.—The lumina of the ducts become blocked with epithelial debris and pus. This causes a soft enlargement of the organ. Later fibrosis occurs, and the prostate becomes smaller and harder.

Clinical features are extremely varied.

1. *Causing Chronic Posterior Urethritis.*—Prostatitis should not be diagnosed unless, after irrigating the urethra and massaging the prostate, the resultant specimen shows fifteen or more pus cells per high power field.

2. *Causing Epididymitis.*—Acute or subacute non-tuberculous epididymitis rarely occurs unless prostatitis is present.

3. *Pain:* (*a*) *Local* pain is a dull ache in the perineum or rectum. It is increased by sitting on a hard chair. (*b*) *Referred* Pain.—Particularly common is low back pain, sometimes extending down the legs (fig. 1449); such pain is usually attributed to lumbago, and many sufferers from prostatitis receive orthopædic treatment and physiotherapy without benefit.

4. *'Silent' Prostatitis.*—Arthritis, myositis, neuritis, and sometimes iritis and conjunctivitis are on occasions explained only when pus has been obtained from the prostate (see diagnosis).

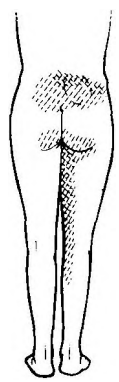


FIG. 1449.—Sites of pain in chronic prostatitis.

5. *Recurring attacks of mild pyrexia,* lasting about three days and accompanied by malaise.

6. *Sexual Dysfunction.*—Premature ejaculations, prostatorrhœa, and impotence are sometimes due to prostatitis.

Many patients with sexual dysfunction due to chronic prostatitis become hypochondriacal.

Diagnosis: (*a*) *A three-glass urine test* is valuable. If the first glass shows urine containing prostatic threads, prostatitis is present.

(*b*) *Rectal examination* of the prostate may or may not confirm the diagnosis. If the organ is soft and boggy, it is obviously abnormal, as also it is if the prostate is smaller and harder than it should be. In mild chronic cases no change can be detected, and reliance must be placed on pathological examinations of the prostatic fluid.

(*c*) *Examination of the prostatic fluid* obtained by prostatic massage. Normal prostatic fluid is slightly opalescent and viscid. A stained specimen shows many pus cells and sometimes bacteria. Non-stained films, in addition to revealing normal epithelial cells, may show trichomonads, if present.

(*d*) *Urethroscopy* reveals inflammation of the prostatic urethra, and pus may be seen exuding from the prostatic ducts. The verumontanum is likely to be enlarged and œdematous.

Treatment.—*Antibiotic therapy* must be administered in accordance with bacteriological sensitivity tests. The blunderbuss administration of antibiotics in this condition is to be deprecated. Probably 0·5 g t.d.s. of a suitable sulphonamide, e.g. Sulphatriad, for six weeks will effect as much as an expensive course of antibiotics. Where trichomonas is the responsible agent, a rapid response is obtained from administration of Flagyl (metronidazole) 200 mg by mouth three times daily, after meals, for seven days to both partners.

TUBERCULOSIS OF THE PROSTATE AND SEMINAL VESICLES

Tuberculosis of the prostate and seminal vesicles is associated with renal tuberculosis in at least 60 per cent. of cases. In 30 per cent. of cases there is a history of pulmonary tuberculosis within five years of the onset of genital tuberculosis.

Tuberculosis of one or both seminal vesicles is more common than tuberculosis of the prostate, and is often discovered when examining a patient with chronic tuberculous epididymitis, there being no symptoms referable to the internal genitalia. On rectal examination the affected vesicle is found to be nodular and tender. In process of time tuberculous seminal vesiculitis may lead to congestion and œdema of the base of the bladder, and later to basal cystitis.

When the Prostate is Involved.—Rectal examination reveals characteristically one or more well-defined nodules, most often near the upper or lower border of one or both lateral lobes. Less frequently a larger solitary mass is felt occupying a more central position. Nodules in the prostate lack the stony hardness of carcinoma.

A patient with tuberculous prostatitis usually presents with one or more of the following symptoms:

A urethral discharge is occasionally the first symptom. Then the diagnosis has to be made from gonorrhœa and abacterial urethritis entirely on bacteriological findings, for the prostate at this early stage is likely to feel normal.

Painful, sometimes blood-stained, ejaculations occur in 20 per cent. of cases.

A mild ache in the perineum is not uncommon.

Infertility.—In patients with tuberculous prostatitis and/or bilateral seminal vesiculitis, fertility is very much reduced; it is safe to say that 80 per cent. of them are sterile. In this connection, owing to the considerable interest in male fertility at the present time, a number of very early cases of tuberculous prostato-vesiculitis are being discovered by culture of the semen.

Urinary Symptoms.—In later cases, when the posterior urethra becomes involved from extension of tubercles from the prostate or by the discharge of a prostatic abscess, there is painful, frequent micturition and sometimes terminal hæmaturia.

Abscess Formation.—If a cold abscess forms in the prostate, it is felt as a slightly tender, soft swelling. Like other prostatic abscesses, it usually ruptures into the urethra, more rarely through the perineum or into the rectum. Occasionally an abscess of the prostate or seminal vesicle bursts into the bladder, and at cystoscopy a ragged, deep ulcer is seen alongside the trigone.

Special Forms of Investigation:

Even in cases without urinary symptoms a complete urological examination is necessary.

Radiography sometimes displays areas of calcification in the prostate and/or the seminal vesicles. Large scattered areas of calcification in the prostate suggest tuberculosis rather than endogenous prostatic calculi.

Bacteriological examination of the seminal fluid yields positive cultures for tubercle bacilli in most cases of tuberculous prostatitis.

Posterior urethrography often reveals one or more dilated prostatic ducts. Typically they are multiple and gaping. Dilated prostatic ducts are not specific for tuberculosis (they can occur in other forms of chronic prostatitis), but dilated ducts plus the finding of tubercle bacilli in the ejaculate establishes an absolute diagnosis.

Transmission of genital tuberculosis to the female partner is exceptional, but it does occasionally occur in the form of tuberculous cervicitis.

Treatment.—The general treatment given is as for renal tuberculosis (p. 1213). On no account must a tuberculous prostate or seminal vesicle be subjected to massage. Urethral instrumentation should be avoided, or reduced to a minimum only to confirm the diagnosis.

If a prostatic abscess forms, it is better to evacuate it by the perineal route than to permit it to rupture spontaneously.

Considerable involvement of the prostate, which goes on to suppuration, is a comparatively unfavourable form of genito-urinary tuberculosis. On the other hand, tuberculous seminal vesiculitis and non-suppurative tuberculosis of the prostate usually heal after an associated lesion of the kidney or epididymis has been eradicated, but stricture of the prostatic urethra may occur.

AFFECTIONS OF THE SEMINAL VESICLES

Acute seminal vesiculitis always occurs in association with prostatitis. Prior to the antibiotic treatment of gonorrhœa, gonococcal vesiculitis was common. When a seminal vesicle is distended it can be palpated per rectum as an acutely tender swelling above and lateral to the prostate. The treatment is discussed on p. 1295.

Abscess of a Seminal Vesicle.—In addition to the usual signs of acute prostatitis, pain is frequently referred to the suprapubic region. If on rectal examination the seminal vesicle is found to be greatly enlarged and tender, the abscess should be drained through an incision in the perineum, viz.⟶ The incision is deepened until the swelling is palpated, and a hæmostat is then thrust into the abscess cavity.

Chronic seminal vesiculitis is less frequently recognised than it should be, because the associated chronic prostatitis overshadows the symptoms of pain on coitus, hæmospermia, aching in the sacral region, recurrent epididymitis, and, at times, associated disturbances of micturition and defæcation. The treatment is the same as for chronic prostatitis, except that in rebellious cases vasotomy and irrigation of the vesicle with normal saline is often rapidly curative.

Tuberculous Seminal Vesiculitis.—The clinical features and treatment are discussed on p. 1283 and above.

Diverticulum of the seminal vesicle occurs occasionally. In such cases the kidney of that side is often absent, and the diverticulum represents an abortive ureteric bud. It is a cause of persistent infection of the urethra.

A cyst of the seminal vesicle is uncommon. It should be removed by dissection through an incision similar to that for perineal prostatectomy.

FIG. 1450.—Palpating an enlarged gland of Cowper.

AFFECTIONS OF COWPER'S GLANDS

Cowperitis.—The diagnosis of Cowperitis is often wanting for lack of a simple examination. On passing the forefinger into the rectum and placing the thumb first on one side and then on the other of the median raphé of the perineum, Cowper's glands can be palpated (fig. 1450). In acute cases the least pressure causes excruciating pain. In this way the condition is differentiated from an ischio-rectal or periurethral abscess. **Treatment.**—In acute cases antibiotic therapy, and rest in bed, often brings about resolution. Should suppuration occur, incision and drainage is necessary.

Fistula formation is the result of an abscess of the gland terminating in spontaneous rupture; it is necessary to excise the fistula and the remnants of the gland.

A cyst of Cowper's gland either bulges into the urethra and causes disturbances of micturition, or gives rise to a unilateral swelling in the anterior part of the perineum. The cyst should be excised.

William Cowper, 1666–1709. Surgeon and Anatomist, London.

CHAPTER 59

THE URETHRA AND PENIS

THE MALE URETHRA

Embryology.—From the internal urinary meatus to the sinus pocularis (uterus masculinus) the urethra is developed from the urogenital sinus. This portion corresponds to the entire female urethra. Beyond the sinus pocularis the male urethra, as far as the glans, is formed by fusion of the edges of the medial labial folds, which also form the corpus spongiosum. The part traversing the glans is formed by canalisation of a down growth of a solid pencil of ectoderm.

CONGENITAL ABNORMALITIES OF THE URETHRA

Meatal Stenosis.—The external urinary meatus, normally the narrowest part of the male urethra, is occasionally the seat of congenital stenosis which is associated with phimosis (p. 1307). In the circumcised, meatal stenosis may also be acquired or increased by fibrosis following meatal ulceration. All degrees of narrowing are encountered. When the opening is reduced to a pin-hole, back-pressure affects the whole urinary system. Pin-hole meatus is occasionally a cause of enuresis, and at any age it may result in chronic retention of urine (fig 1451).

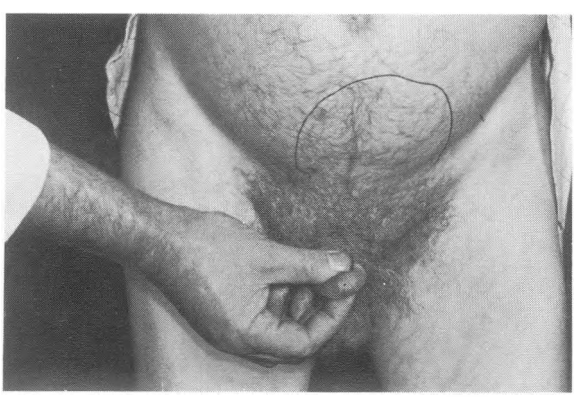


FIG. 1451.—Pin-hole meatus causing chronic retention of urine (distended bladder outlined) in a man of fifty-one. Cured by meatotomy.

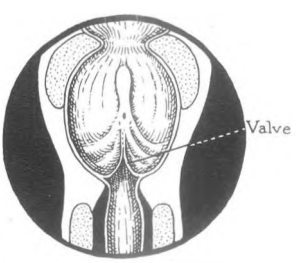


FIG. 1452.—Congenital valves of the posterior urethra. (*After D. Innes Williams.*)

Treatment.—Meatal stenosis, sufficient to (*a*) give rise to symptoms, (*b*) prevent free drainage of the discharge in cases of urethritis, or (*c*) obstruct the passage of full-sized urethral instruments or a cystoscope, should be treated by meatotomy. Lesser degrees of narrowing respond to dilatation with Hegar's dilators.

Meatotomy.—The narrowed section must be split back into normal urethra, and the mucosa stitched to the skin edge. Alternatively, a skin flap can be turned in from the underside of the skin behind the glans.

Congenital stricture of the urethra is very rare; some cases are due to reduplication of the urethra. Usually the symptoms are delayed until adolescence. Treatment by dilatation is effective.

Congenital valves of the posterior urethra are often symmetrical and occur usually just below (fig. 1452), but occasionally above, the verumontanum. They allow the ingress of a catheter but obstruct the outflow of urine. In a few instances

Alfred Hegar, 1830–1914. Professor of Obstetrics and Gynæcology, Freiburg, Germany.

the valves are incomplete and the patient may reach adolescent or adult life without symptoms; however, in such cases the prostatic urethra is greatly dilated and sacculation or diverticula of the bladder are present.

The valves are difficult to see at cysto-urethroscopy, because the irrigating fluid sweeps them into the open position. The most reliable method of confirming the diagnosis is voiding cysto-urethrography, by which is meant that radiographs are taken during attempts to micturate after the bladder has been filled with contrast medium. Alternatively, pressure above the pubes while the radiograph is taken achieves the same objective. In this way the dilated urethra above the valves is outlined, as well as the bladder.

Treatment.—Suprapubic drainage of the bladder is a life-saving measure; several weeks should intervene before attempting to remove the valves. At the second operation the retropubic space is displayed. A vertical incision is made into the bladder just above the prostate and continued downwards into the dilated prostatic urethra. Bleeding-points are coagulated with a diathermy needle and the valves are picked up with toothed dissecting forceps and excised with scissors. The incision into the bladder and urethra is closed with fine catgut sutures, and the bladder is drained by a suprapubic catheter for ten to fourteen days. Infection is controlled by antibiotic therapy. In older children and adults the valves can be excised transurethrally with a resectoscope.

Because of their importance in the differential diagnosis of congenital valves of the urethra, the following causes of chronic retention in childhood are included here:

Obstruction to the neck of the bladder (*syn.* **Marion's disease**) is analogous to hypertrophic pyloric stenosis of infants, and gives rise to symptoms identical with those of congenital valves. The diagnosis can often be appreciated cystoscopically as a hypertrophied interureteric bar. Voiding cysto-urethrography demonstrates that the posterior urethra does not fill, and clarifies the differential diagnosis between the two conditions. Treatment consists in opening the bladder and performing a Y—V plasty (p. 1274).

Neurogenic bladder must be excluded in both the above lesions. Spina bifida, and the accompanying lesions of the central nervous system, is the usual cause of retention of urine of neurological origin in childhood. As with obstruction due to congenital valves, cysto-urethrography shows the dilated prostatic urethra. In neurogenic obstruction, the case with which the bladder can be emptied by manual pressure above the pubis serves as a ready means of distinguishing chronic retention of urine due to this cause from other varieties.

HYPOSPADIAS

Hypospadias is the commonest congenital malformation of the urethra, and it occurs once in every 350 males. The external meatus is situated at some point on the *under*-surface of the penis or in the perineum.

There are five degrees of the deformity, classified as follows:

1. **Glandular Hypospadias.**—There is an ectopic opening on the under-surface of the glans, separated from a blind depression at the normal site of the external urinary meatus. Sometimes a channel connects the ectopic to the normal meatus.

2. **Coronal Hypospadias.**—The meatus is situated at the junction of the under-surface of the glans with the body of the penis.

3. **Penile Hypospadias.**—the meatus opens at some part of the under-surface of the penis (fig. 1453).

4. **Peno-scrotal Hypospadias.**—The urethra opens at the junction of the penis with the scrotum.

5. **Perineal Hypospadias.**—The scrotum is split and the urethra opens between its two halves. This variety is sometimes associated with bilateral maldescended testes, in which event the sex of the child may be difficult to determine.

Jean-Baptiste Camille-Georges Marion, 1869–1960. Emeritus Professor of Urology, Faculty of Medicine, Paris.

Glandular hypospadias is the most frequent variety, and is due to a failure of canalisation of the glans (see embryology). The other varieties are looked upon as an absence of the urethra and corpus spongiosum distal to the ectopic orifice, the absent structures being represented by a fibrous cord. In all except the first variety the penis is curved in a downward direction (chordee). The farther away the opening is from the normal position, the more pronounced is the bowing. In all cases the inferior aspect of the prepuce is poorly developed (' hooded prepuce ').

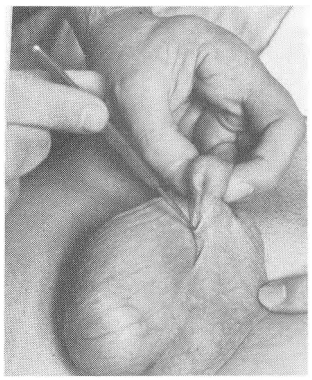


FIG. 1453.—Penile hypospadias. The patient passes urine through the orifice demonstrated by the probe.

Treatment.—In glandular hypospadias no treatment is required, unless the opening is too small, in which case meatotomy is performed. In other varieties a plastic operation, of which there is a great variety, should be carried out. On this account circumcision during infancy should not be performed in these cases as the redundant skin will prove useful later.

Stage 1.—*Straightening the penis* is undertaken preferably between one and a half and two years of age. A transverse incision is made on the ventral surface 1·25 cm distal to the misplaced external urinary meatus, and the skin on either side of the urethra is undermined, exposing the fibrotic corpus spongiosum, which is detached from before backwards by severing its fibrous attachment to the corpora cavernosa. This accomplished, the urethral orifice recedes towards the perineum; thus a coronal hypospadias becomes a penile, peno-scrotal, or perineal, and the penis is no longer tethered. The original transverse incision is further lengthened by carrying it into the under-surface of the prepuce on either side. The incision is then closed vertically, tension being relieved by making a longitudinal incision in the middle line through the skin of the dorsum of the penis and prepuce.

Stage 2.—*Denis Browne's method of constructing a urethra* is undertaken, preferably between five and seven years of age. Diversion of all urine from the seat of operation can be accomplished only by *perineal urethrostomy*. A small catheter is passed into the bladder on an introducer. The introducer is withdrawn slightly, and rotated so that its beak can be felt in the perineum. Strictly in the middle line a small incision is made, and the catheter is seen within the urethra. The wall of the catheter is grasped in a hæmostat while the assistant withdraws the introducer. The butt end of the catheter is then drawn along the urethra and made to emerge in the perineum. Two cotton sutures are tied around the catheter and utilised to anchor it to the edges of the perineal wound.

Fashioning a New Urethra.—An incision is made as outlined in fig. 1454 (A). The flaps are undermined, special attention being paid to freeing the skin in the direction of the perineum, so that when the flaps are united they lie well forward of the old urethral opening. When the undermining involves the scrotum, stab wounds should be made on either side to allow the ready escape of blood and serum. Next, with sharp scissors, a triangular area of glans is bared of skin on either side of the proposed new meatus (fig. 1454 (B)). An incision is made through the skin along the whole length of the dorsum of the penis. This is to relieve tension, and the wound is allowed to epithelialise. The flaps are sutured, but not too tightly. Browne employed glass beads and small sections of soft aluminium tubing, which are crushed, and hold the sutures in place (fig. 1454 (C)). No dressing is employed—only a spray of Nobecutane. Tetracycline is given for two weeks. The sutures are removed in a week, and the catheter is withdrawn on the tenth day. The perineal wound usually closes three to five days later.

Sir Denis John Wolko Browne, 1892–1967. Surgeon, Hospital for Sick Children, Great Ormond Street, London.

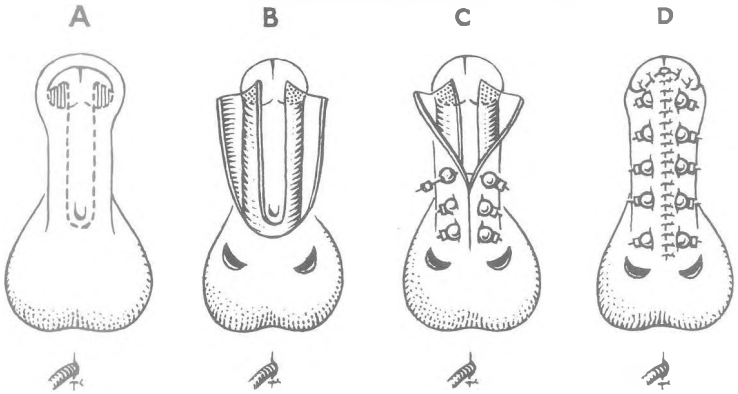


FIG. 1454.—Denis Browne's operation for hypospadias. The buried strip of skin forms the new urethra.

EPISPADIAS

Unlike hypospadias, epispadias is very rare, occurring in one in 30,000 males and one in 400,000 females. The defect may be glandular (fig. 1455), penile, or total, the latter usually associated with ectopia vesicæ. In the first two varieties the urethral orifice is situated on the dorsum, and in the second variety the penis curves upwards as well.

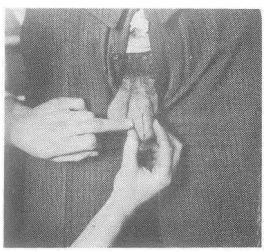


FIG. 1455.—Glandular Epispadias.

Treatment.—The operative treatment is similar in principle to that of hypospadias, and is somewhat less difficult to perform.

INJURIES OF THE URETHRA

Rupture of the urethra is divided into two distinct classes—rupture of the bulbous urethra, and rupture of the membranous urethra (*syn.* intrapelvic rupture) (fig. 1456). Each is again subdivided into complete and incomplete which relates to the circumference of the urethral wall, and total and partial which relates to the thickness of the wall.

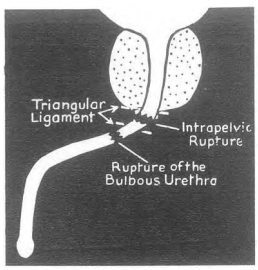


FIG. 1456.—Showing the two varieties of rupture of the urethra.

Rupture of the Bulbous Urethra

Rupture of the bulbous urethra is the more common accident. Almost without exception there is a history of a fall astride a projecting object. In the days of sailing-ships, the common cause was falling astride a spar from aloft To-day cycling accidents and a loose manhole cover (fig. 1457) account for a number of cases.

Clinical Features.—The triad of signs of a ruptured bulbous urethra is urethral hæmorrhage, a perineal hæmatoma, and retention of urine.

Preliminary Treatment and Investigation.—If the condition is suspected, in order to diminish the possibility of extravasation, *the patient should be told not to try to pass urine.* No attempt should be made to catheterise him until he is fit to be taken to an operation theatre, where asepsis can be

assured and operation can be undertaken in necessary cases. A suitable dose of morphine is given; a course of chemotherapy started. When circumstances are extenuating and the bladder is full, it should be emptied by suprapubic puncture (p. 1228) until the patient can be treated as above. If the patient has passed urine when first seen and there is no extravasation, then the rupture is partial and catheterisation should be avoided.

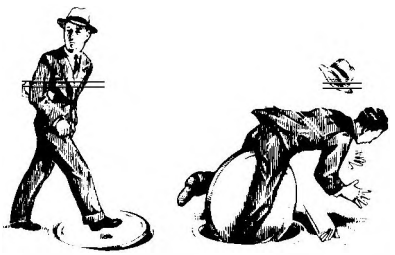


FIG. 1457.—The type of accident which results in rupture of the bulb of the urethra. (*After V. J. O'Conor.*)

Treatment

1. Under strict aseptic precautions an attempt is made to pass a plastic coudé catheter (14–16 F). The roof of the urethra is the most likely part to be intact, so the beak of the catheter should be directed towards this.

2. If the catheter passes it should be left in position for forty-eight hours. A perineal hæmatoma must be drained, and the mid-line wound packed.

3. If the catheter fails to pass, the site of the obstruction is explored, with the patient in the lithotomy position, through a mid-line perineal incision. Should it be impossible to define the proximal end of the ruptured urethra, a suprapubic cystotomy is performed and a fully curved sound passed down from the internal meatus. The exposed ends of the urethra are trimmed and the ventral aspect split longitudinally for 1·5 cm. The circumference is then sutured as a flat ribbon, with fine catgut, the knots being left on the outside of the wall (fig. 1458). The bladder, if opened, is drained by a suprapubic catheter. If the repair has been accomplished without cystostomy the catheter can be passed into the bladder from the urethrostomy (fig. 1458 (B)). A small balloon Gibbon-type catheter (12 F) is very satisfactory. The perineal wound is packed, not stitched.

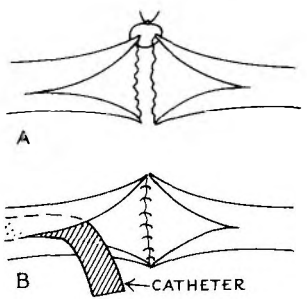


FIG. 1458.—Repair of ruptured bulbous urethra.

After-treatment.—A course of chemotherapy (e.g. Urolucosil 100 mg. six hourly) is continued until healing is complete. The urine must be recultured after a week. The perineal wound is irrigated daily with a weak Eusol (hypochlorite) solution. The catheter is removed after eight to ten days. A perineal leak will persist for a few days. Sounds must be passed two weeks after healing to calibrate the urethra, and a urethrogram, performed two months later, will reveal the result.

Complications:

Subcutaneous extravasion of urine occurs in total rupture if the patient attempts to pass urine (p. 1292).

Stricture.—This common complication is due, for the most part, to infection (p. 1296).

Rupture of the Membranous Urethra
Extraperitoneal rupture of the Bladder

Intrapelvic rupture of the urethra occurs in the membranous portion near the apex of the prostate (fig. 1456). Like extraperitoneal rupture of the bladder it may be due to penetrating wounds, but in civil life is more usually the result of fracture of the pelvis or dislocation of the symphysis pubis.

Norman Otway Knight Gibbon, Contemporary. Surgeon, Sefton Hospital, Liverpool, England.

Only about 10–15% of cases of fractured pelvis sustain either or both injuries.

Complete rupture with floating prostate	1–2%
Incomplete intrapelvic rupture of urethra	4–6%
Extraperitoneal rupture of bladder	4–6%
Combined urethra and bladder damage	1–2%

Clinical Features.—Signs of a fractured pelvis are present, with shock. Blood, and later, urine produce a swelling in the perivesical and peri-prostatic tissues and an assessment of mobility of the prostate may be difficult. In *urethral injuries* some blood leaks from the urethral meatus, and the bladder will distend before extravasation occurs (fig. 1459). In *extraperitoneal rupture* of the bladder, the organ will not be palpable (fig. 1378). However,

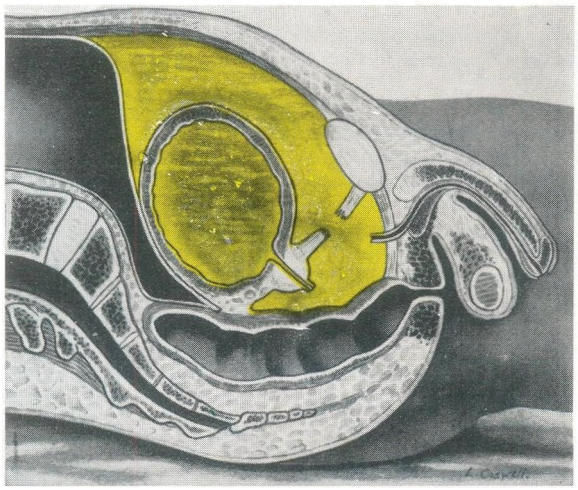


FIG. 1459.—Intrapelvic rupture of the urethra. Note the displacement of the bladder backwards, due to the tearing of the pubo-prostatic ligaments.

dullness and some tenderness extend upwards from the pubis, and an ill defined swelling is appreciable in all but the very obese.

Investigation.—A plain X-ray to demonstrate the fractures is essential, likewise a blood count, blood-grouping and cross-matching for blood transfusion because massive loss from pelvic veins can occur. Urethrography and attempts at cystography or catheterisation are damaging and may destroy any residual intact urethral wall. They must not be permitted. It is less dangerous to wait to see if the patient will pass urine (Mitchell).

Treatment.—Blood loss and shock are treated. As soon as the diagnosis is reasonably certain a suprapubic catheter should be inserted (under general anæsthesia if the patient is fit). On opening the suprapubic tissues, any fluid should be aspirated, but no attempt should be made to displace a hæmatoma, or explore the area behind the pubis.

The bladder is opened and by inserting two fingers it is possible to ascertain that the *prostate is not floating* so high that total rupture is certain. At

John Phillimore Mitchell, Contemporary. Professor of Urology, United Bristol Hospitals.

the same time a depressed fracture of the symphysis can be pushed forward. Provided the prostate is not grossly displaced, the bladder is closed round a large (28F) Malécot catheter. The wound is closed loosely round the catheter to allow drainage from the perivesical space. An antibiotic should be given to diminish the risk of infection in the hæmatoma.

The urethra is subsequently explored three weeks after the injury by careful visualisation via a panendoscope. In many cases this can be manipulated past blood clot and into the bladder. Occasionally a guiding sound passed from the bladder downwards may assist the manoeuvre. If this can be achieved then the suprapubic tube may be clipped off after a further three days and a trial of micturition made. In those cases where the block cannot be negotiated re-exploration and urethroplasty will be necessary.

If the prostate is floating, and there is no doubt that the rupture is complete and total, then the older classical method, railroading a catheter across the gap, and drawing the prostate down on to the triangular ligament, should be adopted.

Railroad technique.—A metal bougie is passed from the bladder through the internal meatus with the retropubic space. A second metal bougie is passed from the external urinary meatus. The two bougies are manipulated until their tips touch (fig. 1460). By slowly withdrawing the first bougie and steadily advancing the second, while keeping their tips in contact, it is possible to guide the second bougie into the bladder past the seat of the rupture. The first bougie is withdrawn and a

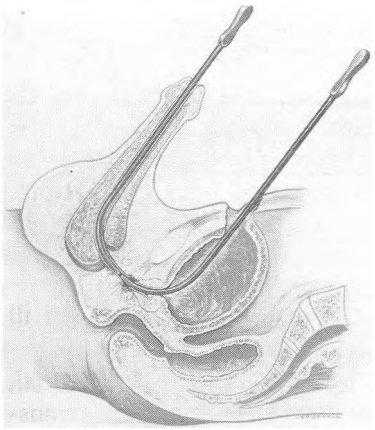


FIG. 1460.— Intrapelvic rupture of the urethra. Showing the tips of the metal bougies in contact.

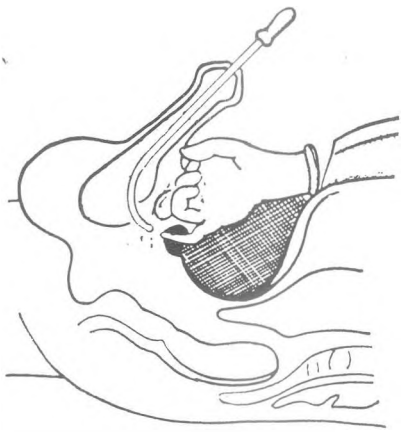


FIG. 1461.—Shahade and Shah procedure (see text).

piece of plain rubber tubing of such a size as to fit tightly is threaded on to the beak of the second bougie, which is withdrawn, carrying with it the rubber tubing. Outside the external urinary meatus the bougie is disengaged from the rubber tubing, and to the latter is fastened, by means of a stitch, the tip of a Foley's catheter. By pulling on the vesical end of the rubber tubing the tip of the Foley's catheter is drawn into the mouth of the suprapubic wound, where it is disconnected from the rubber tube.

At this stage it is advisable to tie a long piece of silk firmly to the tip of the Foley's catheter, and to wind the excess around a wooden spatula. (Thus, should the balloon burst, the Foley's catheter can be replaced by the rail-road method just described).

Achille-Étienne Malécot, 1852–?. He invented his catheter while Interne des Hôpitaux de Paris, France.
Frederic Eugene Baisal Foley, 1891–1966. Urologist, Miller and Ancker Hospitals, St. Paul, U.S.A.

FIG. 1462.—Malécot catheter.

This completed, the balloon (30 ml.) of the catheter is moderately distended with water and the side tube is ligated (fig. 1463).

The *Shahade and Shah procedure* (fig. 1461) employs the little finger instead of the second metal bougie as it makes the operation easier.

Concluding the Operation. — The catheter is drawn down so that the balloon rests upon the bladder neck. The bladder is closed around a Malécot catheter (fig. 1462), and the prevesical space is drained with corrugated rubber.

After-treatment:

Urological. — (1) By the mediation of a length of silk, attached to the bell end of the catheter and transfixing both walls of the catheter, extension is applied to the catheter via a pulley and a weight; thus the severed ends of the urethra are brought into contact (fig. 1463). The weight applied is 900 g for the first three days and 450 g for the next ten days. Bed-cages are arranged so as to prevent the bedclothes touching the taut silk cords.

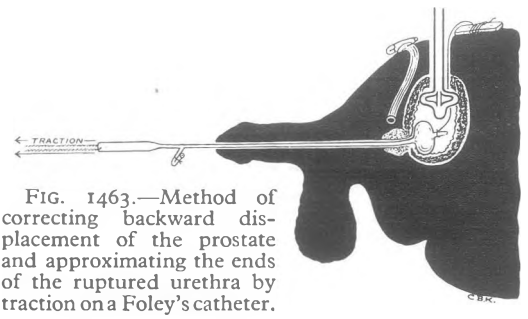


FIG. 1463.—Method of correcting backward displacement of the prostate and approximating the ends of the ruptured urethra by traction on a Foley's catheter.

(2) Suprapubic drainage of the bladder is maintained for fourteen to twenty-one days.

(3) The urine is kept alkaline and antibiotic treatment is given until the patient has a sterile urine and completely healed wounds.

(4) On or about the twelfth day the Foley's catheter is removed, and during the process the silk attached to its tip is unwound from the spatula; thus the silk lies along the whole length of the urethra, one end being tied to the spatula and the other emerging from the external urinary meatus. The suprapubic tube can be clipped off at about the fourteenth day, and if the patient passes urine easily it can be removed. The silk thread should be retained for the passage of bougies by the rail-road method at the end of another week, and then it should be removed, unless the insertion of another catheter becomes necessary.

Orthopædic.—For management of the fractured pelvis, see p. 411.

EXTRAVASATION OF URINE

Superficial extravasation occurs in neglected cases of complete rupture of the bulbous urethra, i.e. when operation is not undertaken within twelve to twenty-four hours, and in ruptured periurethral abscess.

The extravasated urine cannot pass (1) behind the mid-perineal point, because of the attachment of the perineal (Colles's) fascia to the triangular ligament; (2) into the thighs, for the deep layer of the superficial fascia of the abdominal wall (Scarpa's fascia) blends with the pubic portion of the fascia lata just distal to the inguinal ligament; (3) into the inguinal canals, because of the intercolumnar fibres and fascia of the external oblique (external spermatic fascia).

M. G. Shahade, Contemporary. Formerly Professor of Surgery, Govt. Medical College, Surat, Gujarat, India.
V. C. Shah, Contemporary. Surgeon, Rajkotwthin, Gujarat, India.
Antonio Scarpa, 1747–1832. Professor of Anatomy, Pavia, Italy.

It therefore must pass (1) into the scrotum; (2) beneath the superficial fascia of the penis; (3) up the abdominal wall beneath the deep layer of the superficial fascia (fig. 1464).

Treatment.—Urgent operation is a necessity.

Multiple incisions are made in the infiltrated tissues of sufficient depth to penetrate the limiting fascia. By the time extravasation has occurred it is unlikely that the urethra can be repaired in the way described already (p. 1290), for the sutures would cut out of the œdematous inflamed tissues. It is therefore often advisable to adopt the sutureless operation of Rutherfurd. Suprapubic cystostomy is performed and a metal bougie is passed through the internal urinary meatus. The patient having been placed in the lithotomy position, another metal bougie is passed from the external urinary meatus to the perineum, and perineal section is carried out. A Foley's catheter is introduced from the external urinary meatus into the bladder in the same way as that described for intrapelvic rupture of the urethra, and the bladder is closed around a de Pezzer catheter. The perineal wound is left widely open. The urethral catheter is removed after three or four days. In other respects the after-treatment is similar to that for complete rupture of the bulbous urethra.

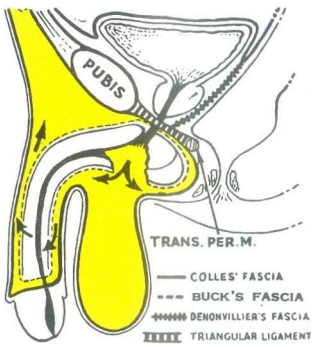


FIG. 1464.—The fascial planes concerned in superficial extravasation of urine. Trans. Per. M. = Transverse Perineal muscle.

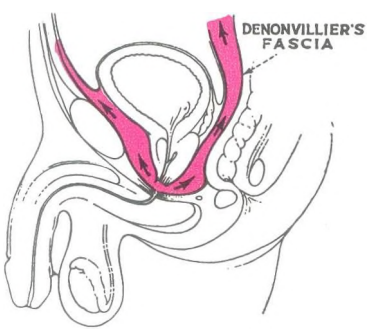


FIG. 1465.—Area occupied by extravasated blood and urine in the case of intrapelvic rupture of the urethra.

Deep extravasation (fig. 1465) occurs in the case of extraperitoneal rupture of the bladder, intrapelvic rupture of the urethra, and after suprapubic puncture, when the bladder has been allowed to refill. It can also result from rupture or perforation of a ureter, or unnoticed damage to the bladder or prostatic capsule during per-urethral resections. In these conditions urine extravasates in the layers of the pelvic fascia and in the retro-peritoneal tissues.

Treatment.—When extravasation is proceeding from the bladder, it is necessary to drain the prevesical space (cave of Retzius) and to perform suprapubic cystostomy. The treatment of the various conditions which give rise to deep extravasation of urine is considered in the appropriate sections.

INFLAMMATIONS

Ulceration of the Urethral Meatus

Ulceration of the meatus in young male children is a common clinical entity. It is never found in the uncircumcised. It is common after circumcision, though an interval of three to eighteen months may elapse between the operation and the onset of symptoms. Lack of protection given by the prepuce is the initial cause. Friction of the clothing and ammoniacal urine are important secondary ætiological factors. The ulcer causes a scab to form which closes the meatus, and the child can only urinate by bursting this scab. This process is usually accompanied by pain and

Henry Rutherfurd, 1861–1929. Surgeon, Royal Infirmary, Glasgow, Scotland.
Oscar Michel Benvenuto de Pezzer, 1853–1917. Assistant in the Urological Department, Necker Hospital, Paris.
Andreas Adolf Retzius, 1796–1860. Professor of Anatomy and Physiology, Carolina Institute, Stockholm.

screaming, and a few drops of blood may be passed. Ulceration and scab formation alternate, and if neglected, cicatrical contracture of the meatus may result eventually in an acquired pin-hole meatus.

Treatment.—A simple regimen of washing the napkins in boric acid crystals after boiling and applying 5% boracic ointment (or Drapolene) locally is usually curative. These measures neutralise the alkaline urine. A few cases require meatotomy (p. 1285).

Urethritis

Gonorrhœa.—A sexually transmitted disease due to *neisseria gonorrhœa* (gonococcus), a gram negative kidney shaped diplococcus, which affects primarily the anterior urethra in the male and the urethra and cervix in the female.

Gonorrhœa in the male.—Three to five days after intercourse with an infected partner there occurs some discomfort on micturition often progressing to a scalding pain, a urethral discharge ensues, seropurulent at first, becoming purulent and yellowish. In chronic cases and male carriers (5 per cent of the total), a slight purulent morning discharge, or some slight staining of the underclothes may be the only signs.

Investigations.—The gram stained urethral smear will show numerous pus cells and both intra- and extra-cellular gram negative diplococci. The two glass urine test:—urine in the first glass is hazy, but clear in the second glass. Smears taken soon after micturition may show no organisms, and in those from carriers and chronic cases, the diplococci may be scanty and predominantly extra-cellular. Urethral cultures should always be taken, but treatment need not await confirmation when smear and history are typical. The Gonococcal Complement Fixation Test (G.C.F.T.) is of limited value. It is useless in early uncomplicated cases, frequently negative in chronic complicated cases, and sometimes positive in the absence of any gonococcal infection past or present.

Complications.—Local complications are now comparatively rare and metastatic complications very rare. All can be prevented by effective early treatment followed by full tests of cure.

Local complications include posterior urethritis when urine in the second glass is hazy and contains threads; Prostatitis acute or chronic (p. 1280); acute epididymitis, peri-urethral abscess, and urethral stricture (p. 1296). The glands of Littré are frequently involved and Cowpers glands more rarely. Systemic (metastatic) complications include gonococcal arthritis, iridocyclitis, septicæmia and endocarditis.

Treatment.—Over 90% cure rates are claimed for the following schedules. Ampicillin 3 g stat. with Probenecid 2 g to delay excretion; Procaine Penicillin 1·2 to 2·4 mega units i.m. with Probenecid; Co-Trimoxazole (Septrin) 4 tablets b.d. for two days. Spectinomycin 2–3 g stat.; Kanamycin 2 g stat.

Septrin and Kanamycin are non-treponemicidal and will not mask a concomitant syphilitic infection.

Non-gonococcal Urethritis (N.G.U.) or Non-specific Urethritis (N.S.U.)—is an acute or subacute inflammation of the male urethral mucosa,

Alexis Littré, 1658–1726. Surgeon and Anatomist, Paris.
William Cowper, 1666–1709. Surgeon and Anatomist, London.

uncertain in origin, but related to sexual activity and with a tendency to relapse. The commonest of the sexually transmitted diseases, the cause remains undiagnosed in well over one half of all cases. Of the known causes *trichomonas vaginalis* accounts for 5% or more of all cases (fig. 1466). When present in the male urethra it is often symptomless, the male acting as a carrier, but if it produces a urethritis the signs are indistinguishable from other causes of N.G.U. and the parasite is difficult to demonstrate by hanging drop or culture. Urethritis due to candida albicans, chemicals, trauma, or secondary to upper urinary tract infection together account for less than 5 per cent of all cases. *Bacterial causes.*—Staphylococci, Streptococci, coliforms, diphtheroids, hæmophilus vaginalis, and pleuro-pneumonia-like organisms (P.P.L.O., mycoplasma) have all been incriminated as causative but their role as primary pathogens is doubted being regarded as contaminants or secondary invaders or, as in the case of P.P.L.O., being found in a significant proportion of clinically normal, sexually active men and women. *Chlamydia trachomatis*, a member of the Trachoma, Lymphogranuloma, Psitticosis group of virus-like particles can be isolated from the discharge in 30 to 70 per cent of cases of N.G.U. and from the cervical scrapings in a significant proportion of their consorts. Positive isolates may be obtained from these sites from the parents of an infant who develops neonatal inclusion conjunctivitis.

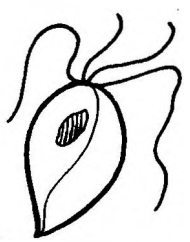


FIG. 1466.—*Trichomonas vaginalis.*

Symptoms include a mucopurulent or purulent urethral discharge 10–14 days after risk, slight discomfort on micturition, or rarely, marked dysuria with frequency and urgency—sometimes symptomless or a slight seropurulent morning discharge.

Investigations.—Urethral smear and culture are negative for the gonococcus. Urine is hazy in the first glass, clear in the second. Hanging drop and culture for *T. Vaginalis*, urethral chlamydia culture and cervical culture from the consort.

Complications.—Chronic prostatitis. Reiter's syndrome (1% of cases) below.

Treatment.—Tetracycline 250 mg 1 q.d.s. for 10 days. A long acting Tetracycline ('Deteclo', 'Tetrabid' 'Ledermycin' etc. 1 b.d. for 3–4 weeks). Metronidazole 250 mg 1 t.d.s. for 7 days or 400 mg 1 b.d. for 5 days in cases where *T. vaginalis* is proved or found in the partner.

REITER'S DISEASE

The condition is venereal in origin and commences as a subacute urethritis four to six weeks after contact. The discharge is usually devoid of organisms, being clear and viscid. A few days later conjunctivitis occurs, at first unilateral, and then bilateral. Usually in ten days to two weeks arthritis supervenes (acute hydrarthrosis of at least one joint is present in every case). Another concurrent manifestation that often accompanies the onset of arthritis is keratoderma blennorrhagicum, consisting of nodules, vesicles, and pustules, frequently found on the soles of the feet (fig. 1467). Pleuropneumonia-like organisms have been isolated from the various lesions of this condition in approximately 30% of cases.

Hans Reiter, 1861–1969. President of the Health Service and Honorary Professor of Hygiene, Berlin. Described Reiter's Disease in 1916. The syndrome had been noted previously by Sir Benjamin Brodie.

FIG. 1467.—Reiter's disease—keratoderma blennorrhagicum of the heel.

Differential Diagnosis.—In untreated gonorrhœa, arthritis and ophthalmic infection were not rare in the past. The absence of the gonococcus is of the highest importance in coming to the conclusion that the symptoms are due to Reiter's disease. In Reiter's disease the urethritis, as well as the arthritis, is milder and the incubation period is much longer than in gonorrhœa. Furthermore, never are the ocular manifestations of Reiter's disease so destructive as those of gonorrhœa.

Prognosis.—The urethritis and conjunctivitis frequently subside in a few weeks, but more often than not the arthritis persists for many months.

Treatment.—Clearly the ophthalmic complications must be treated thoroughly following the usual lines (eye-baths and shades). Various antibiotics and fever therapy induced by graduated doses of triple typhoid vaccine (TAB) have been tried, the last being given for the arthritis. There is increasing interest in work which may implicate trachoma infection and its treatment in certain cases of non-specific urethritis.

URETHRAL STRICTURE

Congenital.

Traumatic — Bulbous. / Membranous.

Inflammatory — Post-gonorrhœal / Post-urethral chancre. / Tuberculous.

Instrumental — Indwelling catheter. / Following the passage of a large calibre endoscope, notably a resectoscope.

Post-operative — Prostatectomy. / Amputation of the penis.

Post-gonorrhœal stricture, which has become very much less common since the introduction of the antibiotic treatment of gonorrhœa, is situated most frequently (*a*) in the bulb (70 per cent.); (*b*) at the peno-scrotal junction; (*c*) in the distal part of the spongy urethra, in that order. The membranous and prostatic parts of the urethra are exempt.

Multiple strictures are relatively common. When there are two strictures, the deeper is the narrower; when there are three strictures, the deepest is the narrowest. If a stricture in the penile urethra has a very narrow orifice, there is seldom another stricture behind it.

Pathology.—Following inadequately treated gonorrhœa, infection persists in the periurethral glands, and spreads to the periglandular tissues, which become infiltrated with round cells and fibroblasts. Gradually the infiltrated periurethral tissues contract with the formation of scar tissue, localised thrombophlebitis of the corpus spongiosum playing a part in the more dense varieties. Whereas in the bulbous urethra the fibrosis is most in evidence in the roof, in the penile urethra it predominates in the floor. Most strictures develop during the first year after gonorrhœal infection, but they may not give rise to difficulty in micturition for ten to fifteen years.

Clinical Features.—In a stricture of large calibre the only symptoms are the occasional passage of flakes (desquamated epithelium) in the urine and a

varying amount of urethral discharge (gleet), most in evidence in the early morning (the morning 'dew-drop'). Often these symptoms are neglected until the diminished calibre of the urethra causes considerable difficulty in micturition. In contradistinction to obstruction due to an enlarged prostate, the patient finds he must strain to empty the bladder. Another distinguishing feature is the patient's age. He is often considerably younger than the prostatic sufferer, or the symptoms date back to some time prior to the fiftieth year. The stream becomes progressively narrower, micturition is prolonged, and after it has seemingly ended dribbling occurs. This is due to urine trickling from the dilated urethra above the stricture. Increasing frequency of micturition, at first during the day and then both by day and by night, is another common complaint, due either to incomplete emptying of the bladder at each act of micturition, or to cystitis, or to both. In long-standing cases it is often possible to palpate the stricture from without as an induration in the urethral floor. The evil effects of urethral obstruction upon the bladder, ureters, and kidneys are similar to those of prostatic obstruction (p. 1265). Untreated, sooner or later retention of urine supervenes. Sometimes acute retention sets in while the stricture is still of moderate calibre; it is then due to superadded œdema of the urethral mucous membrane in the neighbourhood of the stricture, brought about by voluntary retention, alcoholic excess, or recrudescence of local infection. In other cases narrowness of the stricture results in increasing inability to expel residual urine, and acute-on-chronic retention, or retention-with-overflow, supervenes.

Urethroscopy renders the diagnosis of urethral stricture very precise.

The stricture can be seen as a white scar of fibrous tissue, and its position in the urethra, the size of its contained lumen, and its dilatability can be judged (fig. 1468).

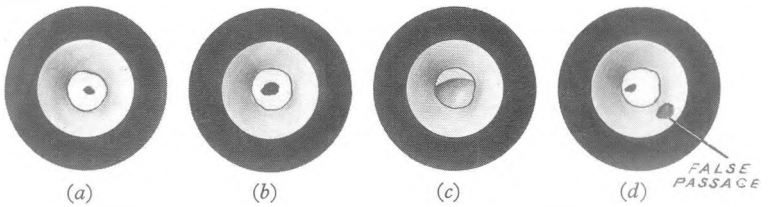


FIG. 1468.—Urethroscopic appearances. (*a*) Fine-bore stricture. (*b*) Moderate-bore stricture. (*c*) Crescentic stricture of the roof. (*d*) Stricture with false passage.

Often a stricture encircles the whole urethra, and the lumen is more or less centrally placed, but from time to time a variety of urethroscopic appearances is noted in individual cases; for instance, the stricture may take the form of a crescent.

False passages may be seen. They are recent penetrations of the urethra in front of the stricture due to unskilful attempts to pass a bougie. False passages are particularly liable to bleed, and it is essential to abandon æro-urethroscopy if urethral hæmorrhage occurs, for fatal air embolism has resulted from air being pumped into the cavernous tissue through a urethral wound.

Urethrography (p. 1165) supplies information concerning the length of a stricture, of dilatation or diverticulum formation above the stricture, or failure of the medium to pass a stricture (figs. 1469 and 1470).

Instrumental Treatment.

Intermittent Dilatation.—In the majority of instances, provided the patient attends regularly, dilatation at suitable intervals is a satisfactory form of treatment. Before each dilatation the patient should pass urine, the glans

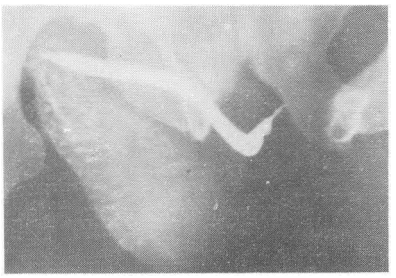


FIG. 1469.—Urethrogram showing a stricture of the membranous urethra following fracture of the pelvis.

FIG. 1470.—Gonnorrhœal stricture of the bulbous urethra. Note that some contrast has entered the penile veins (p. 1165).

penis and urinary meatus are cleansed and the urethra is filled with a mild antiseptic solution, and local anæsthetic in a methyl cellulose base (Lidothesin). Dilatation must be carried out gently with bougies of increasing size.

On no account should the stricture be forcibly or over-dilated, both of which result in trauma that induces inflammatory œdema and subsequent formation of more fibrous tissue.

Strictures of very small calibre should be dilated twice a week at first. With this exception it is sufficient for the patient to attend for treatment at regular intervals, and an erstwhile reminder to the patient was that he must return for treatment 'once a week for a month, once a month for a year, and every year on his birthday'. A

FIG. 1471.—Gum-elastic bougie.

few strictures are cured by full dilatation, but in the majority the patients must be kept under supervision for the remainder of their lives.

Gum-elastic bougies (*syn.* French bougies) (fig. 1471) should be the standard instruments in the early stages of treatment of all but the very finest strictures. It is usual to commence with a No. 10 French, and if this cannot be passed, progressively smaller, or even filiform bougies are employed.

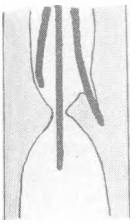


FIG. 1472.—'Faggot' method of introducing a bougie through a stricture.

Filiform bougies are gum-elastic bougies varying in size from 1 to 3 French. If one fails to pass, it is often valuable to insert several as far as the stricture. By manipulating each back and forth, frequently one of them can be induced to negotiate the stricture (fig. 1472). If this method fails, it is sometimes possible to pass a filiform bougie through the stricture under the vision afforded by a urethroscope. If any bleeding has been occasioned by the attempt to pass bougies, urethroscopy should be postponed for several days. Very few urethral strictures are impassable, but it often requires patience to insinuate even the finest urethral guide: only when even a filiform bougie cannot be passed on three successive occasions is a stricture held to be impassable.

Filiform Bougies with Followers.—Filiform bougies furnished with threaded hollow mounts at their proximal ends are to be preferred, because screw-ended gum-elastic bougies of a larger size can be attached and guided by the filiform through the stricture. In this way many strictures of very small calibre can be dilated sufficiently to render subsequent dilatations less tedious.

FIG. 1473.—Filiform bougie with follower.

By means of gum-elastic bougies a stricture is dilated up to the size of 14 French. Thereafter metal bougies are to be preferred.

FIG. 1474.—Lister's metal bougie.

Lister's metal bougies (*syn.* English bougies) (fig. 1474) are indicated after the second or third attendance of the patient, when it has been proved that the stricture is responding to dilatation by the French bougies. Metal bougies of a smaller size than 7/9 English should not be used, for fear of making a false passage.

Continuous dilatation necessitates some days of in-patient treatment, but it is of immense benefit in cases where little or very slow progress is made by intermittent dilatation. In a number of instances continuous dilatation obviates the necessity for operative treatment. After dilatation with gum-elastic bougies a catheter of corresponding size is tied into the urethra. On its removal two or three days later it will be found possible to pass much larger bougies.

Operative Treatment

1. External urethrotomy (Wheelhouse's operation) is the operation of choice when a patient presents with acute retention and the stricture proves impassable.

With the patient in the lithotomy position the Wheelhouse staff (fig. 1475) is passed down to the face of the stricture, and the urethra distal to the stricture opened by cutting down on to the groove in the staff. Stay sutures hold the edges open to display the face of the stricture. The mid-line incision is then carried backwards

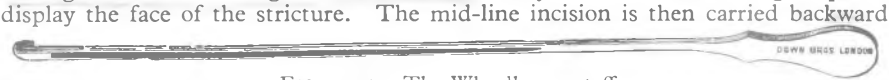


FIG. 1475.—The Wheelhouse staff.

until the dilated proximal dilated urethra is opened. Urine will usually leak out under some pressure. A catheter is now passed from the external meatus across the opened urethra into the bladder, and the wound packed lightly. *Alternatively* the operation can be concluded formally as the first stage of a urethroplasty (see below) by stitching skin edge to mucosal edge.

2. Internal Urethrotomy.—Used to be widely practiced employing the Maisonneuve or Thompson Walker urethrotome as a blind procedure, becoming less popular with the advent of urethroplasties. Now with instruments as the Stortz urethrotome this simple operation is becoming popular again. It can only be used if the stricture is passable—a ureteric catheter being passed under vision through the stricture and the knife used to cut all the fibrous bands on the dorsum of the urethra. A catheter is left indwelling for 2–3 days. Urethroscopy should be repeated in three months.

3. Urethroplasty.—Nowadays surgical cure by means of a two-stage urethroplasty is applicable to all strictures of the bulbar and penile portions, and is the treatment of choice.

Johanson's Urethroplasty.—The urethra is exposed by a mid-line incision in the skin overlying the stricture. A Wheelhouse staff as described above is used to open the normal urethra for 2 cm. distal to the stricture. The urethra is then cut clearly in the midline, through the stricture and 2 cm. into proximal normal urethra. The cut edges of skin and urethra are stitched to each other, leaving a defect in the urethra

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Claudius Galen Wheelhouse, 1826–1909. Surgeon, General Infirmary, Leeds, England.
Jules Germain Francois Maisonneuve, 1809–1897. Surgeon, France.
Sir John William Thomson-Walker, 1870–1937. Surgeon, St. Peter's Hospital for Stone, London.
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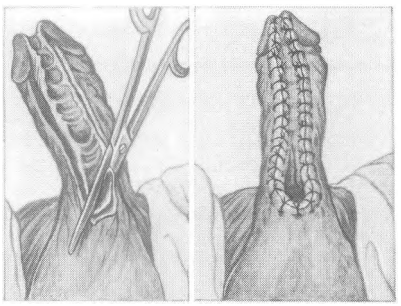
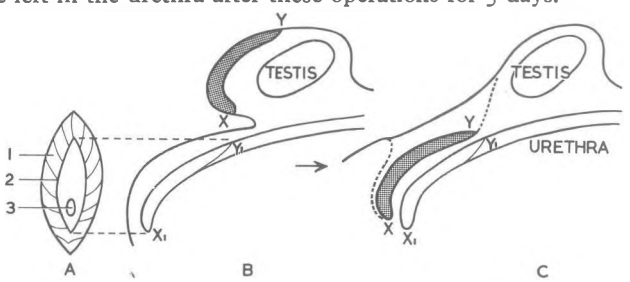


FIG. 1476.—Johanson's urethroplasty. In this case much of the urethra was strictured from the meatus.

through which urine will be passed while the stricture contracts (fig. 1476). When the contraction is complete (3–4 months) the urethra can be reconstructed by employing the principle of the Denis Browne repair of hypospadias (p. 1287), or constructing a urethra from the adjacent skin (fig. 1477). The skin outside the incision is mobilised to close in front of the reconstructed urethra, but a dorsal relieving incision may be necessary.

Where the stricture is in the bulb the first stage is similar. The urethral floor is divided up to and into normal urethra. In many cases this entails splitting the urethra through the external sphincter up into the wide prostatic urethra. In order to stitch skin between the edges, either a perineal skin flap has to be constructed at the time of skin incision (Blandy), or a scrotal funnel (Turner-Warwick) displaced backwards to be sutured to the open edges of the urethral defect (fig. 1478). Second stage closure is simple, a complete tube being reconstructed. A small catheter should be left in the urethra after these operations for 5 days.

FIG. 1477.—Urethroplasty for the posterior part of the bulb using a funnel of scrotal skin.
1. Depressed funnel of skin.
2. Edge of urethra.
3. Membranous urethra opening.



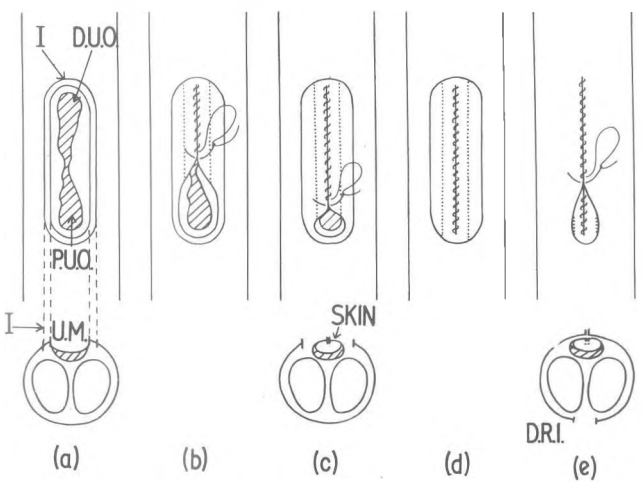


FIG. 1478. — Repair of urethral stricture. Diagrams show underside of shaft of the penis. (*a*) Incisions (I) are made to encompass the whole area — D.U.O.= distal urethral opening, P.U.O.=proximal urethral opening, U.M.= urethral mucosa. (*b*), (*c*) (*d*) 3/0 chromic catgut suture of innermost skin edge to form a tube. (*e*) Covering skin sutured and tension avoided by a dorsal relieving incision (D.R.I.).

Other Causes of Urethral Stricture

~~**Congenital stricture has been considered on p. 1285.**~~

Traumatic stricture follows unskilful or delayed treatment of rupture of the urethra. If dilatation is unsatisfactory, a urethroplasty should be done.

The so-called stricture following neglected or untreated rupture of the membranous

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urethra is often not a stricture at all, but a complete loss of continuity of the urethra (fig. 1459). If, as frequently occurs, it proves impossible to keep a passage dilated by the usual methods, the only alternative to transplantation of the ureters is Badenoch's pull-through operation. After the bladder has been opened suprapubically, with the patient in the lithotomy position, a metal sound is passed up the urethra and when arrested by the stricture its tip is cut down upon in the perineum. The distal urethra is then freed as a tube for 4 to 6 cm. A large metal sound is passed suprapubically down the urethra and cut down upon from the perineum until it can be forced through the area of fibrosis. A rubber catheter is attached to it by the butt end and pulled back through the suprapubic opening. The freed distal urethra is threaded over the lower end of the catheter and its end stitched to the catheter some 10 cm. from its tip with interrupted chromic catgut stitches. Traction on the catheter then guides the distal urethra into the prostatic urethra where it is held by attaching an artery forceps to the catheter as it emerges on to the abdominal wall (fig. 1479). Finally, the perineum is closed and suprapubic cystostomy temporarily established.

Post-instrumental stricture can occur in any part of the urethra from trauma due to the passage of too large an endoscope, or from urethritis from an indwelling catheter. To avoid the former, some surgeons advise performing perineal urethrostomy in order to pass a resectoscope in patients with a narrow urethra.

Post-operative Stricture.—A stricture develops in about 4 per cent. of cases after prostatectomy, irrespective of the method employed. The stricture is usually situated in the proximal end of the prostatic urethra. In many cases the stricture can be dilated by regular intermittent dilatation. When the stricture takes the form of a shelf at the junction of the bladder with the prostatic bed, the bladder must be opened and the shelf excised.

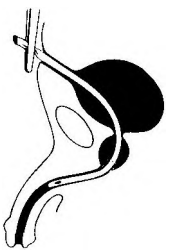

FIG. 1479.—Badenoch's pull-through operation completed.

Post-operative stricture can also follow partial or complete amputation of the penis. Methods of avoiding this complication are described in the section dealing with these operations (p. 1313). Regular dilatation is satisfactory.

Complications of Urethral Stricture

1. Retention of urine (p. 1227).
2. Urethral diverticulum.
3. Periurethral abscess.
4. Urethral fistula.
5. All the attendant evils of 'back pressure', culminating in bilateral hydronephrosis, combined with a susceptibility to urinary infection and an increased liability to urinary calculus.
6. Hernia, hæmorrhoids, or rectal prolapse may be induced by the straining.

Diverticulum of the male urethra (*syn.* urethral pouch)

1. Congenital.
2. Due to increased intraurethral pressure behind a stricture.
3. Due to the long-standing presence of a urethral calculus.
4. Indwelling catheters in paraplegics.

In many cases the pouch can be seen and those which are not obvious at first become so when the patient interrupts the stream of urine.

Treatment is excision of the diverticulum and removal of the cause if possible.

PERIURETHRAL ABSCESS

There are two main varieties of acute abscess:

Penile periurethral abscess arises as an infection of one of the glands of Littré, and is usually a complication of acute gonococcal urethritis. A tender induration can

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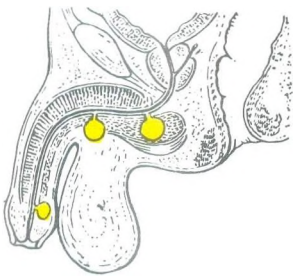

FIG. 1480.—Common situations of a periurethral abscess. (*After F. Papin.*)

be felt on the under-surface of the penis (fig. 1480). Left to nature, frequently the abscess bursts externally, and a urinary fistula is liable to result.

Treatment.—The passage of a bougie often causes the abscess to burst into the urethra. When this is unsuccessful, a ureteric meatotome is passed through a urethroscope and the abscess is opened by diathermy. When an abscess lies behind a stricture of the urethra it must be drained externally.

Bulbar periurethral abscess runs a variable course. Its most acute form, formerly termed periurethral abscess with extravasation of urine, is better termed periurethral phlegmon, because in 50% of cases there is no stricture of the urethra present. Of the remainder, the majority have a passable stricture, while in the minority the stricture is impermeable. Consequently, in the majority of cases there is no reason why urine should extravasate. The condition is due to a spreading cellulitis caused by streptococci and anaërobic organisms invading the same cellular plane as that of superficial extravasation of urine (p. 1292).

Clinical Features.—There is pain in the perineum, a high temperature, repeated rigors, and a rapid pulse-rate. In early cases the tenderness and swelling are limited to the perineum. Later, often in a matter of hours, the scrotum and then the penis becomes œdematous, and the infection spreads beneath the superficial fascia of the abdominal wall.

Treatment.—Antibiotics have greatly improved the prognosis.

Operation.—The perineal abscess should be opened. In addition, if spreading cellulitis is present, incisions deep enough to divide the superficial fascia are made into the infiltrated scrotum, penis, and abdominal wall, wherever considered necessary. When there is partial or complete retention of urine and if the infection is limited to the scrotum and the penis, after changing gloves and instruments, suprapubic cystostomy should be performed. If the superficial layer of the abdominal wall is implicated, it is best to drain the bladder by performing perineal urethrostomy. It should be noted that the abscess usually communicates with the urethra by a minute opening which cannot be found.

After-treatment.—After instilling hydrogen peroxide into them, the various incisions are irrigated with weak potassium permanganate solution. Later sitz baths[1] of the same solution are given. When the infection has been overcome, the stricture of the urethra, if present, is treated by one of the methods described already.

Chronic periurethral abscess is nearly always situated in the perineum (fig. 1481) and is associated with much periurethritis. It is almost invariably the result of a long-standing stricture of the bulbous urethra. The abscess should be opened, together with the various pockets that are often present. Later the associated stricture must receive adequate treatment. The condition is liable to be complicated by a urethral fistula which occurs either spontaneously or as a result of incision of the abscess.

[1] Sitz bath = in which the patient sits in a bath-tub, bathing only the hips and the buttocks.

URETHRAL FISTULA (p. 1244)

The most frequent cause of an acquired external urethral fistula is bursting or incision of a periurethral abscess. When the opening is situated in the penile urethra or at the peno-scrotal junction, the amount of urine that escapes at each act of micturition is often small. A fistula following a periurethral abscess of the bulbous urethra can be either single or multiple. In the latter case the fistulæ originate behind a tight stricture and the patient passes most or all of his urine through the various fistulæ (watering-can perineum). A fistula can also follow external urethrotomy when there is a stricture situated more distally.

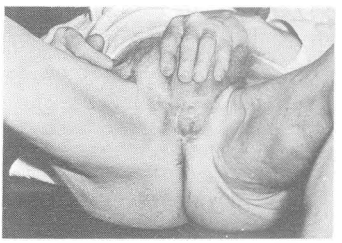

FIG. 1481.—Chronic periurethral abscess.

Treatment.—A small fistula often closes spontaneously after repeated dilatation of the urethra. Searing the track with the diathermy needle often encourages closure. Occasionally urethroplasty (p. 1299) is indicated.

URETHRAL CALCULUS

Calculi occur less frequently in the urethra than in any other part of the urinary tract. A urethral calculus can arise primarily in the urethra behind a stricture or in an infected urethral diverticulum. The latter are composed of phosphates. Less rarely a calculus which has migrated from the ureter becomes arrested in the prostatic, bulbous, or penile portions of the urethra. Migratory calculi are arrested in the urethra relatively frequently in children under the age of two, the explanation being that the comparatively large vesical neck allows them to pass out of the bladder.

Clinical Features.—In the case of a migratory calculus arrested in the urethra, typically there is a history of an attack of ureteric colic two or three days previously. During micturition the patient experiences sudden pain in the urethra and the stream of urine ceases abruptly. A few drops of blood-stained urine follow, and then there is retention of urine. A stone can be palpated readily through the floor of the urethra. When the stone has been arrested in the prostatic urethra a rectal examination usually reveals a tender, hard nodule in the middle line of the prostate, generally near its apex. A calculus forming behind a urethral stricture often does so without causing much additional discomfort and it sometimes attains a considerable size before giving rise to retention of urine or painful dysuria. In some cases such a stone can be felt easily; in others, owing to periurethral thickening, its presence is not suspected until it is seen at urethroscopy, or a grating sensation is experienced on passing a gum-elastic bougie or a characteristic click is heard if a metal bougie has been employed. In all but superficially placed calculi giving rise to acute symptoms, radiography is necessary to confirm the presence, and particularly to reveal the size of the calculus (fig. 1482) before commencing treatment.

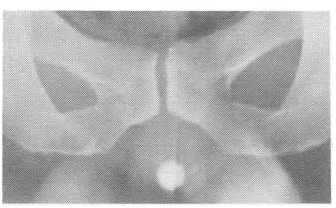

FIG. 1482.—Radiograph showing calculus impacted in the bulbous urethra.

Treatment:

(1) *Prostatic Urethra.*—When the stone is arrested in the prostatic urethra a general anæsthetic is given, and a metal bougie is inserted along the urethra as far as the calculus. In cases of recent impaction the stone can be pushed into the bladder, where it can be crushed by litholapaxy and the fragments evacuated. In cases of long-standing the stone is hour-glass shaped, and impacted firmly, in which event it is best approached through the bladder.

(2) *Membranous* and (3) *Bulbous Urethra.*—Occasionally a small stone can be removed from the deeper parts of the urethra by means of urethral forceps passed through a urethroscope. On no account must aero-urethroscopy be employed (danger of air embolism if bleeding occurs). A stone impacted in the proximal urethra sometimes can be manipulated forward if the urethra is distended with lotion. Another method is to pass a number of filiform bougies beyond the stone, twist them, and endeavour

to dislodge the calculus by pulling all the bougies out together. A stone impacted in the fossa navicularis, or manipulated into this position, sometimes can be removed by urethral forceps, or by inserting a probe behind it.

Operative Measures (situations 2 and 3).—Meatotomy is often required to permit the stone to be withdrawn. When a stone cannot be removed by one of the expedients described above, an incision is made through the floor of the urethra on to the stone (external urethrotomy). After the stone has been extracted, in the absence of a stricture and gross infection, the urethra can be closed in layers.

NEOPLASMS OF THE MALE URETHRA

Polypi are usually multiple and occur most frequently in the region of the verumontanum. As seen at urethroscopy, each polypus is a pale, finger-like projection (fig. 1483) with blood-vessels coursing over it. Polypi are never found without chronic infection. Sometimes, if the infection can be cured, the polypi disappear: conversely, if polypi are destroyed, chronic urethritis, which previously resisted treatment, often responds to it. Possibly this condition should be classified as a granuloma.

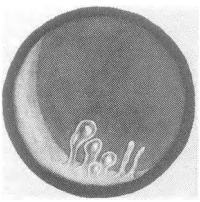

FIG. 1483.—Polypi in a male posterior urethra. They occur also in the posterior urethra of females.

Papilloma.—From time to time a solitary papilloma occurs, most often within the fossa navicularis and, as it enlarges, it protrudes from the external urinary meatus.

Multiple papillomas associated with penile papillomas (p. 1311) sometimes arise from mucous membrane just within the external urinary meatus.

Papillomatosis of the urethra is most often associated with papillomas of the bladder—the posterior urethra is considerably more often affected than the anterior urethra.

The typical symptom of papilloma of the urethra is slight hæmaturia immediately preceding micturition.

Angioma is rare. The hæmaturia to which it gives rise is often profuse, and may occur independently of micturition.

Treatment of all the foregoing neoplasms is diathermy coagulation through a urethroscope. Exceptionally the neoplasm is so accessible that it can be fulgurated without employing a urethroscope.

Carcinoma is rare. Usually a urethral stricture is antecedent to the carcinoma. Spread to the inguinal lymph nodes often occurs. Blood-borne distant metastases are uncommon unless the cavernous tissue of the penis becomes implicated.

Clinical Features.—As a rule, the first and only symptom is a profuse urethral discharge. Carcinoma of the urethra is therefore one of the many causes of ' urethritis'. Later, the discharge becomes blood-stained and the symptoms of stricture of the urethra supervene. The chronic nature of the condition in a man past forty years of age, the presence of blood in the discharge or in the urine, a tendency to bleed easily during instrumentation, and above all palpable induration, are factors that lead to a suspicion of carcinoma. In the deeper parts of the urethra, sometimes the first manifestation is an indolent periurethral abscess that after incision not only fails to heal, but the periurethal induration increases.

Biopsy then establishes the diagnosis.

Treatment.—When the growth is in the anterior part of the penile urethra partial amputation of the penis can be carried out, and provided the growth has been diagnosed reasonably early, long survival can be expected. Complete amputation with the construction of a perineal meatus is required when the neoplasm is situated more posteriorly.

When the carcinoma is situated further back a still more extensive operation, which includes radical prostatectomy, is the only hope of eradicating the disease.

THE FEMALE URETHRA

Urethritis.—As in the male, acute urethritis can be due to gonorrhœa or to non-gonococcal urethritis. Likewise chronic urethritis may or may not be due to gonorrhœa, and in its severe forms is a urethro-trigonitis. The symptoms are increased frequency, pain on micturition, urgency, terminal hæmaturia, and low back pain. Urethral tenderness is an important sign of

infection of Skene's tubules. Pressure on the urethra causes pus to exude from the orifices of the ducts surrounding the urethral opening. Incomplete cure of the infection in these tubules is a source of chronicity in anterior urethritis. In cases of posterior urethritis, urethral polypi are often present and can be seen on urethroscopy.

Gonorrhœa in the Female.—Gonorrhœa affects primarily the urethra and the cervix in the female and is often symptomless. It can never be diagnosed on clinical grounds alone. Almost three quarters of all female cases attend initially as a result of the diagnosis in their male partners, not because of their signs or symptoms. These may be completely absent or consist of a slight dysuria or a slight to moderate vaginal discharge often unnoticed by the patient. The signs may include a bead of pus at the meatal orifice after emptying Skene's tubules by milking the urethra down against the posterior ramus of the pubis; some reddening or erosion of the cervix with a mucopurulent cervical plug. A copious vaginal discharge is more likely to be due to concomitant trichomonal vaginitis, except in the young prepubertal girl where a primary gonococcal vaginitis may occur.

Investigations.—Gram-stained smears of urethral and cervical secretions. Cultures are essential. Wet film and culture of the vaginal secretion for *T. vaginalis*. These investigations must be repeated three times with negative results to exclude the diagnosis or as tests of cure after treatment.

Complications.—Gonococcal proctitis occurs in about 40 per cent of cases, often symptomless, it is spread from the vaginal discharge. *Salpingitis.*—Ten per cent of cases will present evidence of involvement of one or both tubes, acute or subacute. Lower abdominal pain and tenderness is present, with thickening and tenderness of the affected tube on bimanual palpation. Infertility due to bilateral gonococcal salpingitis is now rare. *Bartholinitis*, leading to an acutely tender Bartholin's abscess. *Systemic complications and treatment* as in the male (p. 1294).

Gonococcal Ophthalmia Neonatorum.—Infection of the eye during birth was an important cause of blindness, now fortunately rare. Unless rigorously treated the condition may progress rapidly disrupting the orbit. If unilateral, the unaffected eye should be covered, and the affected eye irrigated with normal saline as frequently as necessary to prevent accumulation of pus, followed by penicillin or chloramphenicol eye drops and I.M. penicillin. Smears and cultures for the gonococcus must always be taken before commencing treatment.

Senile urethritis is an atrophic lesion due to deficiency in endogenous ovarian hormone. Suppositories containing diethylstilbœstrol 0·1 mg. in a topical anæsthetic agent are extremely effective.

Prolapse of the Urethra.—Prolapse of the posterior margin of the urethra occurs in many women past the menopause, and is symptomless unless it is associated with senile urethritis when, on account of straining on micturition, the condition is progressive. Prolapse of the urethral mucous membrane occurs also as a congenital condition. When half or more of the circumference prolapses the local discomfort, especially on walking, is proportionate to the degree of the prolapse. The urinary meatus is examined while the patient strains: when the prolapse is complete the opening of the urethra is central; when it is partial, the opening is eccentric.

Treatment.—The associated urethritis, if present, must be treated first. Often lesser degrees of prolapse can be cured by making one or more linear grooves in the long axis of the mucous membrane of the urethra with a diathermy needle, employing the coagulating current. Subsequent contraction of the scar reduces or obliterates the

Alexander Johnston Chalmers Skene, 1838–1900. Professor of Gynæcology, Long Island College Hospital, New York.
Caspar Bartholin, 1655–1738. Professor of Medicine, Anatomy and Physics, Copenhagen.

prolapse. More advanced cases are treated by transfixing the whole thickness of the prolapse as far from its extremity as possible with four catgut sutures placed at equidistant intervals. Redundant urethral mucous membrane is excised distal to the sutures, which prevent retraction of the stump into the canal. The cut edges of the mucous membrane are then united.

Urethral Stricture.—An adult female urethra which fails to admit freely a No. 20 French bougie is the seat of a urethral stricture.

The causes are (*a*) urethritis, not necessarily gonococcal, and (*b*) trauma as the result of difficult labour. Inflammatory stricture is situated at the external meatus; traumatic stricture usually affects the middle or posterior part of the urethra.

Sometimes the stricture will only admit a guide, and in these cases acute retention of urine is particularly prone to occur. Dilatation of the stricture yields satisfactory results. The recognition and treatment of a stricture often clears up an obscure case of pain on micturition.

Urethral diverticulum (*syn.* urethrocele) is more common in women than in men. Arguments favour a congenital origin in one of Gärtner's ducts[1], but some diverticula are acquired by (*a*) rupture of a distended urethral gland or (*b*) injury of the urethra during parturition. A small uninfected pouch may be symptomless. As the diverticulum enlarges, inability to pass all the urine at one time, or dribbling after micturition, occurs. Once infected—and infection is almost inevitable if the diverticulum is not evacuated completely at each micturition—recurrent attacks of cystitis perpetuated by reinfection from the diverticulum continue in spite of antibiotics and chemotherapy. On digital examination a swelling can be felt on the anterior vaginal wall in the line of the urethra, and when it is compressed, urine, usually obviously purulent, is expressed. If the beak of a curved metal bougie is passed gently along the floor of the urethra, from time to time the orifice of the diverticulum is large enough for the beak to enter the sac. In 10% of cases a stone forms in the diverticulum, and hæmaturia may then occur.

Treatment.—The simplest method is to open the diverticulum through an incision in the vaginal wall. After irrigating the interior of the diverticulum, the mucous membrane is curetted, and the cavity is packed lightly with Oxycel. The incision in the diverticulum and vaginal wall is then closed by interrupted catgut sutures. An indwelling catheter remains in place for a week. It is presumed that fibrosis obliterates the sac (Lane). This procedure is so often successful that formal excision is seldom required, and is reserved for diverticula with wide mouths.

Calculus impacted in the female urethra is exceptional. When it occurs it can be removed by grasping it with forceps while a finger on the anterior vaginal wall presses it forward.

Urethral caruncle is common in middle-aged and elderly women. The condition presents as a soft, raspberry-like, pedunculated, granulomatous mass about the size of a pea, attached to the posterior urethral wall near the external urinary meatus. Histologically it is composed of highly vascular connective tissue stroma infiltrated with polymorphonuclear leucocytes and covered by squamous epithelium.

Clinical Features.—There is increased frequency of micturition, and often great pain during and after micturition. Terminal hæmaturia often occurs, and there may be a blood-stained discharge independent of micturition. The condition can be diagnosed on inspection, although it must be differentiated from prolapse of the mucous membrane. With a probe it can be determined that the protrusion arises from a pedicle attached to the posterior urethral wall. The mass is exquisitely tender and bleeds readily.

Treatment.—The pedicle should be divided flush with the floor of the urethra with a diathermy needle, using a cutting current, after which that portion of the urethra from which the pedicle arose is coagulated with the diathermy current. The chronic urethritis with which the condition is always associated should be treated by Furacin suppositories and intermittent urethral dilatation until the patient is symptom-free.

Papillomas acuminata occur on the external urinary meatus, and spread on to the labia minora. They differ in no respect from, are acquired in the same way as, and the treatment is similar to that of, papillomas acuminata of the penis (p. 1311). In female Africans papillomas acuminata are common, and they increase at an alarming

[1] The unobliterated distal end of the mesonephric duct.

Hermann Treschow Gärtner, 1785–1827. Surgeon and Anatomist, Copenhagen.
Daniel Francis Victor Lane, Contemporary. Surgeon, Meath Hospital, Dublin.

rate during pregnancy. Towards term, so great does the neoplastic mass become that it may obstruct labour and necessitate Cæsarean section (Bowesman).

Carcinoma of the urethra occurs twice as frequently in the female as in the male. Whether or not a caruncle can become malignant is disputed, but a visible bleeding protrusion is the commonest manifestation of a carcinoma of the urethra, and the most common site of the carcinoma in its early stages is the external urinary meatus. A carcinoma commencing within the urethra gives rise to dysuria, hæmaturia, and sometimes retention of urine. Usually induration distinguishes it from an innocent tumour.

Treatment.—Interstitial irradiation by gold grains or radium needles is the method of choice. If this fails to cure the condition, the advanced state of the disease necessitates total urethro-cystectomy.

THE PENIS

PHIMOSIS

While the condition can be acquired as a result of chronic or acute inflammation of the lining of the prepuce, usually it is due to congenital narrowing of the preputial orifice, often associated with an unduly long foreskin. In extreme examples of congenital phimosis, when the patient micturates the prepuce balloons out first, and a thin, weak stream of urine follows. Difficulty of micturition, with residual urine, hydroureters, and hydronephroses are rarely due to phimosis, but more often occur as a result of atresia meati which may lie hidden by the phimosis. The treatment is circumcision.

Circumcision[1]

Indications.—(*a*) *In infants and young boys*, because of a request by the parents (religious and personal); because of recurrent balanitis with inability to retract the prepuce; and, rarely, because of a very long prepuce. Except for the ritual operation most circumcisions are unnecessary: it is normal for the prepuce to be long and adherent to the glans within, for these parts become satisfactorily separated and the prepuce mobile in the first few years of life. Recurrent balanoposthitis and phimosis often follow attempts by the parents forcibly to retract the prepuce. (*b*) *In adults*, because of inability to retract for intercourse, a tight frenum, balanitis, and sometimes prior to radiotherapy for carcinoma a dorsal slit may suffice.

Technique in an Infant.—The parts are washed with soap and water, and painted with a non-irritating antiseptic. If it is not possible to retract the prepuce, the preputial orifice should be stretched with a hæmostat in order that the post-coronal sulcus can be cleansed of contained smegma. A pair of sinus forceps or, better still, bone forceps, is applied obliquely to the prepuce, parallel to the corona, just beyond the tip of the penis (fig. 1484), and the skin distal to the forceps is excised with scissors close to the forceps, which are then removed. The outer layer of the preputial skin retracts, leaving the glans covered by the inner layer, which is divided in the middle line anteriorly by introducing a blade of delicate scissors between it and the glans. Each leaf of the deep

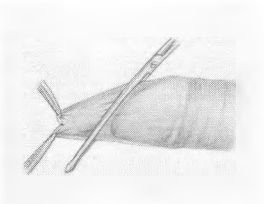

FIG. 1484.—Applying sinus forceps preparatory to excising the redundant skin.

[1] Apparently circumcision did not originate among the Jews: they took the custom from either the Babylonians or the Negroes, probably the latter. It has been practised in West Africa for over 5,000 years.

Charles Bowesman, Contemporary. Formerly Surgical Specialist, Colonial Medical Service, Kumasi, Ghana.

layer of skin (formerly called the mucosa) is excised, leaving a narrow collar around the corona. The frænal artery usually requires ligation (fig. 1485), as well as several small vessels on the dorsum. The cut edges of the deep and superficial layers are approximated with fine catgut sutures. A wisp of cottonwool soaked in tinct. benzoin co. makes an excellent waterproof dressing, which becomes detached when the patient is sat in a bath six days later.

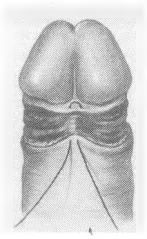

FIG. 1485.—The 'four-in-one' frænal stitch.

In adolescents and adults the following method is preferable. The prepuce is retracted until its tense orifice is apparent, or until the tip of the glans comes into view, and on to the edge of the prepuce are placed three hæmostats, one in the middle line ventrally and two on either side of the middle line dorsally. The prepuce is then slit up in the middle line dorsally to within 1·25 cm. of the corona. The under-surface of the prepuce having been completely separated from the glans and the corona, the layers of each flap are excised, keeping 1·25 cm. distal to the corona. The superficial layer is retracted and bleeding-points are secured and ligated. The inner layer of the prepuce having been trimmed to 3 mm. from the corona, the two cut edges are approximated accurately with fine interrupted catgut stitches (fig. 1486). The cut edges in the immediate vicinity of the frænum can be drawn together neatly by a mattress suture. It is best dressed by wrapping a thin layer of wool round and painting this with Tinct. Benz. Co. (Friar's Balsam).

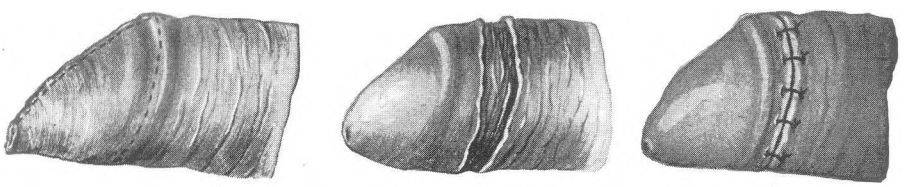

FIG. 1486.—Method of circumcision in an adult.

PREPUTIAL CALCULI

Late in life, chronic posthitis (p. 1309) resulting in fibrosis constricts an already inadequate orifice of a preputial sac. When the prepuce has not been retracted nor its interior cleansed for many years, preputial calculi are wont to form. Three types are described: 1. Those resulting from inspissated smegma. 2. Those consisting of a mixture of smegma and urinary salts. 3. Those consisting entirely of urinary salts. The treatment is circumcision.

PARAPHIMOSIS

The tight prepuce has been retracted but cannot be returned, and it is constricting the glans which is engorged and œdematous. The diagnosis is apparent at a glance.

Treatment.—One ml. of normal saline containing 150 turbidity units of hyaluronidase is injected into each lateral aspect of the swollen ring of prepuce. Fifteen minutes later the swelling is much reduced, and in early cases reduction (fig. 1487) can be accomplished with ease. If this is unsuccessful, a general anæsthetic must be given and the constricting band is incised, and the narrow cuff of skin which formed the constricting band excised (fig. 1488). There is now no obstruction to reduction, and it remains only to remove a broad cuff of normal

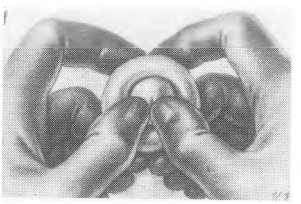

FIG. 1487.—Reducing a paraphimosis.

skin on the proximal side of the gap and unite the cut free edges, thus performing circumcision.

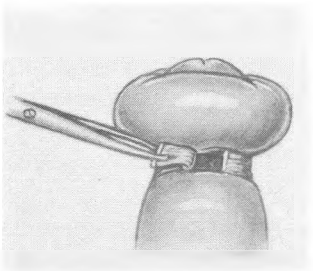

FIG. 1488.—Excising the constricting band.

INJURIES

Avulsion of the Skin of the Penis.—Entanglement of clothing in rotating machinery accounts for the majority of these injuries. Repair can be carried out by burying the shaft of the penis in the scrotum (fig. 1489), with subsequent surgical release at a propitious time.

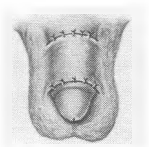

FIG. 1489.—Covering the denuded shaft of the penis by burying it in a scrotal tunnel.

Fracture of the penis is a very uncommon accident due, most often, to the erect organ being bent forcibly downwards. Following trauma, the erect organ suddenly becomes flaccid. The extravasation of blood, which is considerable, causes great pain and swelling. In early cases incision, clearing out blood-clot, and suture of the ruptured corpus cavernosum has yielded good results.

STRANGULATION OF THE PENIS BY RINGS

Removal is prevented by venous engorgement. Consequently aspiration of the corpora cavernosa may assist in removal of the ring in early cases, otherwise a ring cutter or a hacksaw must be employed. If a tight ring is not removed within six hours, unless the bladder is drained suprapubically, or perineal urethrostomy performed, rupture of the urethra with extravasation may ensue.

INFLAMMATIONS

Balanoposthitis.—An inflammation of the prepuce is known as posthitis; an inflammation of the glans penis is called balanitis. Frequently the opposing surfaces of the prepuce and the glans are implicated in the inflammatory process—hence the term balanoposthitis.

N.B.—It is imperative to bear in mind that cancer, chancre, or sugar in the urine may be the primary factor, and that the condition is nearly always associated with some degree of phimosis (fig. 41). In mild cases the only symptom is itching and some discharge. Acute cases show a red, swollen and tender foreskin with exuding pus. Monilial (candida) infections are quite common.

Treatment.—A dorsal slit of the prepuce is often essential to promote drainage, but it should be delayed twelve hours while a broad-spectrum antibiotic is given. Local cleaning can then be effective. Later a formal circumcision may be necessary.

HARD CHANCRE (*syn.* HUNTERIAN CHANCRE)

Hunterian chancre is described on p. 45, and soft chancre on p. 49.

GENITAL HERPES

The commonest of genital ulcerative lesions, it is caused by herpes virus hominis type 2. Recurrent attacks are common. Lesions occur anywhere in the anogenital area in both male and female. Rarely intraurethral lesions cause symptoms of urethritis. In the female cervical lesions can be recognised by the characteristic changes in exfoliative cytology which should be repeated annually because of the association with carcinoma of the cervix.

Clinical Course.—After 3–6 days a group of tiny vesicles appears, preceded by irritation. The vesicles erode, forming small, rounded, shallow yellow based, tender ulcers which may coalesce forming a serpiginous outline.

Investigations.—Serum tests to exclude syphilis and viral cultures.

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London. He bequeathed 13,682 specimens, prepared and mounted by his own hands. Many were destroyed by enemy action in 1941, but the remainder now form part of the Hunterian Museum of the Royal College of Surgeons of England.

LYMPHOGRANULOMA INGUINALE (*syn.* LYMPHOGRANULOMA VENERUM)

A sexually transmitted tropical disease caused by a virus of the trachoma, psitticosis, lymphogranuloma group.

The Primary lesion is a fleeting, painless, genital papule or ulcer, often unnoticed by the patient.

The Secondary lesion.—It is at this stage, 2–6 weeks after infection, that the diagnosis is almost invariably made. In the male, and also in the female if the Primary lesion is on the anterior part of the vulva, the inguinal glands become enlarged and matted together, both above and below the inguinal ligament, causing the sign of the groove. The overlying skin becomes purple and central fluctuation of the bubo occurs. Multiple fistulæ may ensue. In the female, and in male homosexuals, the lymphatic drainage from the primary sore is more usually into the pararectal glands. The late stages are due to lymphatic blockage. Distal œdema may develop resulting in elephantiasis of the genitals. Vulval elephantiasis in the female (esthiomene) may occur and the intense pararectal inflammation and subsequent fibrosis may cause stricture of the rectum.

Confirmatory Tests.—Lymphogranuloma complement fixation test (L.G.V.C.F.T.). A titre of 1 in 32, or better still a significant rise in titre during the course of the disease, is considered positive.

Treatment.—On no account should the bubo be incised, aspiration of the fluctuant bubo is preferable together with a course of sulphonamide 1 g four times daily for 10–14 days and/or Oxytetracycline 250 mg four times daily for 10–14 days.

GRANULOMA INGUINALE (*syn.* GRANULOMA VENERUM)

A chronic, slowly progressive, ulcerative disease affecting the genitalia and surrounding tissues. A tropical disease, most often seen in negroes, the mode of transmission is uncertain but believed to be sexual. The causative organism is an intracellular parasite, *klebsiella granulomatis*. The incubation period is variable, average 10–14 days.

Clinical Course.—A painless vesicle or indurated papule usually situated on the penile shaft, labia, perineal region and rarely on the vaginal wall or cervix. This becomes eroded to form an ulcer with a beefy red, granulomatous base which extends peripherally and subcutaneously. The ulcerated area bleeds readily but is surprisingly painless. Complete healing rarely takes place without thorough treatment, although partial healing may occur leaving keloid scars.

Confirmatory Tests.—Material taken from active edge of the lesion will show 'Donovan' bodies—short gram-negative rods within the cytoplasm of the large mononuclear cells.

Culture on the yolk sac of chick embryos is possible but difficult.

Treatment.—Oxytetracycline 250 mg four times daily or 500 mg thrice daily, repeated if necessary.

CHORDEE[1]

Chordee is a fixed bending or bowing of the penis due to (*a*) hypospadias or (*b*) acutely as a result of general urethritis. Erection is grotesque and very painful. The treatment is stilbœstrol 6 mg. daily.

INDURATIO-PENIS-PLASTICA (*syn.* PEYRONIE'S DISEASE)

Usually the patient is over forty years of age and seldom is there a history of venereal disease. At the onset pain and curving of the penis on erection cause the patient to seek advice, but after a few weeks the pain disappears. On palpation an

[1] *Cordee* (Fr.) = corded.

Charles Donovan, 1863–1951. Lieutenant-Colonel, Indian Medical Service.
François de la Peyronie, 1678–1747. Surgeon to Louis XIV and Founder of the Royal Academy of Surgery, Paris.

indurated plaque can be felt on the dorsal surface of one corpus cavernosum. The condition is slowly progressive and sometimes extends across the middle line. Its ætiology is unknown ; bygone trauma is a possible explanation.

Treatment has remained an unsolved problem for over 200 years. In some cases there is a tendency to slow resolution over three to five years.

PERSISTENT PRIAPISM

The penis remains erect, and is painful. The glans and the corpus spongiosum are not involved. Most often the erection is due to idiopathic thrombosis occurring in the prostatic venous plexus. Less frequently it is associated with leukæmia or sickle-celled anæmia. Secondary malignant deposits in the corpora cavernosa, or in the pelvis, account for about 7 per cent of cases. In another completely different category are cases due to spinal injury or disease.

Diagnosis.—A low spinal anæsthetic will cause priapism of neurogenic origin to abate temporarily. A blood-count is essential to exclude leukæmia.

Treatment.—Local applications are useless. Anastomosis of the saphenous vein to one corpus cavernosum can be very effective if done within the first 48 hours. The corpora should be washed out with saline. Alternatively, the corpora cavernosa can be anastomosed to the corpus spongiosum. The use of heparin and streptokinase in late cases has not given very good results.

NEOPLASMS OF THE PENIS

Papillomas Acuminata[1] (*syn.* venereal warts), the commonest benign growths of the penis, occur both in the uncircumcised and the circumcised. Most often they are situated in the coronal sulcus (fig. 1490), but in the male they can occur anywhere on the penis, urethra, scrotum, perineum, and the anal region. Usually they are moist, and attended by an offensive serous discharge. Often they are transmitted by sexual contact and are possibly due to a virus infection.

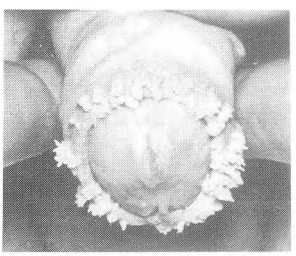

FIG. 1490.—Penile warts.

Treatment:

(*a*) **Fulguration** of the growths by diathermy.

(*b*) **By Podophyllin.**—The parts are cleansed thoroughly and then unaffected skin should be protected by smearing with a zinc oxide ointment. A 10 or 20 per cent. solution of podophyllin in tinct. benzoin is applied with a sterile applicator, and the prepuce is held back for three to five minutes until the tincture dries. The patient is instructed to wash the part thoroughly in thirty minutes. Thereafter he must pay particular attention to cleanliness. The papillomas gradually disappear. Often such treatment is followed by considerable local reaction, and is painful. For this reason fulguration is advisable, especially if it is necessary to circumcise the patient.

(*c*) **Acetic Acid.**

CARCINOMA OF THE PENIS

Ætiology.—For a reason that is only partially understood, circumcision correctly performed soon after birth confers almost total immunity against carcinoma of the penis. On the other hand (and this is difficult of explanation) circumcision after early infancy does not provide the same degree of protection. Mohammedans, who are circumcised between the ages of four and nine years, exemplify this. For practical purposes, then, carcinoma of the penis occurs only in men who have not been circumcised in early infancy, and undoubtedly the development of this neoplasm is favoured by chronic balanoposthitis. Furthermore, there are definite pre-carcinomatous states, viz.:

[1] *Acuminatus* (Lat.) = sharp pointed.

(*a*) **Leukoplakia of the glans** is exactly comparable to the well-known condition of the tongue.

(*b*) Long-standing **penile papillomas** (p. 1311).

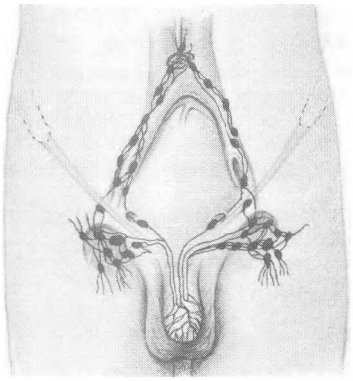

FIG. 1491.—The lymphatic drainage of the penis. The superficial lymphatic vessels drain into the inguinal while the deep lymphatics drain into the iliac lymph nodes. (*After Archie L. Dean.*)

(*c*) **Paget's Disease of the Penis** (*syn. Erythroplasia of Querat*).—' I have seen a persistent rawness of the glans like a long-standing balanitis followed by cancer of the substance of the penis ' (Sir James Paget). It is possible that penile Paget's disease escapes detection until carcinoma has developed. Treatment is by radium or diathermy excision.

Pathology.—There are two types of squamous carcinoma of the penis—the flat or infiltrating, and the papilliferous. The former may be associated with leukoplakia; the latter commences in a papilloma of long standing. The growth remains purely local for months. The earliest spread is to the inguinal, and then to the iliac lymph nodes. Direct spread to the body of the penis is prevented for many months by the fascial sheath of the corpora cavernosa, but once this barrier becomes broken the growth extends more rapidly and the iliac lymph nodes (fig. 1491) become involved. Distant metastatic deposits are infrequent.

Clinical Features.—It is a mistake to believe that this is a disease confined to the elderly—40 per cent. of the sufferers are under forty years of age. The progress of the disease is slow; the first symptoms are a mild irritation and a purulent discharge from the prepuce. These symptoms are often neglected (fig. 1492), and by the time the patient reports, sometimes more than a year after symptoms have appeared, there is a blood-stained, foul discharge. Pain is singularly absent. The inguinal lymph nodes are enlarged in over 60 per cent. of cases, but in only half of these is the enlargement due to secondary deposits, the remainder being due to sepsis. In most cases the prepuce cannot be retracted, and in order to view the lesion the prepuce must be slit. In all cases a biopsy should be performed.

Untreated, the whole glans becomes a fungating and particularly foul-smelling mass. Later, the inguinal lymph nodes fungate through the skin of the groin, and often death relieves the victim by torrential hæmorrhage following erosion of the femoral or external iliac artery.

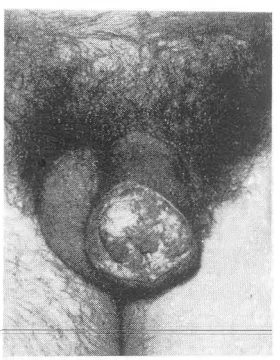

FIG. 1492.—Carcinoma of the penis.

Treatment:

Radiotherapy gives good results (60 to 70 per cent. five-year survival) with small well-differentiated tumours. **Surgery** is needed for large anaplastic growths; if there is infiltration of the shaft of the penis; when radiotherapy has failed; in elderly men who do not mind the mutilation as

Sir James Paget, 1814–1899. Surgeon, St. Bartholomew's Hospital, London.

much as the pain to be expected from the extensive reaction to radiotherapy. *Partial amputation* is used for distal growths providing there is at least 2 cm. of the dependant shaft which is not involved. With an advanced, infiltrating, or anaplastic lesion *total amputation* must be performed.

Radiotherapy methods are: implantation with flexible radio-active tantalum wires (total dose 6,000 rad in five to seven days); medium or high-voltage X-rays (5,000 to 6,000 rad given in divided doses over five weeks); by radium mould applicators worn intermittently or continuously (not exceeding 6,000 rad in seven to ten days). If not already performed, a dorsal slit is a prerequisite for treatment and nursing.

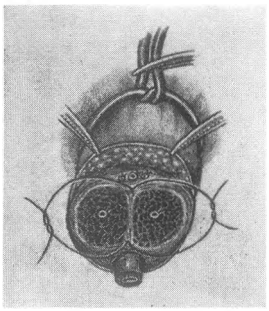

FIG. 1493.—Partial amputation of the penis.

Partial Amputation Technique (guillotine amputation).—A tourniquet, in the form of a No. 6 French rubber catheter, is placed around the most proximal portion of the penis, being tied once, and the knot clipped in a hæmostat. An abdominal pack is placed over the glans penis and with a piece of strong silk is tied tightly to the shaft of the penis at the proposed level of section. A guillotine amputation is then performed, and the pack, with its contents, is dropped into a receptacle. The exposed cross-section of the penile stump is examined for extensions of the growth. If, as is usual, none is present, the dorsal vessels are ligated. The corpus spongiosum is dissected from the corpora cavernosa for 1 cm. A long straight needle carrying a double stout catgut suture is passed from the ventral to the dorsal aspect of the fibrous septum separating the two corpora cavernosa, the needle entering just distal to the attached portion of the corpus spongiosum. The needle having been removed (fig. 1494), each corpus cavernosum is ligated firmly. The free portion of the urethra and its surrounding corpus spongiosum is divided on the dorsal aspect for nearly 1 cm. The tourniquet is now removed. The urethra is carefully attached to the adjacent skin by interrupted stitches that pass through the whole thickness of the corporus spongiosum, thereby avoiding a post-operative stricture. Bleeding-points having been ligated, the remaining portions of the skin edges are approximated vertically over the cut corpora cavernosa. An indwelling catheter is employed until the dressings are dispensed with.

Complete amputation includes the removal of the corpora cavernosa from the pubic bones and division of the corpus spongiosum so as to leave at least 1·3 cm. protruding from the perineum, as a perineal urethrostomy via an incision behind the scrotum. Careful suture of the divided urethra to the skin avoids a subsequent stricture.

The Treatment of Associated Enlarged Inguinal Lymph Nodes.—It is advisable to wait for at least three weeks after the local lesion has been dealt with by one of the methods described. If the enlargement is due to inflammation, the lymph nodes will decrease in size or disappear with antibiotic treatment. When these lymph nodes remain unaltered at the end of this period, block dissection of the more affected side should be undertaken, followed by contralateral block dissection at a later date. The five-year survival rate falls to some 35 per cent. in these cases.

If the enlarged lymph nodes are massive and fixed (inoperable), X-ray therapy causes some temporary regression.

Secondary carcinoma of the penis occurs from time to time, and about a hundred cases have been reported. The primary source of the disease is usually the bladder, rectum, or prostate.

The metastasis arrives in the penis in one of three ways: (*a*) by direct spread; (*b*) by retrograde lymphatic transport; (*c*) by retrograde venous embolism via the dorsal vein of the penis. The condition must be differentiated from Peyronie's disease. As would be expected, secondary carcinoma of the penis occurs only late in the course of the disease; nevertheless, in many instances active treatment is worth while.

CHAPTER 60

THE TESTES AND THE SCROTUM

CONGENITAL ABNORMALITIES

Anterior inversion is said to be present in one in every fifty males. The epididymis lies anteriorly, and the body of the testis and the tunica vaginalis posteriorly. When the organ is diseased this anomaly may cause confusion in diagnosis.

FIG. 1494.—A. Normal. B. Inverted. C. Horizontal inversion.

Polar inversion is less frequent. In some cases when there is complete inversion the globus major lies inferiorly (fig. 1494(B)). In other cases it lies horizontally (fig. 1494(C)). Both varieties predispose to torsion (p. 1319). Complete inversion, impossible to detect clinically, is an incidental finding at operation, when it is observed that the hydatid of Morgagni is situated at the lower, instead of the upper, pole.

IMPERFECT DESCENT OF THE TESTIS

Under this heading are included two conditions :

Incomplete Descent.—The testis is arrested in some part of its path to the scrotum.

Ectopic Testis.—The testis is abnormally placed outside this path.

Development of the Testes.—The testes develop from the genital fold, which lies medial to the mesonephros (Wolffian body), and therefore in early fœtal life they lie in the cœlomic cavity behind the peritoneum and below the developing kidneys. The primitive testis is attached to the posterior abdominal wall by a mesentery, the mesorchium, which contains the testicular blood-vessels and nerves, derived from the tenth and twelfth dorsal segments respectively. About the tenth week of intrauterine life some of the transverse tubules of the mesonephros unite with the rete testis to form the vasa efferentia; others remain rudimentary. Most of the Wolffian body disappears, but the Wolffian duct becomes the epididymis and vas deferens. About this time tissue in a fold of peritoneum (plica inguinalis) develops, containing the precursor of the gubernaculum[1]; it is attached to the junction of the vas deferens and epididymis, and can be traced through the abdominal wall to end in the region of the developing phallus. The processus vaginalis appears as a dimple of peritoneum about the tenth week and precedes the testis through the layers of the abdominal wall down into the scrotum. Muscular fibres develop in the mesoderm of the plica inguinalis to form the gubernaculum but there is still no certainty as to the part this structure plays in regulating testicular descent.

Chorionic gonadatrophin from the maternal circulation stimulates the growth of the testis and may play some part in the migration of the organ. Undoubtedly imperfect development of a testis is a significant factor in incomplete descent of the organ.

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[1] Gubernaculum = a rudder. It was first described by John Hunter, who believed that its function was to guide the testis into the scrotum.

Giovanni Battista Morgagni, 1682–1771. Professor of Anatomy, Padua, Italy. He held the Chair for fifty-six years, and is regarded as the founder of Pathological Anatomy.

Kaspar Friedrich Wolff, 1733–1794. Professor of Anatomy and Physiology, St. Petersburg (now Leningrad), U.S.S.R.

John Hunter, 1728–1793. Surgeon, St. George's Hospital, London.

Morphology.—Most of the misplacements of the testes in man are a counterpart of the varying normal placements of the testes in animals. In the whale and the elephant the testes remain in the undescended abdominal position throughout life. Rodents and hibernating animals, such as the hedgehog, the mole and the bat, maintain open inguinal canals, and the testes are housed in the abdomen, to descend into the scrotum only during the breeding season.

INCOMPLETELY DESCENDED TESTIS

Incidence:

In the Neonatal Period.—At birth, and for a variable number of weeks afterwards, the cremasteric reflex, which is so active in young boys, is absent. Scorer examined 2,000 newborn male infants and found that the incidence of incomplete descent on one or both sides was 4 per cent. in full-term infants and 30 per cent. in premature infants. A follow-up of all cases showed that in more than 50 per cent. the testis or testes reached the scrotum during the first month of life. More often than not incomplete descent of the testis is not detected during infancy.

In later childhood and puberty the incidence is 2 per cent. Frequently the condition still remains unrecognised unless a routine examination is made by the school medical officer. In a few cases the presence of a hernia, pain in the region, or acute torsion, in that order of frequency, directs attention to the abnormality.

In Adult Life.—It is inconceivable that a man can fail to notice the absence of one or both testes in his scrotum, yet there must be many with this abnormality who do not seek advice about it unless symptoms develop. Sometimes the condition is first discovered at the medical examination for entrance to one of the Public Services. In an examination of 10,000 recruits during World War II the incidence was found to be 0·8 per cent. In 10 per cent. of unilateral cases there is a familial history.

Pathology.—Up to the age of six years there are no microscopical differences between an incompletely descended testis and a normal testis. After that time, due, it is believed, to the higher temperature to which it is subjected, the development of the incompletely descended organ becomes progressively retarded.[1] By the time puberty has been reached the incompletely descended testis is flabby and hardly more than half the size of its intrascrotal counterpart. Histologically, the epithelial elements are grossly immature, and by the age of sixteen irreversible destructive changes have occurred in the germinal epithelium.

The *external secretory mechanism* of an incompletely descended testis functions but feebly, and often after a few months or years ceases; thus its power of spermato-genesis is negligible.[2]

The internal secretory activity of an incompletely descended testis is reduced. In bilateral cryptorchism about half the normal amount of androgen is produced: notwithstanding, the secondary sexual attributes of a cryptorchid are seldom noticeably in abeyance.

If an incompletely descended testis is brought down satisfactorily *before puberty* it often develops and functions satisfactorily.

Clinical Features.—The right testis alone is affected in 50% of cases, the left alone in 30%, while double arrested descent occurs in 20%. Other

[1] The scrotum is a thermo-regulator to the testes, and keeps them at a temperature at least 1° C lower than that of the inguinal canals.

[2] Sir Astley Cooper taught that patients with bilateral retained testes were usually sterile, whereupon one of his pupils, a cryptorchid, left the room and committed suicide. At the necropsy motile spermatozoa were demonstrated. It is therefore unsafe to deny the possibility of parentage to those with retained testes.

Charles Gordon Scorer, Contemporary. Surgeon, Hillingdon Hospital, Uxbridge, Middlesex, England.
Sir Astley Paston Cooper, 1768–1841. Surgeon, Guy's Hospital, London.

abnormalities of the urinary tract may be present. Felton found that intravenous pyelograms demonstrated major abnormalities in 13·5% of children with cryptorchism. The testis may be:

1. *Retained within the abdomen* extraperitoneally, usually just above the internal abdominal ring.

2. *In the Inguinal Canal.*—Early in life the testis is a soft structure, and when, as in this instance, it is submerged in the non-resisting floor of the inguinal canal, and shielded by the overlying tendinous aponeurosis of the external oblique, it cannot be felt.

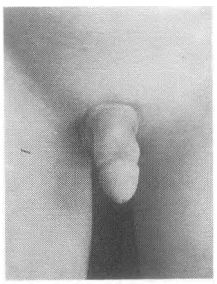


FIG. 1495.—A cryptorchid, aged 12. Note the retracted, underdeveloped scrotum. In cases of retractile testes the scrotum is comparatively well-developed.

When both testes are in the abdomen or the inguinal canals and are consequently impalpable, the condition is known as cryptorchidism (hidden testes) (fig. 1495).

3. *In the Superficial Subinguinal Pouch.*—Very frequently during childhood the testes are mobile, each being withdrawn by the contraction of the cremasters into the superficial subinguinal pouch (fig. 1496), a space lined by loose areolar tissue lying beneath Scarpa's fascia and superficial to the external oblique aponeurosis (Denis Browne), or into the inguinal canal. Reflex retraction occurs from a very slight stimulus such as touching the thigh or the abdomen, or even by exposure of the parts. Not infrequently this testicular mobility continues throughout childhood on both sides, or less frequently on one side. At all times it is liable to be mistaken for incomplete descent unless a special method of examination is undertaken, if necessary on more than one occasion. Retractile testes should be suspected if the scrotum is normal; in incomplete descent the corresponding side of the scrotum is undeveloped. This inspection completed, the pulps of two fingers are placed over the superficial inguinal pouch, exerting moderate pressure. By drawing the fingers towards the neck of the scrotum a testis of the retractile type can be pushed into the upper part of the scrotum, where it is grasped between the finger and thumb of the other hand. *Only if the testis cannot be made to touch the bottom of the scrotum*[1] *is it imperfectly descended.* When the testis *can* be placed in a normal position temporarily it is not truly an imperfectly descended testis, and it should be known by its fitting title '*retractile testis*'. Such testes require neither endocrine nor operative treatment. In course of time (it may be delayed until puberty), the testis will take up a normal position permanently. Another valuable method of encouraging the testes to drop down into the scrotum is to make the child squat, the thighs being brought up on to the abdomen.

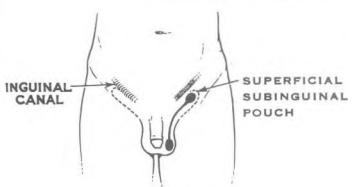


FIG. 1496.—Mechanism of retractile testis. (*After the late Sir Denis Browne, London.*)

[1] Only by applying the test carefully and correctly in every case will the extravagant claims of some of those who employ endocrine therapy be dissipated.

Lester Mahan Felton, Contemporary. Urologist, Worcester Hahnemann Hospital, Worcester, Mass., U.S.A.
Antonio Scarpa, 1747–1832. Professor of Anatomy, Pavia, Italy.
Sir Denis John Wolko Browne, 1892–1967. Surgeon, Hospital for Sick Children, Great Ormond Street, London.

In infancy 80% of non-apparent testes are retractile. In the remaining 20% true incomplete descent is present.

The Hazards of Incomplete Descent

1. *Sterility* in bilateral cases.

2. *Pain.* An inguinal testis is liable to oft-repeated trauma.

3. *An associated indirect inguinal or interstitial hernia* is present in 70% of cases, and in adolescent and adult patients it is frequently the hernia that causes the symptoms.

4. *Torsion* (p. 1319).

5. *Epididymo-orchitis.*—Right-sided epididymo-orchitis occurring in an incompletely descended organ is extremely difficult, if not impossible, to differentiate from an acute appendicitis. It is, however, very rare because the abnormal testis is less likely to become involved by descending infection.

6. *Atrophy*, even before puberty, sometimes occurs when the testis is situated in the inguinal canal. This is attributed to recurrent trauma.

7. *Increased Liability to Malignant Disease.*—All types of malignant testicular tumour have been recorded as developing in undescended testes, and in those brought down surgically. It is definite that seminoma is more common possibly because the testis is frequently histologically abnormal. Some compute that 1 in 20 abdominal and 1 in 80 inguinal testes become malignant; others say this estimation is far too high. Reliable statistical evidence as to whether orchiopexy diminishes the liability to tumour formation later, is not available. However, at the worst it should improve the prospect of early diagnosis.

Treatment by Orchiopexy.—Most surgeons favour performing the operation between the ages of six and eight years. Many choose the earlier age in order to reduce the incidence of thermal damage to the germinal epithelium. After the age of puberty the percentage of successful operations, which at the optimum age approaches 80%, falls considerably. In cryptorchidism one side should be operated upon at a time, with an interval of six months between the operations.

The operation consists of (1) mobilising the spermatic cord and the testicular vessels; (2) retaining the mobilised testis in the descended position.

1. **Mobilising the Spermatic Cord.**—The inguinal canal is opened. The cord and testis are freed from surrounding structures and a concomitant inguinal hernial sac is dealt with. The dissection of the diaphanous sac from the cord is greatly facilitated by opening the sac and injecting a small amount of normal saline just underneath that part which is adherent. Near the internal inguinal ring all the coverings of the cord are divided; every muscle fibre and fibrous band is severed, leaving only the vas and its blood-vessels and the spermatic blood-vessels. Sometimes this provides sufficient length for the testis to be placed in the scrotum. If not, the spermatic vessels are dissected from the peritoneum to which they are adherent. This abolishes the wide, outward curve of the vessels, thereby gaining up to 2·5 cm. It is most exceptional for the above measures to fail to elongate the pedicle sufficiently for the testis to be placed in the scrotum, and even beyond it: the avoidance of vascular tension is the keystone of plastic surgery. The empty half of the scrotum is stretched thoroughly with the index finger passed into it through the inguinal incision.

2. **Retaining the testis in the scrotum** is effected by one of a number of ingenious methods, of which the following are popular:

(*a*) *Narrowing the neck of the scrotum and fixation of the testis to the bottom of the scrotum*, is all that is needed in many cases. The neck of the scrotum is narrowed with catgut sutures, and the testis is anchored below by an unabsorbable suture passed through the scrotal skin, this suture being removed after one week.

(*b*) *Dartos pouch.*—The testis is retained in the scrotum by placing it in a dartos pouch just under the scrotal skin.

(*c*) *Ombrédanne's operation* is similar, but the testis is passed through a gap in the scrotal septum, which is then narrowed round the cord. This is useful when the length of cord is short as the septum can be drawn across to the external ring. The weight of the opposite testis will supply gentle traction which will gradually draw the undescended one further down (figs. 1497 and 1498).

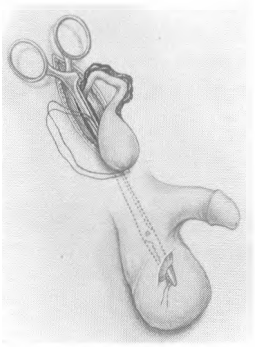


FIG. 1497.—Method of passing the suture attached to the testis through the septum dartos and the skin of the opposite side of the scrotum.

Failure to bring the testis down.—(1) Sometimes a two-stage procedure is successful: mobilisation as far as possible, and anchorage with an unabsorbable suture, followed six months later by further mobilisation. (2) *Orchiectomy*, if the other testis is normal, and the incompletely descended testis is hopelessly atrophic, or, after puberty.

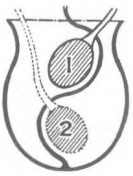


FIG. 1498.—The disposition of the testes in relation to the septum dartos following bilateral Ombrédanne's operation.

Orchio-cœlioplasty (abdominal replacement of the organ). This measure is justifiable when the contralateral organ has been removed previously and orchiopexy is found to be impossible. It is then the only method of preserving a supply of internal testicular secretion and protecting the organ from injury.

Hormone Treatment.—The only indications for hormone treatment are:

1. Cases of bilateral incomplete descent associated with hypogenitalism and obesity.
2. In lieu of waiting for retractile testes to descend, thereby avoiding jibes that small boys endure from others in the changing rooms of swimming baths, etc. (Scowen).
3. In cases of uncertainty as to whether the case is one of testes retracting into the inguinal canal or one of true incomplete descent.

Of many preparations having a gonadotrophic action, chorionic gonadotrophin extracted from the placenta (Pregnyl) is the one usually employed. It should be given at about eight years of age, and 1,000 international units are given twice a week until the testes descend, the limit of the course being ten weeks, after which it must never be repeated, otherwise secondary male characteristics may develop prematurely.

ECTOPIC TESTIS

An ectopic testis may be found, in order of frequency:

1. Anchored in the superficial inguinal pouch. 2. In the perineum. 3. At the root of the penis. 4. In the femoral triangle.

Unlike the imperfectly descended testis, an ectopic organ often develops well, if not fully. Its main hazard is that it is liable to injury.

Ætiology.—To explain the appearance of the testis in these anomalous positions, Lockwood advanced the ingenious theory of many gubernacular tails. His theory postulates that in ectopic testis the scrotal tail becomes ruptured. As a consequence the testis, adrift from its usual mooring, follows one of the accessory rudders. Lockwood stated that the accessory gubernacula depicted in fig. 1499 could be demonstrated in the fœtus.

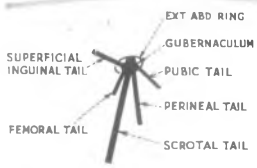


FIG. 1499.—The 'gubernacular tails' of Lockwood.

INJURIES OF THE TESTIS

Closed rupture of the testis from blows and the like is an uncommon accident owing to the extreme mobility

Louis Ombrédanne, 1871–1956. Surgeon, Hopital des Enfants Malades, Paris.
Sir Eric Frank Scowen, Contemporary. Formerly Professor of Medicine, St. Bartholomew's Hospital, London.
Charles Barrett Lockwood, 1856–1914. Surgeon, St. Bartholomew's Hospital, London.

of the organ within the scrotum. Severe contusion and rupture are each associated with a hæmatocele which surrounds the testis, rendering the differential diagnosis impossible without exploration. Operation is advisable in these cases in order to:

1. Evacuate the hæmatoma.
2. Repair the tunica albuginea, if split, excising only that part of the testicular tissue that extrudes. If the testis is severely damaged, orchiectomy should be performed.

Hæmatocele (p. 1326).

Traumatic Displacement of the Testis.—As a result of a blow the testis occasionally becomes displaced and by reason of blood-clot anchored, usually in one of the positions described under ectopic testis. Unless the displacement is reduced early by manipulation, an operation is required to replace the organ in the scrotum.

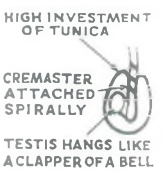
TORSION OF THE TESTIS (*syn.* TORSION OF THE SPERMATIC CORD)

Predisposing Causes.—Torsion of the testis (fig. 1500) is uncommon. It does not occur in a normal fully descended testis: its anchorage prevents rotation. Therefore one of several anomalies must be present.

(*a*) *Inversion of the Testis* (p. 1314).—This appears to be the commonest predisposing cause.

FIG. 1500.—Torsion of the testis. Note the inversion of the organ.

(*b*) *High investment of the tunica vaginalis* causes the testis to hang within the tunica like a clapper in a bell, viz.——————→



Very occasionally torsion is extravaginal.

(*c*) *In cases where the body of the testis is separated from the epididymis*, torsion of the body can occur without involving the cord. The twisting is confined to the mesentery that joins the testis to the epididymis, viz.——————→

Exciting Causes.—Normally the cremaster contracts *pari passu* with violent contraction of the abdominal musculature. Contractions of the spirally attached cremaster favour rotation around the vertical axis in relevant cases. Straining at stool, lifting a heavy weight, and coitus are all exciting causes, but often the history fails to reveal any one of them. Occasionally torsion develops during sleep.

Clinical Features.—The highest incidence is between ten and twenty-five years of age, and the second most common age period is during infancy. The symptoms vary with the degree of torsion present. Most commonly the patient experiences sudden and agonising pain in the groin and lower abdomen,[1] and vomits. Upon theoretical grounds one might think that the

[1] But, in about 23 per cent. of K. H. Smith's patients, the first symptom was a dull ache in the loin or hypogastrium. It is insufficiently appreciated that true testicular pain is situated in the lower abdomen at the level of the internal inguinal ring in accordance with Brown's law.[2]

[2] Brown's law: Pain, produced in an organ which has migrated from its primary position, and which has not acquired an additional nerve supply in its secondary or permanent position, is invariably localised in the primary relative position of that organ.

Kenneth Halsted Smith, Contemporary. Surgeon, Llandudno General Hospital, Llandudno, Caernarvon, Wales.
Francis Robert Brown, Surgeon, 1889–1967. Royal Infirmary, Dundee, Scotland.

diagnosis is simple, but sometimes it is practically impossible to distinguish *torsion of an imperfectly descended testis* from a strangulated inguinal hernia until the parts have been displayed by operation. The fact that the side of the scrotum is empty and œdematous is certainly in favour of the tender lump at the external abdominal ring being the testis with its twisted cord. *Torsion of a completely descended testis* is a less difficult problem. Sometimes the actual twists in the cord can be felt, thus establishing the diagnosis. At other times the condition can be mimicked exactly by a small, tense, strangulated inguinal hernia compressing the cord and causing congestion of the pampiniform plexus, viz. ——————————→

Torsion of the fully descended testis can also simulate closely acute epididymo-orchitis; after a lapse of six hours or so the skin of the scrotum becomes reddened (fig. 1501) and the temperature is raised slightly—37·2° C. Elevation of the scrotum usually relieves the pain in epididymitis, but increases it in torsion of the spermatic cord. Therefore in a boy (if mumps has been excluded) and in a man (if urethritis has been eliminated), a diagnosis of torsion of the testis should be *insisted* upon, and almost invariably when the testis is exposed the diagnosis will be substantiated.

FIG. 1501.—Torsion of the left testis of twenty-four hours duration in a boy aged thirteen. The acutely tender testis and the inflamed scrotum simulate acute epididymo-orchitis.

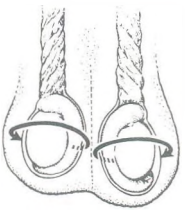


FIG. 1502. — Usually the testis *twists* in a direction away from the septum dartos, but it may twist in either direction.

Treatment.—In the first hour or so an attempt to untwist the testis may immediately relieve the pain and gentle twisting should be tried in one direction first. If this is the correct way, continue until relief is obtained. If one has chosen the wrong direction pain will increase. If untwisting can be achieved it removes the urgency, but operation and fixation should be performed as quickly as possible.

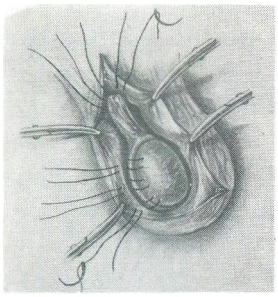


FIG. 1503.—Method of anchoring the testis with non-absorbable sutures to prevent torsion.

Urgent exploration of the scrotum is indicated if untwisting is not successful in relieving pain. If after undoing the torsion the testis is viable, then it must be fixed to prevent a recurrence (fig. 1503). The opposite testis should also be fixed at the same time, as the anatomical variation responsible is likely to be bilateral. A totally infarcted testis should be removed. Should the patient not be operated upon, but only seen days or weeks later when pain has subsided little is to be gained by exploring the affected side; the testis will slowly become woody hard and shrink to a fibrous nodule. The opposite testis must be fixed at an early date.

Torsion of an Appendage of the Testis.—Vestigial structures related to the testis and epididymis are liable to undergo axial rotation. The most common of these structures to twist is the appendix of the epididymis (the pedunculated hydatid) (fig. 1504). Torsion of an appendage of the testis is essentially a lesion occurring before or at the age of puberty. The condition is often mistaken for acute epididymo-orchitis of unknown origin and frequently, although the symptoms are comparatively mild, they cannot be distinguished with certainty from those of torsion of the testis. Immediate operation with ligation of the pedicle and amputation of the twisted appendage cures the condition.

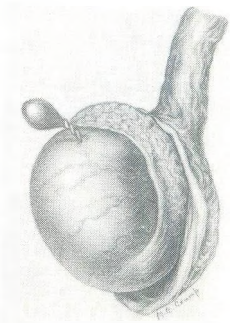


FIG. 1504.—Torsion of the appendix of the epididymis (pedunculated hydatid of Morgagni).

Idiopathic Scrotal Œdema (4–12 years) (fig. 1505) may occur in childhood and has to be differentiated from torsion of testis or appendix. The scrotum is very swollen but has not the exquisite tenderness of torsion, and pain is slight. The swelling may extend to the perineum or groin (see extravasation p. 1292) and penis. The swelling subsides after 1–2 days. It may be a recurrent phenomenon. It might also be allergic in origin—occasionally an eosinophilia is present.

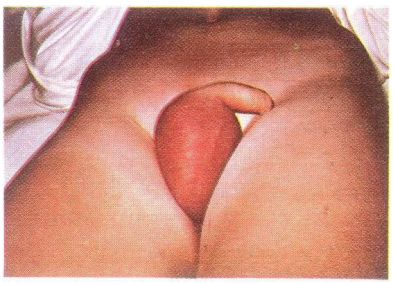


FIG. 1505.—Œdema of the scrotum.

VARICOCELE

Varicocele is a state of varicosity of the testicular veins.

Surgical Anatomy.—The veins of the testis and epididymis form an anastomosing plexus—the pampiniform plexus—which is the most bulky constituent of the spermatic cord. As the veins pass upward through the inguinal canal they become reduced in number to between four and eight. Coalescence in the neighbourhood of the internal inguinal ring brings about further reduction in their number, and by the time the posterior abdominal wall is reached, a single, or a duplicated testicular vein pursues its cephalad course behind the peritoneum, to empty into the renal vein on the left, and the inferior vena cava on the right. Only near their terminations are the testicular veins provided with valves, and not infrequently even these valves are absent, in which case valves will be found nearby in the renal veins. The cremasteric veins anastomose with the testicular veins, so providing an alternative (collateral) venous return and draining mainly into the inferior epigastric veins.

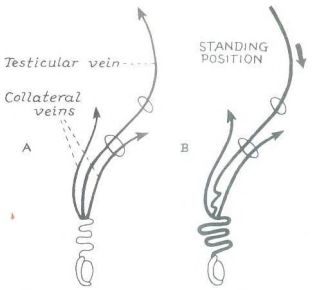


FIG. 1506.—(*a*) Normal drainage of the pampiniform plexus; (*b*) reflux along the testicular vein in varicocele. (*After J. W. Lane.*)

Ætiology.—(1) Most varicoceles are noted in the 'teens' or early adult life. The left side is affected in 95 per cent. of cases. The left testicular vein joins the renal vein and reflux into this vein can be demonstrated by angiograms. Nathan has shown that the left testicular artery arches over the left renal vein in 16 per cent. of cases and may obstruct it (fig. 1507).

(2) Hanley and Harrison, however, maintain that in many cases the dilated vessels are cremasteric veins, and not veins of the pampiniform plexus. The cremasteric veins drain into the inferior epigastric veins mainly,

Hilel Nathan (formerly Notkovich), Contemporary. Professor and Chairman, Department of Anatomy and Anthropology, Tel-Aviv University Medical School, Tel-Aviv, Israel.
Howard Granville Hanley, Contemporary. Surgeon, St. Paul's Hospital, London.
Ronald George Harrison, Contemporary. Derby Professor of Anatomy, Liverpool, England.

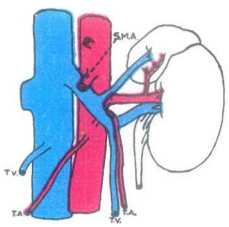


FIG. 1507.—Arching left testicular artery compressing the renal vein of the corresponding side. (*After H. Nathan.*)

but also communicate freely with the veins in the pampiniform plexus.

(3) Obstruction of the left testicular vein by growth of a hypernephroma along the left renal vein is a rare cause (0·4 per cent. Riches) of a recent varicocele arising in middle life.

Clinical Features.—Varicocele is more frequent and more troublesome in hot climates; in all parts of the world tall, thin, visceroptotic men are frequently affected, whereas short, fat individuals are seldom so. Usually symptoms are entirely absent, but in some cases the patient experiences a dragging pain in the affected side. Pain is due to the fact that the elongated scrotum no longer supports the testis, whose full weight is now borne by the cord. The scrotum, particularly the left side, hangs lower than normal (fig. 1508), and on palpation with the patient standing, the varicose plexus feels like a bag of worms. If, while lightly holding the varicocele between the fingers and thumb, the patient is instructed to bow, tension within the veins becomes appreciably less. An impulse is often felt when the patient coughs. When the patient lies down and the scrotum is elevated, the veins will be emptied by gravity and the opportunity of comparing the size of the left testis with its fellow should be taken. In long-standing cases the left testis is somewhat smaller and softer than the right, due to a minor degree of atrophy. Infertility can be caused by a varicocele.

FIG. 1508.—A large varicocele. A left inguinal hernia is also present.

Varicocele and Spermatogenesis.—The testis is supported in the scrotum by the cremaster and the dartos muscles. The main function of the cremaster is one of defence, for a fright or threat of trauma will cause reflex retraction of the testis towards the external inguinal ring, especially in the young. The dartos bears the weight of the testis and is reflexly controlled by temperature changes and acts as a kind of thermostat for efficient spermatogenesis at some 2·5° C. lower than rectal temperature. On exposure to cold it raises the testis to the body so conserving heat, while a too warm environment causes relaxation of the dartos. Hanley and Harrison, who have reawakened interest in the effects and treatment of varicocele, explain the characteristic elongation of the scrotum in this condition on the grounds that venous engorgement in the varicosities reduces the temperature differential (scrotum/rectum) to about 0·1° C. which may seriously depress spermatogenesis. The dartos relaxes in a vain attempt to maintain the temperature differential. It also follows that the modern tendency to wear tight and poorly ventilated pants and scrotal supports is to be deprecated.

Sir Eric William Riches, Contemporary. Consulting Surgeon, Middlesex Hospital, London.

Treatment.—Operation for varicocele is not indicated unless there is (1): pain; (2) serious depression of spermatogenesis (oligospermia). The simplest procedure of ligating the testicular vein above the inguinal ligament is the best. Only occasionally, if this leaves a group of tortuous varicosities, is local excision necessary.

As a result of this operation the testis becomes cooler, and the dartos contracts, supports the testis, and relieves pain, while cases of oligospermia show a 70 to 80 per cent. improvement with a conception rate of about 30 per cent.

HYDROCELE

Congenital (fig. 1509).
Acquired: Primary, or idiopathic.
Acquired: Secondary, due to disease of testis: Acute. Chronic.

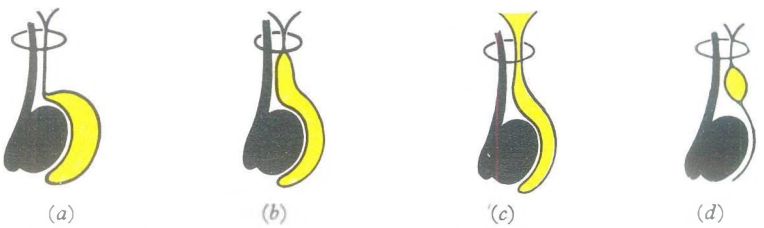


FIG. 1509.—(*a*) Vaginal hydrocele (very common); (*b*) 'infantile' hydrocele (unusual); (*c*) congenital hydrocele; (*d*) encysted hydrocele of the cord.

A hydrocele is a collection of serous fluid in some part of the processus vaginalis, usually in the tunica. Four anatomical varieties of congenital hydrocele are encountered.

Ætiology.—A hydrocele can be produced in four ways:

1. By excessive production of fluid within the sac, e.g. secondary hydrocele.
2. By defective absorption of hydrocele fluid by the tunica vaginalis.

Defective absorption appears to be the most common cause of the common variety of primary hydrocele, but the reason for the defective absorption is obscure. Damage to the endothelial wall by low-grade infection seems to be the probable explanation.

3. By interference with the drainage of fluid by the lymphatic vessels of the cord.
4. By connection with the peritoneal cavity, as in the congenital variety.

Hydrocele fluid is amber coloured, and registers a specific gravity of 1·022 to 1·024. It contains water, inorganic salts, 6 per cent. of albumin, and a quantity of fibrinogen. The last constituent confers upon the fluid a characteristic feature. If the contents of a hydrocele are allowed to run through a cannula into a receptacle, the fluid does *not* clot, but if a few drops of blood which have come into contact with cut tissues are stirred into even a large quantity of hydrocele fluid, the whole clots firmly. In old-standing cases the fluid sometimes contains so much cholesterol that it is irridescent, and occasionally tyrosine crystals are found.

DIAGNOSTIC RULES FOR ALL HYDROCELES

Ninety-nine out of every 100 hydroceles are translucent. On examination it is possible 'to get above the swelling'.

(*a*) **Primary vaginal hydrocele** most often appears in middle-aged or

elderly men, but it is not uncommon in early childhood (fig. 1510). The condition is particularly common in tropical countries. The only complaint of the patient is the swelling, and occasionally he does not seek relief until the sac has attained enormous dimensions.

FIG. 1510.—Right vaginal hydrocele in an infant. It is possible to get above the swelling which is brilliantly translucent.

About 5 per cent. of inguinal herniæ are associated with a vaginal hydrocele of the same side.[1] Often a large hydrocele obscures a small inguinal hernia, even if the latter has been especially looked for.

(*b*) **Infantile hydrocele** does not necessarily appear in infants. The tunica and the processus vaginalis are distended up to the internal abdominal ring, but the sac has no connection with the general peritoneal cavity.

(*c*) **Congenital Hydrocele.** — The processus vaginalis communicates with the peritoneal cavity. Usually the communicating orifice is too small for the development of a hernia. When the patient is horizontal, e.g. at night, the fluid in the pouch disappears within the abdominal cavity, but it returns when the erect posture is resumed. The hydrocele cannot be emptied by digital pressure, as this results in an 'inverted ink bottle' effect at the internal ring. Especially in bilateral cases, ascites or ascitic tuberculous peritonitis should be suspected.

(*d*) **Encysted Hydrocele of the Cord.**—There is a smooth, oval swelling associated with the spermatic cord, which is liable to be mistaken for an irreducible inguinal hernia. If, with gentle traction upon the testis, the swelling moves downwards and becomes less mobile, the diagnosis of hydrocele of the cord is confirmed.

Hydrocele of the canal of Nuck is a condition comparable to the foregoing. It occurs in females, the cyst being in relationship to the round ligament. Unlike a hydrocele of the cord, a hydrocele of the canal of Nuck is always wholly, or partially, in the inguinal canal.

Hydrocele en Bisac.—A rare condition where the hydrocele has two intercommunicating sacs, one above and one below the neck of the scrotum. The upper lies superficial to or partly within the inguinal canal and it may insinuate itself in between the muscle layers.

Complications of a Hydrocele

1. *Rupture* is usually traumatic, but may possibly be spontaneous. On rare occasions a cure results after absorption of the fluid.
2. *Hernia of the hydrocele sac* sometimes occurs in old-standing cases. Tension of fluid within the tunica causes herniation through the dartos muscle (fig. 1511).
3. *Transformation into a hæmatocele* occurs both spontaneously and as the result of trauma (p. 1326).
4. *Calcification* of the sac wall sometimes occurs in long-standing cases.

[1] Edward Gibbon, 1737–1794, the English historian, who is best known for his 'History of the Decline and Fall of the Roman Empire', was greatly embarrassed by a large hydrocele. The second time this was tapped the hydrocele became infected, and Gibbon died a few days after the operation. The hydrocele was associated with a large scrotal hernia which, probably, was punctured.

Anton Nuck, 1650–1692. Professor of Anatomy and Medicine, Leiden, Netherlands.

Treatment of a Hydrocele.—*Lord's Operation.*—This procedure has replaced subtotal excision when the tunica is not thickened. No tissue dissection is performed, no reactionary œdema occurs, bleeding is minimised and recurrence is unlikely as the tunica cannot surround the testis again.

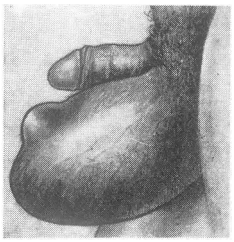


FIG. 1511.—A hernia of a hydrocele.

A small incision in between the lines of scrotal blood vessels is made through all layers, including the tunica, well away from the testis. The testis is allowed to prolapse through the wound so that the tunica is totally everted.

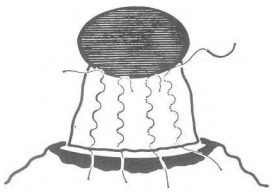


FIG. 1512.—Lord's operation. *(Peter Lord, F.R.C.S., High Wycombe, England and Karger Verlag, Basel, Switzerland.)*

A series of 10–12 catgut or Dexon sutures are woven radially from the cut edge of the tunica to the reflection of the tunica from the testis and epididymis. When these are tied the whole tunica is bunched into a 'ruff' at the edge of the testis (fig. 1512).

Sharma and Jhawer's minimal dissection technique involves the minimal incision consistent with the delivery of the testis through the opening in the sac (no 'ruffing' with sutures), and they lodge the testis in a pocket created by blunt and finger dissection between the fascial layers of the scrotum.

Subtotal excision necessitates a larger incision, complete delivery of the hydrocele through the wound, excision of the major part of the tunica and the most scrupulous attention to hæmostasis.

Tapping (fig. 1513).—After transillumination, the swelling is made tense by manual compression. A fine trocar and cannula is inserted into an unquestionably translucent area and the fluid evacuated. After a varying interval of weeks or months the sac usually refills. Many patients are content to be relieved at regular intervals in this way. Repeated tapping is liable to be followed by a little oozing into the sac. Deposits of blood on the walls of the sac in time increase its thickness, and so diminish its translucency. After tapping, the testis should always be examined.

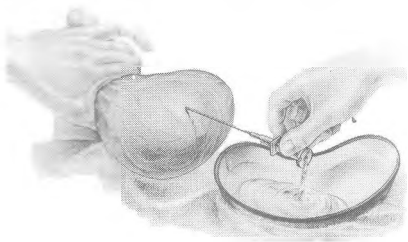


FIG. 1513.—Tapping a hydrocele.

Secondary hydrocele is an effusion into the tunica vaginalis that accompanies certain affections of the testis. It is frequently associated with acute and chronic epididymo-orchitis. It is nearly always present in the syphilitic affections of the testis, and occasionally complicates malignant disease of the organ. A secondary hydrocele rarely attains a large size, and in most instances is lax and does not interfere unduly with the palpation of the testis and its epididymis. Occasionally the question as to whether a hydrocele is or is not secondary to some underlying disease of the testis can be settled only after the contents of the hydrocele have been aspirated.

In the case of acute epididymo-orchitis usually a secondary hydrocele subsides *pari passu* with the primary lesion.

Peter Herent Lord, Contemporary. Surgeon, High Wycombe General Hospital, High Wycombe, Bucks, England.
Laxman Sharma, Contemporary. Surgeon, Medical College, Indore, M.P., India.
Pramod Jhawer, Contemporary. Surgeon, Bombay Hospital, India.

Post-herniorrhaphy hydrocele is a secondary hydrocele that appears after an operation for inguinal hernia in 0·2 per cent. of cases in which there was no evidence of a hydrocele pre-operatively. Possibly this is due to damage of the lymphatic vessels of the tunica vaginalis, which pass with those of the testis, along the spermatic cord, mostly superficial to the blood-vessels.

Hydrocele of a Hernial Sac.—The neck of a hernial sac becomes plugged with omentum or occluded by adhesions, and a hydrocele results.

Filarial Hydroceles and Chyloceles.—As one proceeds from temperate climates towards the Equator, the incidence of vaginal hydrocele increases. Filariasis (p. 49) accounts for 80 per cent. of hydroceles in some tropical countries. In most instances neither are microfilariæ discovered in nocturnal samples of blood, nor is a history of filariasis elicited. Usually filarial hydroceles follow repeated attacks of filarial epididymitis, and develop rapidly or gradually and can be large or small: frequently they are bilateral. In early cases the hydrocele fluid is similar to that found in the idiopathic variety. In cases of some standing, if the fluid is placed in a tall glass, after a few hours a film of liquid fat (chyle) will be floating on the surface. This is rich in cholesterol, and is derived from rupture of a lymph varix into the tunica. The presence of chyle is proof-positive of the filarial origin of a hydrocele. Adult worms of the *Wuchereria bancrofti* have been found in the epididymis removed by operation, or at necropsy on these cases. In long-standing chyloceles dense adhesions form between the scrotum and its contents. Filarial elephantiasis of the scrotum (p. 1337) supervenes in a small proportion of cases.

Treatment.—Acute cases should be treated by rest and aspiration. The more usual chronic cases can be cured by excision of the sac.

Hæmatocele can be either recent, or present as the clinical entity ' old clotted hæmatocele ' (see below).

Recent hæmatocele is usually the result of injury of a small blood-vessel during tapping or aspiration of a hydrocele. Prompt refilling of the sac with considerable pain and tenderness, and poor or absent transillumination, leave no doubt as to the diagnosis. The treatment should be urgent operation, with evacuation of blood and excision or eversion of the sac. If this course is not followed a clotted hæmatocele will almost certainly result.

Acute hæmorrhage into a previously normal tunica vaginalis can result from a blow on the testis. The treatment should be similar to the above for the reasons given, and also because without exploration it is impossible to tell whether or not the testicle is ruptured. Furthermore, a neoplasm can sometimes present in this way.

Old Clotted Hæmatocele.—Curiously, slow hæmorrhage into the tunica vaginalis can occur spontaneously, and apparently painlessly, for most of the patients presenting with an old clotted hæmatocele give neither a history of trauma to the testis nor of pain in the organ. An old clotted hæmatocele simulates a neoplasm of the testis so closely that pre-operative differential diagnosis is sometimes impossible.

FIG. 1514.—An old clotted hæmatocele. The long-standing pressure has flattened the testis.

Treatment.—Unless exposure of the organ leaves no doubt as to the innocent nature of the swelling, unquestionably orchiectomy should be performed. As a rule it is impossible to be certain of the diagnosis until the mass has been bisected. As can be seen in fig. 1514, the testicle is often so compressed as to be virtually functionless, so the responsibilities of removing it unnecessarily are not great. Russell Howard, renowned in his day as a surgical diagnostician and teacher, was wont to recount how he had performed orchiectomy for old clotted hæmatocele on the mistaken diagnosis of malignant testis on three consecutive occasions.

CYSTS CONNECTED WITH THE EPIDIDYMIS

1. Cysts of the Epididymis.—A cyst of the epididymis, filled with crystal-clear fluid (as opposed

Otto Eduard Heinrich Wucherer, 1820–1873. German Physician who practised in Brazil.
Joseph Bancroft, 1836–1894. Surgeon, General Hospital, Brisbane, Australia.
Russell John Howard, 1875–1942. Surgeon, The London Hospital.

to the barley-water-like fluid of a spermatocele or the amber-coloured fluid of a vaginal hydrocele), is a common condition.

Ætiology.—A cyst of the epididymis is due to cystic degeneration of:
(*a*) The paradidymis (or organ of Giraldes), which is the most likely cause.
(*b*) The appendix of the epididymis (pendunculated hydatid of Morgagni) (fig. 1515).
(*c*) The appendix of the testis (sessile hydatid of Morgagni).
(*d*) The vas aberrans (of Haller).

Clinical Features.—Cysts of the epididymis usually are found during middle life and, due no doubt to their congenital origin, they are often bilateral. They are tense, as opposed to the softness of a spermatocele, and they consist of an aggregation of a number of small cysts which on careful palpation feel like a bunch of tiny grapes. Cysts of the epididymis are situated *behind* the body of the testis, and on transillumination, because of the numerous septa, their brilliant translucency is finely tesselated, giving an appearance like that of a Chinese lantern.

Treatment.—As epididymeal cysts are multilocular, aspiration is useless. If causing discomfort, which is unusual, they must be excised. They should not be operated on without considering the fact that excision will almost certainly cause infertility from blockage.

Cyst of an appendage of the testis is a separate clinical entity. It forms a small globular swelling at the superior pole of the testis; it is usually unilateral, and when pedunculated (fig. 1515) it is liable to undergo axial rotation (p. 1321). The treatment of this variety of cyst is removal by ligation and severance of its pedicle.

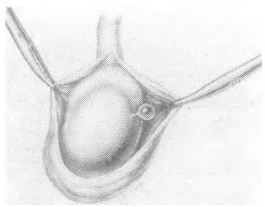


FIG. 1515.—Cyst of the pedunculated hydatid of Morgagni.

2. Spermatocele is a unilocular retention cyst derived from some portion of the sperm-conducting mechanism of the epididymis.

Clinical Features.—A spermatocele is nearly always situated in the head of the epididymis, and is therefore above and behind the body of the testis. Filled with fluid resembling barley water and containing spermatozoa, the swelling is typically softer than other cysts occurring within the scrotum but, like them, is translucent. Spermatoceles give rise to few symptoms, and are usually small and unobtrusive. Less frequently they are large enough to attract notice.[1]

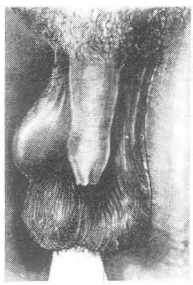


FIG. 1516.—A large spermatocele.

Treatment.—Small spermatoceles can be ignored. Larger ones, if causing symptoms, can be aspirated or excised via a scrotal incision. The thin coverings of the cyst are carefully peeled off, layer by layer, and any residual attachments are ligated and divided.

ACUTE EPIDIDYMO-ORCHITIS

In many instances inflammation remains confined to the epididymis, in which case it is referred to as epidi-

[1] Sometimes the patient may believe that he has a third testicle (fig. 1516). It is recorded that in the fourteenth century the Pope granted a petition from a gentleman to marry two wives because he possessed three testicles.

Joachim Albin Cardazo Cazado Giraldes, 1808–1875. Professor of Surgery, Paris.
Albrecht von Haller, 1708–1777. Professor of Anatomy, Physiology, Surgery, and Botany, Gottingen, Germany.

dymitis; when the infection spreads to the body of the testis the condition is known as epididymo-orchitis.

Mode of Infection.—As a rule infection reaches the epididymis via the lumen of the vas, and affects the globus minor first. It is secondary to infection of the urethra, prostate and seminal vesicles. Blood-borne infections affect the globus major of the testis primarily.

Clinical Features.—The initial symptoms are those of acute prostatitis (p. 1280). Some days later an ache in the groin with some increase in fever heralds the onset of epididymitis. Severe pain and swelling of the testis occurs rapidly: the scrotal wall becomes red, œdematous and shiny. The epididymis may become adherent to the scrotal skin and may soften and, later, discharge. Resolution is heralded by scaling of the scrotal skin. Complete resolution of the swelling in the epididymis may take 6–8 weeks.

Acute epididymitis due to *Esch. coli*, streptococcus, staphylococcus or proteus is a not uncommon clinical entity. As a rule there is no evidence of urethritis. Especially in *Esch. coli* infection, it is sometimes unaccompanied by infection of the urine (an initial specimen should be sent for bacteriological examination). In the absence of any local cause, the infection is assumed to be blood-borne.

Acute epididymitis can follow any form of urethral instrumentation. It usually occurs when there is an associated infection in the prostate. Indwelling catheters encourage organisms normally found in the distal urethra to migrate upwards.

Epididymitis complicating non-gonococcal urethritis is more frequent than it is in gonorrhœa, and unless correct antibiotic therapy (p. 1295) is given early, suppuration is usual.

Acute gonococcal epididymitis is much less common than formerly, as a result of the antibiotic treatment of acute gonorrhœa. Epididymitis usually commences during the second or third week of gonococcal posterior urethritis. In 90 per cent. of cases the infection is unilateral. When the epididymis becomes involved the urethral discharge lessens and pain commences in the testis. On the following day the urethral discharge ceases, but the urine is hazy with pus cells. The temperature rises to 38·9° to 39·5° C, and the patient feels so ill that he goes to bed. About the third day the skin of the scrotum often becomes reddened and adherent to the globus minor, but in cases where the gonococcus is the sole infecting organism suppuration does not occur. After the fifth day the symptoms commence to abate, and by the eighth to tenth day the condition subsides. Induration of the globus minor sometimes persists for months. In cases of persistent infection of the corresponding vesicle, subacute attacks of recurrent epididymitis are not infrequent.

Epididymitis from Retrograde Passage of Urine.—Epididymitis can develop during an unusual exertion or violent strain while the bladder is full, and it is caused by the urine being forced along the vas deferens. The degree of inflammation that results depends upon whether the urine is infected, or if there is an active or latent infection in the prostate or seminal vesicles. This 'reflux' epididymitis can also occur due to the presence of a stricture in the urethra.

Acute Tuberculous Epididymitis.—In cases where the vas is thickened and there is little response to the usual treatment, the possibility of the infection being due to tuberculosis of the epididymis, which is not rare, must receive due consideration.

Acute epididymo-orchitis of mumps develop in about 18 per cent of males suffering from mumps, usually as the parotid inflammation is waning. The testis becomes swollen and painful. Rarely, the testicular precede the salivary manifestations. Resolution or fibrosis, as opposed to suppuration, nearly always occurs, but in 55 per cent. of cases atrophy of the testis follows. It often takes many months for signs of testicular atrophy to become apparent. A small incision into the tunica vaginalis and drainage, if undertaken early in the attack, prevents this atrophy and relieves the intense pain. It is often stated that the body of the testis is alone involved, but the epididymis is also acutely inflamed, as has been witnessed at operation. Especially in

infants, epididymo-orchitis of mumps can occur without parotitis.[1] Partial atrophy is commonly associated with a tendency to recurrent pain in the testis.

Acute post-operative epididymitis was a serious and frequent complication of prostatectomy. It can be abolished by prophylactic division of the vasa deferentia (p. 1270), and has been greatly reduced by closed drainage and catheter care which have reduced the rate of post-operative urinary infection.

Other causes include Bornholm disease,[2] Brucellosis, and Lympho-granuloma venereum.

Treatment.—The patient must be kept in bed until the acute symptoms have abated. A broad spectrum antibiotic such as tetracycline should be given (500 mg. q.d.s.) until the result of the urine culture is available. The scrotum is supported on a sling formed by broad adhesive plaster attached across the thighs (fig. 1517). Upon the sling is placed a nest of cotton wool, and in this the inflamed organ rests and cooling lotions are applied. The urine is rendered alkaline, and a high fluid intake is necessary. Antibiotic treatment should be continued for two weeks or until the inflammation has subsided. If suppuration occurs, drainage is necessary. Atrophy of the testis may follow as a sequela in any case of epididymo-orchitis.

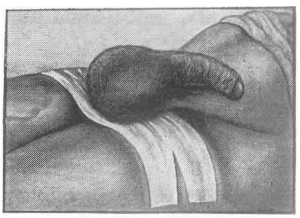


FIG. 1517.—Treatment of acute epididymo-orchitis. Broad adhesive strapping is applied to the thighs to support the testicles.

CHRONIC EPIDIDYMO-ORCHITIS

Chronic Tuberculous Epididymitis.—Nearly 90 per cent. of cases of chronic epididymo-orchitis are tuberculous, and the great majority commence insidiously.

Ætiology.—The frequency with which the globus minor is first attacked indicates that infection is commonly retrograde along the vas deferens from an infected seminal vesicle. Blood-borne infection commences in the globus major.

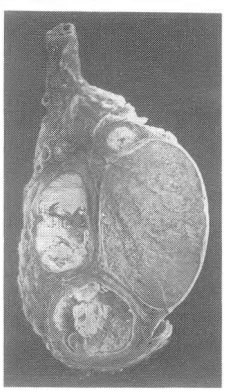


FIG. 1518.—Tuberculous epididymitis.

Clinical Features.—A slight ache in the testis or a trivial injury, calls the patient's attention to the swelling in relation to the testis. An early manifestation is a discrete, indurated, slightly tender nodule in the globus minor—rarely in the globus major. As the disease progresses other nodules appear, and eventually the entire epididymis becomes involved, and is felt as a firm, craggy, often painless, mass situated behind the testis, which feels normal. In 30 per cent. of cases a lax secondary hydrocele is present. When the testis is anteverted, these changes are found in front of the testis. The vas on the affected side sometimes becomes 'beaded' due to submucous tubercles. Rectal examination reveals a thickened, and later an irregular, indurated seminal vesicle of the corresponding side, and sometimes of the

[1] A complement fixation test for the virus of mumps is sometimes positive.
[2] Epidemic myalgia. Bornholm is a Danish island in the Baltic sea.

Sir David Bruce, 1855–1931. Major-General, Royal Army Medical Service.

contralateral vesicle as well. The prostate in advanced cases contains one or more discrete nodules. In 20 per cent. of cases the patient first presents himself with a 'cold' abscess in the lower and posterior aspect of the scrotum or with a discharging sinus, which may have healed and reopened several times. Untreated, after a varying time (from a few months to a year or so), the contralateral epididymis becomes similarly diseased. The body of the testis remains uninvolved for a long period, often years.

In two-thirds of the cases there is either active tuberculosis in the renal tract or evidence of previous disease. In the remainder the patient is apparently healthy.

In all cases of chronic epididymitis the urine is examined for tubercle bacilli, if necessary on several occasions, and pyelography should be undertaken. The chest is X-rayed (evidence of disease in about 50 per cent.). Culture of the semen can also be a useful means of confirming the tuberculous nature of an epididymitis.

Treatment.—When tuberculous epididymitis is secondary, the primary lesion should receive priority. The epididymitis may well resolve during the treatment.

When renal tuberculosis is present, the renal lesion is treated as detailed on p. 1213. Rarely active pulmonary tuberculosis will be present.

Treatment with streptomycin and associated anti-tuberculous drugs (p. 37) is less effective in genital tuberculosis than in urinary tuberculosis. If there is no sign of resolution within two months, epididymectomy or orchiectomy is advisable. Subsequently, in the absence of evidence of active tuberculosis elsewhere, a further three months' course of suitable and anti-tuberculous drugs is all that is necessary. Care of the patient in a sanatorium is an insurance that he will adhere to the treatment.

Chronic non-tuberculous epididymitis either follows an acute attack or is chronic from the commencement. The condition is difficult to distinguish from tuberculosis, but as a rule the epididymis in non-specific epididymitis is larger and smoother. It is essential to exclude a urethral stricture.

Treatment.—In most cases associated posterior urethritis, vesiculitis, or prostatitis must be eradicated. If the condition fails to resolve after six to eight weeks of conservative treatment and antibiotic therapy, epididymectomy or orchiectomy may be needed.

ORCHITIS

Syphilitic Orchitis.—Syphilis attacks the body of the testis, and like other late manifestations of the disease, all three varieties have become uncommon, interstitial fibrosis the least so. The three varieties of syphilitic orchitis are:

(*a*) *Bilateral orchitis* occurs in congenital syphilis (p. 47).

(*b*) *Interstitial fibrosis* is bilateral, symptomless, and leads to gradual destruction of the seminiferous tubules. The testes are not enlarged. They are firmer than normal, and there is a gradual loss of testicular sensation. In bisecting the testis, irregularly distributed streaks of fibrosis are seen traversing the parenchyma.

(*c*) *Gumma.*—The condition is nearly always unilateral. The body of the testis enlarges slowly and painlessly. At this stage it feels extremely hard. Testicular sensation is lost, and there is nearly always an associated secondary hydrocele. Later there is softening anteriorly and the skin of the scrotum becomes involved, and a

gummatous ulcer forms. If the response to treatment is not rapid it is better to perform orchiectomy than risk the possibility of overlooking a neoplasm with purely coincidental positive serological tests.

Leprous Orchitis.—Over 25 per cent. of male lepers have testicular atrophy due to fibrosis engendered by the direct action of the *Mycobacterium lepræ*, and in 20 per cent. of those so affected the orchitis is associated with gynæcomazia.

NEOPLASMS OF THE TESTIS

The lymphatics of the testis run upwards in the spermatic cord, and, passing through the internal inguinal ring, they branch fountain-wise (behind and adherent to the posterior peritoneum) towards the para-aortic lymph nodes in the region of the origin of the spermatic vessels. Above this level the lymph drains into the thoracic duct which extends through the mediastinum to the left supraclavicular fossa, where it drains into the left innominate vein. Lymphatics from the medial side of the testis may run with the artery to the vas and drain into a lymph node at the bifurcation of the common iliac artery.

When lymphatic spread of a neoplasm occurs, contralateral lymph nodes can be affected. Inguinal lymph nodes are only affected if the scrotum is involved.

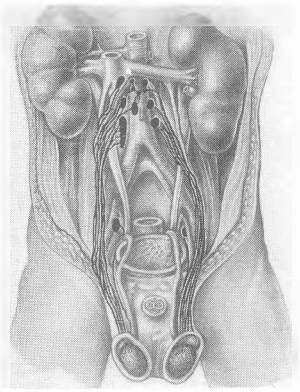


FIG. 1519. — The lymphatics of the testes. (*After Rouvier.*)

MALIGNANT NEOPLASMS OF THE TESTIS

About 99 per cent. of neoplasms of the testis are malignant, and though they only constitute about 1–2 per cent. of malignant tumours in the male, they are one of the commonest forms of cancer in young adult males. Maldescent undoubtedly predisposes to malignancy (p. 1317). It is curious that a testicle bearing such a tumour, contained as it is in a bag of skin bereft of fat, and thus more readily accessible to the examining fingers than a tumour of any other organ in the body, only too often escapes detection until it has metastasised. In this connection, perhaps the worst error is to plunge a trocar and cannula into the enlarged testis in the belief that it is a hydrocele, for want of applying the test of translucency. This has been done more often than the reader may be inclined to believe.

Types of Tumour.—Tumours of the testis are classified as follows:

(1) Seminoma	40 per cent.
(2) Teratoma	32 per cent.
(3) Combined Seminoma and Teratoma	14 per cent.
(4) Interstitial tumours	1·5 per cent.
(5) Lymphoma	7 per cent.
(6) Other tumours	5·5 per cent.

The percentage of the first two subdivisions varies in different series. Those series emanating from hospitals for Service personnel show a higher percentage of teratomata. The reason for this is that patients in such hospitals are likely to be of a younger age-group than found in the community at large; **the peak incidence** of teratoma being between twenty and twenty-five years of age, while that of seminoma is between thirty and thirty-five. Seminoma has only once been recorded before puberty.

Seminoma commences in the mediastinum of the testis, and as it increases in size the neighbouring testicular tissue is compressed (fig. 1520). Macroscopically the enlarged testis is firm and smooth, and on section homogeneous and pink or cream in colour. Occasionally fibrous septa give it a lobulated appearance. In rapidly growing tumours areas of necrosis are sometimes present, in which case the consistency is variable.

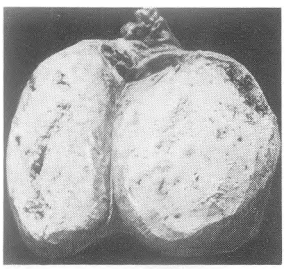


FIG. 1520.—Seminoma of the testis.

Histologically it is composed of rounded, slightly oval cells with clear protoplasm and large rounded nuclei containing acidophile nucleoli. The cells are arranged in sheets separated by a fine fibrous stroma. Derived from the seminiferous tubules, the cells resemble spermatocytes. Stromal and tumour infiltration by lymphocytes suggest a good host reaction (? autoimmune reaction) and tends to improve the prognosis.

A seminoma metastasises almost exclusively by the lymph vessels to lymph nodes; only occasionally does it disseminate by the blood-stream and give rise to secondary deposits in other situations.

Teratoma arises in the rete testis from toti-potent cells; accordingly, elements of ectoderm, mesoderm, or endoderm are often represented. Usually one of these predominates and attains malignancy. The tumour varies in size from that of a peanut to that of a coconut, and even when large, as it is moulded by the tunica vaginalis, the shape of the testis is more or less retained but the surface is usually slightly irregular.

The most usual variety[1] is yellowish in colour and shows cystic spaces (fig. 1521) containing gelatinous fluid. Cartilaginous nodules are often present.

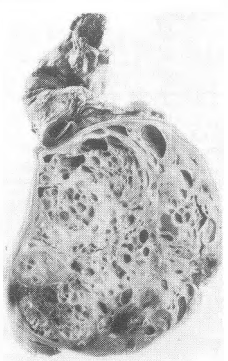


FIG. 1521.—Teratoma of the testis containing solid and cystic areas. (*Dr. Keith Simpson, London.*)

Histology.—The Testicular Tumour Panel have classified teratomas as follows:

(1) **Teratoma Differentiated (T.D.)** (1 per cent.[2]).—The best-known variety of this type of tumour is a dermoid cyst. If a teratoma has no histologically recognisable malignant components, it is termed differentiated, but it cannot be considered to be benign, because such growths may have metastasised. Many patients have been cured following simple orchiectomy. With radical orchiectomy the outlook is improved, the five-year survival rate being over 80 per cent.

(2) **Malignant Teratoma Intermediate (M.T.I.—A and B)** (30 per cent.), consisting of definitely malignant and incompletely differentiated tissues. In type A some mature tissue will be seen. Type B is more malignant and no fully differentiated tissue can be found. The tumours are mostly radio-resistant (see prognosis, p. 1336).

(3) **Malignant Teratoma Anaplastic (M.T.A.)** (1·5 per cent.) is composed of quite undifferentiated cells, which are radio-resistant.

(4) **Malignant Teratoma Trophoblastic (M.T.T.)** (1 per cent.) contains within the other tissue types a syncytial cell mass with malignant villous or papillary cytotrophoblast (choriocarcinoma). In some cases it is associated with the production

[1] Formerly known as ' fibro-cystic ' disease.

[2] The percentages given refer to all cases of testicular neoplasm.

of chorionic gonadotrophin and gynæcomazia. Spread is by the blood-stream as well as by lymphatics while the primary tumour is small, and it is often described as the most malignant tumour known. It is usually radio-resistant. Methotrexate may produce a temporary regression but death results within months. However, a choriocarcinoma is frequently associated with teratoid tissue in the form of a terato-choriocarcinoma, and patients with this hybrid choriocarcinoma have survived five years following *radical* orchiectomy (p. 1335).

Interstitial Cell Tumour.—A tumour of the testis arising in the interstitial cells of Leydig occurs at an early age or the prime of life, and arises either in the cells of Leydig or those of Sertoli.

A Leydig cell tumour masculinises; a Sertoli cell tumour feminises. For clinical reasons these tumours are classified as those occurring before or after puberty.

Prepubertal tumour is characterised by an excessive output of androgen, which is responsible for sexual precocity and extreme muscular development. This results in an infant Hercules, in all respects similar to the condition produced by an adrenal cortical tumour (see p. 653), except that on palpation a swelling of the testis can be elicited. Exceptionally the tumour is bilateral, but it must be noted that in this condition the contralateral normal testis frequently becomes hypertrophied. In so far as the neoplasm is concerned, removal of the testis is curative; in some cases the symptoms regress, in others (where the contralateral testis is hypertrophied) the regression is incomplete.

Post-Pubertal Tumour.—In a high percentage of cases the tumour arises in the cells of Sertoli, and consequently the output of feminising hormone becomes excessive. Gynæcomazia, loss of libido, and aspermia result; in such cases the Aschheim-Zondek test is positive. As a rule the tumour is innocent, and orchiectomy is curative.

Human Chorionic Gonadotrophin in a 12-hour collection of urine, may be estimated by a radio-immuno assay technique. It is likely to be very high in cases containing chorionic elements (T.T.). Normal levels are up to 100 iu HCG.

Clinical Features of Testicular Tumours.—Various clinical types are met with.

1. **Typical.**—The patient may not seek advice until four to six months after the first symptom, which is enlargement of the testicle. A sensation of heaviness is complained of when the testis has reached two to three times its normal size. Pain is complained of in only about 30 per cent. of cases (Fergusson). There is a history of trauma to the affected side in over 10 per cent. It is generally conceded that injury merely calls attention to the testicular enlargement and in no way initiates the neoplasm.

On examination the body of the testis is found to be enlarged, firm, smooth, and heavy. Later one or more softer bosses may be palpable. In no other disease is testicular sensation lost so early or so completely, but the greatest gentleness must be exercised in eliciting this sign for fear of disseminating the neoplasm. A secondary hydrocele is present in 10 per cent. of cases; it is usually lax, and seldom obscures the enlarged testis. The epididymis is normal at first; later it becomes so flattened or incorporated in the growth that it is imperceptible. The spermatic cord remains normal for a considerable time. It then becomes thickened, due to cremasteric hypertrophy and enlargement of testicular vessels. The vas is never thickened. Rectal examination reveals no abnormality in the prostate or seminal vesicles.

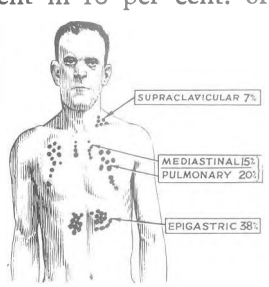


FIG. 1522.—Distribution of metastases in teratoma of the testis.

Next, the sites of possible metastases (fig. 1522)

Franz von Leydig, 1821–1908. Professor of Histology, Bonn, Germany.
Enrico Sertoli, 1842–1910. Professor of Experimental Physiology, Milan, Italy.
Selmar Aschheim, 1878–1965. Formerly Honorary Professor of Gynæcology, University of Berlin.
Bernhard Zondek, 1891–1966. Professor of Obstetrics and Gynæcology, Hadassah Medical School, Hebrew University, Jerusalem, Israel.
John Douglas Fergusson, 1910–1979. Surgeon, St. Paul's Hospital, London.

should be examined. The abdomen is palpated for secondary retroperitoneal deposits, their most usual situation being just *above the umbilicus* on the same side, the opposite side, or both sides. The left supraclavicular region is examined, and occasionally the first presenting sign is an enlarged supra clavicular lymph node. An X-ray of the chest may show metastases in the lungs, particularly in cases of teratoma (fig. 1523).

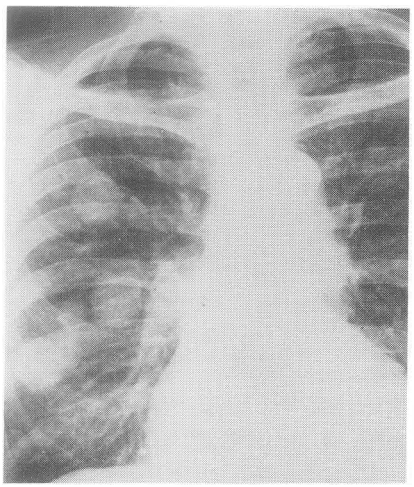


FIG. 1523.—'Cannon ball' metastases from a testicular tumour.

2. **When the predominant symptoms are due to metastases:** (*a*) with a seminoma the patient complains of abdominal or lumbar pain, and on examination a mass is discovered in the epigastrium. (*b*) Pulmonary metastases from a teratoma of the testis give rise to few, if any, early symptoms. Later the patient may complain of pain in the chest, dyspnœa, and perhaps hæmoptysis.

In either case the enlargement of the testis may not have been noticed by the patient, and is discovered only by a thoughtful clinician. Sometimes the primary growth is tiny and will only be discovered by operative exploration.

3. **Atypical Cases.**—The variations are:

(*a*) Some cases (7 per cent.) simulate *epididymo-orchitis*; there may even be organisms in the urine. Failure to respond to a short sharp course of antibiotics should make one say 'this could be a tumour' and action must be taken. Rarely, patients will present with severe pain and acute swelling due to a hæmorrhage. Between 1 to 5 per cent. of cases have gynæcomastia (mainly the teratomas).

(*b*) *The Hurricane Type.*—Metastases from a highly malignant tumour bring about a fatal termination in a matter of weeks (Gordon-Taylor).

(*c*) *The Slowly Growing Type.*—In a few cases of teratomas, the patient notices an increasing enlargement of the testis for two or three years.

Management of a Suspected Case of Neoplasm

The action to be taken when the clinical diagnosis is made is:

(1) Collect a 12-hour sample of urine for gonadotropin estimation by radio-immune assay (normal = < 100 iu HCG).
(2) Take a chest X-ray to exclude secondary deposits.
(3) Orchiectomy (or exploration).
(4) Confirmation of pathology by serial histology.
(5) Radiography (I.V.P. and Lymphangiography).
(6) Radiotherapy.

Sir Gordon Gordon-Taylor, 1878–1960. Surgeon, Middlesex Hospital, London.

Orchiectomy (retrograde simple[1] orchiectomy).—This should be done at once. Through an inguino-scrotal incision the inguinal canal is opened to display the spermatic cord, which is divided and ligated at the level of the internal inguinal ring. The spermatic cord and the testis are then removed from above downwards by separating them from their attachments. The wound is closed, and radiotherapy starts as soon as the pathology is confirmed and the sutures have been removed.

Exploration.—On occasion when the clinical diagnosis is in doubt, delay is still dangerous, and nothing is lost by exposing the testis first, providing a ' soft ' clamp is placed across the cord above to prevent dissemination. The testis may have to be split open and any doubtful material subjected to immediate frozen section. Wedge biopsy without frozen section and needling should not be attempted for fear of involving the scrotum and opening up an alternative way of lymphatic spread (to inguinal lymph nodes) (p. 1331).

Confirmation of Pathology.—The pathologist will report the type and grade of the tumour (p. 1332).

Radiography should be used as follows:

(*a*) Pre-operative X-ray of the chest to exclude metastases.

(*b*) Excretory urography (I.V.P.). This has two purposes: (1) Protection of kidney from radiotherapy, for besides ascertaining the function of the kidneys, their position must be marked out so that they may be shielded during irradiation in order to prevent radiation nephritis and hypertension. (2) Detecting the presence and extent of retroperitoneal metastases by the displacement of the ureters or deformity of a renal pelvis. Changes in radiographs after irradiation may indicate the radio-sensitivity of the tumour.

(*c*) Lymphangiography can demonstrate secondary deposits in para-aortic lymph-nodes. Its greatest value lies in outlining enlarged nodes which can be observed during radiation for shrinkage.

Radiotherapy.—Radiotherapy has made an enormous difference to the prognosis in testicular tumours. Most, but not all, seminomas are highly radio-sensitive. Teratomas are less sensitive. Well over 80% of patients suffering from seminoma of the testis without pulmonary metastases survive more than five years after simple orchiectomy followed by radiotherapy.

From its inception, radiotherapy of tumours of the testis has been based on the assumption that retroperitoneal lymphatic metastases have occurred whether or not they are palpable. Such irradiation should be given by a cobalt or linear accelerator X-ray unit to permit the desired deep dose to be given in shorter time, with greater certainty, with less injury to superficial tissues, and with less general reaction than is possible with machines of lower voltage. When there is no clinical or radiological evidence of metastases the radiation is confined to the area between the scrotum and the 10th dorsal vertebra, with suitable screening of the remaining testis. In patients who are eager to have more children the remaining testis is shielded by lead blocks and moulds. Even so there is some danger of mutations. Only careful follow-up of descendants will show whether this procedure is the

[1] *Simple*, as opposed to the radical operation p. 1336.

correct one. The average dose is 3,000 rad for a seminoma, and 4,500 rad for a teratoma, given over some five or six weeks. It may take longer if interrupted by an excessive fall in the platelet count.

In more advanced cases radiotherapy is given through one inguinal, four abdominal, two mediastinal and two left supraclavicular portals. Chlorpromazine may be given to control the vomiting of radiation sickness, so common in patients receiving radiotherapy in high dosage.

Pulmonary Metastases.—Sometimes irradiation results in regression, but almost invariably this is followed by fresh deposits. Frequently the dose of X-rays converts a condition that was symptomless into one accompanied by severe pulmonitis, discomfort and respiratory deterioration, and it is doubtful if the X-ray treatment of pulmonary metastases is worth while (Cox).

Block Dissection of Retroperitoneal Lymph Nodes (radical operation).—With improvements in radiotherapeutic apparatus and techniques this operation is not often employed. When it is attempted, in up to 25% of cases some nodes are inoperable; 50–60% show no node involvement.

Principles of Technique.—An extensive retrograde exposure is required to allow the removal of spermatic vessels and lymphatics from the internal ring up to the region of the renal vessels (Lymphatics, p. 1331). The vas is transected above the bladder. Only part of the incision is made to start with, for through this the peritoneum is opened and the upper abdomen is explored to exclude very high fixed lymph nodes which would make the case inoperable.

Chemotherapy.—In disseminated seminoma and teratoma, chemotherapy may be given a trial and perhaps in 20% of cases some benefit may be achieved. Methylhydrazine 100 mg b.d. orally has produced some good results in anaplastic teratoma. Mithramycin given intravenously to a dose of 600 μg is also being used with some hopeful reports, while methotroxate, or a combination of this drug with Actinomycin D and chlorambucil, is most useful in chorionic teratoma. Hopeful results are also being achieved with cisplatinum.

Prognosis of Testicular Tumours.—The histological appearance of the tumour and the presence or absence of metastases at the time of operation are the two main factors in assessing prognosis:—

Seminoma.—No evidence of metastases; with orchiectomy and radiotherapy 85 per cent. survive five years. If metastases are present, only 25 per cent. survive five years.

Teratoma.—The histological appearances are very important (p. 1332). Excluding Teratoma Differentiated, if no metastases are present, up to 40 per cent. survive five years. If metastases are present only 1 to 5 per cent. survive three years.

Testicular tumours in children most commonly appear during the first year of life and are exceedingly rare after the third birthday. The tumours at this time of life are teratomas, many of these being anaplastic (T.A.) in which event, in spite of treatment, the patient dies within a few months. Now and then the tumour proves to be differentiated (T.D.) when, following orchiectomy alone, the prognosis is exceptionally good. On rarer occasions still, the tumour proves to be an interstitial cell tumour (p. 1333), in which case, following orchiectomy, the prognosis is also excellent.

Tumours of the Epididymis.—Mesothelioma is a unique innocent tumour that usually springs from the globus minor : most examples have been about the size of a cherry. Sarcoma, and very rarely carcinoma, arise in the epididymis, and those who put their trust in the aphorism that swellings of the epididymis are invariably inflammatory may one day rue it. If the swelling in question does not respond to rest and

Robert Cox, Contemporary. Consulting Surgeon, Westminster Hospital, London.

antibiotic therapy quickly, it should be treated by epididymectomy or, if obviously malignant, by orchiectomy.

THE SCROTUM

Prepenile scrotum is an exceedingly rare congenital condition. The scrotum, suspended from the mons pubis, is situated anterior to the penis.

Idiopathic Scrotal Œdema is described on page 1321.

FIG. 1524.—Fournier's gangrene.

Idiopathic gangrene of the scrotum (*syn.* Fournier's gangrene) is uncommon but fearful in effect. It is a vascular disaster of infective origin. The three cardinal characteristics of this disease are: (1) sudden appearance of scrotal inflammation in the midst of apparently good health; (2) rapid onset of gangrene (fig. 1524); (3) absence of any cause in over half the cases. It has been known to follow minor injuries or procedures in the perineum, such as a bruise, scratch, dilatation of stricture, injection of anal fissure or opening a periurethral abscess.

The hæmolytic streptococcus (sometimes micro-ærophilic) associated with other organisms (staphylococci, *E. coli*, *Cl. Welchii*) set up a fulminating inflammation within the scrotal subcutaneous tissues that results in obliterative arteritis of the arterioles supplying the overlying skin (cf. gangrene of the abdominal wall, p. 1131).

The clinical features are: sudden pain in the scrotum, prostration, pallor, and pyrexia. At first only the scrotum is involved: still unchecked, if the patient survives, the cellulitis spreads along those planes so well known in superficial extravasation of urine (p. 1292). Sometimes the entire scrotal coverings slough, leaving the testes hanging exposed, bared to their tunica, though, remarkably, free from gangrene.

Treatment.—Pending the bacteriological report, chloramphenicol or Gentamycin and Ceporin are the antibiotics of choice as the organisms are usually sensitive.

The scrotum should be incised. If gangrene has commenced, wide excision of all sloughing areas saves time, rids the patient of scrotal tissue that is doomed, provides the freest possible drainage, and stops the spread of gangrene.

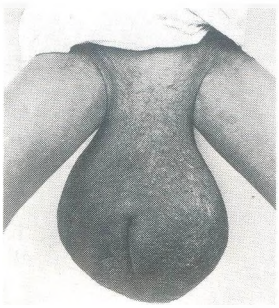


FIG. 1525.—Elephantiasis of the scrotum, burying the penis. (*Professor M. Bahadur Khan, Hyderabad, India.*)

Filarial elephantiasis of the scrotum is confined to those who dwell, or have dwelt, in tropical and subtropical countries, and is due to obstruction of the lymphatic vessels of the pelvis by *Wuchereria bancrofti* (p. 1326), with superadded infection and lymphangitis. The earliest manifestation is an attack of funiculitis with the development of a secondary hydrocele. Repeated attacks cause the scrotum to remain œdematous and the scrotal skin and subcutaneous tissue greatly thicken. In long-standing cases the scrotum becomes immense, and the penis is buried within it (fig. 1525).

Treatment.—There is no medical treatment for the condition. The principle of operative treatment is the construction of new lymphatic pathways. This can be achieved by constructing cellular-cutaneous bridges

Jean Alfred Fournier, 1832–1914. Founder of the Venereal and Dermatological Clinic, Hôpital St. Louis, Paris.

of generous size uniting the scrotum to the thigh, for the purpose of permitting the scrotal lymph to be absorbed via the uninvolved thighs. In very advanced cases excision of all involved skin together with implantation of the testes into the thighs and skin grafting of the bared shaft of the penis, if necessary, is the only curative treatment. It should be especially noted that the *inner layer* of the prepuce is rarely involved: it should therefore be spared and in many instances it can be utilised to cover the bare shaft of the penis (Bowesman). In all cases concomitant hydroceles are dealt with by excision of the sac at the time of the operation.

FIG. 1526.—Sebaceous cysts of the scrotum.

Non-filarial elephantiasis can be acquired in non-tropical as well as tropical climates, and it is consequent upon fibrosis of lymphatic vessels due to lymphogranuloma venereum. Non-filarial elephantiasis never reaches the extravagant proportions of tropical elephantiasis. There is no effective medical treatment: the scrotum-thigh cutaneous tunnel operation described above is recommended.

Sebaceous cysts are common in the skin of the scrotum. They are usually small and multiple (fig. 1526).

Carcinoma of the Scrotum.—In the last century squamous epithelioma of the scrotum was rife amongst chimney sweeps.[1] With improved working conditions, this occupational disease ceased to exist in as far as chimney sweeps were concerned, but with the mechanisation of the cotton industry, lubricating oil from the spinning jenny soaked the crutch of the mule spinners' trousers, and proved more carcinogenic than soot. Now that a lubricating oil free from impurities has been substituted it is hoped that carcinoma of the scrotum will no longer attack[2] mule spinners in particular. A few cases still occur in tar and shale oil workers, but in the majority of cases encountered at the present time there is no known ætiological factor. It is remarkable that, in contradistinction to carcinoma of the penis, carcinoma of the scrotum is almost unknown in India and Asiatic countries.

Clinical Features.—The growth commences in a wart or an ulcer (fig. 1527). As it extends it may involve the underlying testis.

Treatment.—The growth, together with a considerable margin of healthy tissue, is excised by diathermy. If, as is often the case, the inguinal lymph nodes are enlarged and do not subside with antibiotic treatment, a block dissection of these nodes, and of the external iliac nodes, should be carried out, usually on both sides.

FIG. 1527.—Carcinoma of the scrotum with bilateral metastases in the inguinal nodes. *(Department of Medical Photography, Cardiff Royal Infirmary.)*

[1] This disease was first described in 1775 by Percivall Pott (1714–1788), Surgeon, St. Bartholomew's Hospital, London. In those days the chimney sweep's apprentice climbed up and down the chimney.

[2] As it may take up to twenty years for the result of exposure to carcinogens to become manifest, the outcome as yet is uncertain.

Charles Bowesman, Contemporary. Formerly Surgical Specialist, Colonial Medical Service Kumasi, Ghana.

MALE INFERTILITY

Causes.—These are as follows:

1. *Impotence* due to (*a*) malformations or loss of the penis; (*b*) psychic causes; (*c*) chromosome defects.

2. *Oligospermia & aspermia* due to small (fibrotic) or soft (partially atrophic) testes resulting from mumps (quarter of cases), varicocele (p. 1321), cryptorchism, over-exposure to X-rays, and occasionally bilateral epididymo-orchitis.

3. *Aspermia Due to a Faulty Sperm-conducting Mechanism.*—Leading examples are (*a*) urethral fistula, (*b*) tight urethral stricture, (*c*) obliteration by scar tissue of both common ejaculatory ducts or both vasa deferentia. Fifteen per cent. of all cases of infertility are due to gonorrhœa, (*d*) absent vasa and vesicles.

Investigation includes: (*a*) three semen analyses at weekly intervals, and an adjudication of the average, (*b*) plasma FSH, LH, prolactin, testosterone, and œstrodiol when there is a low sperm count, (*c*) search for sperm antibodies when there is a normal count, (*d*) testicular biopsy and vasogram when there is azoospermia and normal sized testes, (*e*) chromosomal studies particularly when the testes are atrophic.

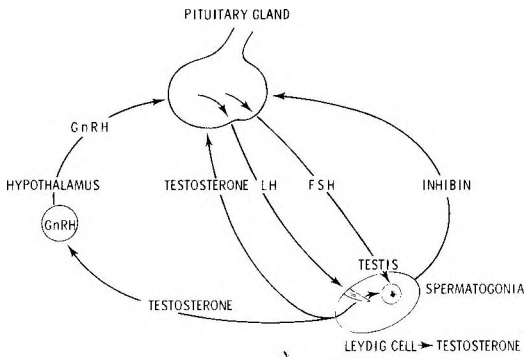


FIG. 1528.—Hormonal interrelationships between the pituitary gland, the hypothalamus and the testis.

Treatment.—(1) Avoid wearing tight pants (see p. 1322). (2) Cold bathing of the scrotum. (3) Clomiphene in cases of low FSH levels and maturation defect. (4) Oral testosterone (mesterolone 100 mg daily) in patients with a low count, relatively normal semen and low plasma testosterone. Or fluoxymesterone (5 mg daily) where motility is reduced. (5) Vitamin B_{12} has been reported to improve semen morphology. (6) Operation for varicocele, if present, and if there is a low count and low motility (p. 1323). (7) Epididymo-vasotomy in cases of obstruction, when vas is proved patent and testicular biopsy is normal:

Operation.—The vas is divided at least 2·5 cm above the portion considered to be obliterated, and in order to make certain of its patency, the proximal end is catheterised with a suitably-sized piece of silkworm gut, and patency down to the urethra proved by the injection of a weak solution of methylene blue while observing the verumontanem with a urethroscope. The silkworm gut is removed and the free end of

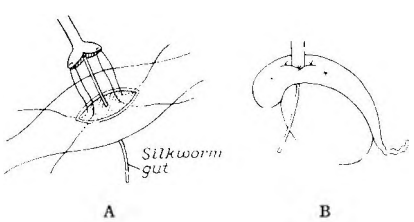


FIG. 1529.—End-to-side vaso-epididymal anastomosis. (*After S. Popelka* et al.)

the vas is split for a short distance. The silkworm gut is then reinserted. End-to-side anastomosis is carried out with 4/0 silk, the flaps resulting from the bifurcation being carried into the lumen of the epididymis (fig. 1529A). The silkworm gut splint

is brought out between the epididymis and the body of the testis (fig. 1529B), and it is also well to pass this through a minute puncture in the scrotum and withdraw it after several days, when the anastomosis can be presumed to have united.

Vasectomy for Sterilization

Vasectomy for the purpose of sterilisation should be undertaken only after very careful counselling, and this is best done by the general practitioner who knows the family thoroughly. Many requests for reversal follow the break-up of a marriage within a year of vasectomy. Also it is unwise to give a 100% guarantee that natural rejunction cannot occur.

The operation should be performed carefully under general anæsthesia, the edges of the external spermatic fascia should be held on hæmostats so that the lower cut end of the vas can be double-tied bent on itself and pushed back beneath this fascia while the upper end, similarly ligated, can be held outside this fascia, the edges of which are closed around it. The upper end therefore lies beneath the skin and dartos muscle.

Reanastomosis can be successful in up to 80% or more if carefully performed and sperm leak prevented. Results are best within the first 3–4 years of vasectomy—later the results are poorer. On this account little of the vas should be excised at vasectomy.

CHAPTER 61

ORGAN TRANSPLANTATION

THE transplantation of tissues and organs has a venerable history. Skin transplantation dates back to about 1500 B.C. Corneal and bone transplantation came more to the front in the second half of the nineteenth century and were followed in the early years of this century by whole organ transplantation after the work of Alexis Carrell.

Most tissue and organ transplants are rejected when the donor and recipient are genetically dissimilar. This rejection is an immunological phenomenon in which the recipient produces antibodies to antigens which the donor possesses but the recipient does not. Classically this rejection takes between 10–14 days to develop and to become fully effective. The result of this rejection is widespread infiltration of the transplant with lymphocytes, and microvascular changes which result in failure of the blood supply to the transplant.

Many potential recipients may previously have received sensitising challenges such as blood transfusions or previous transplantations. In these circumstances the recipient patient may show a modified response, resulting in an accelerated rejection in some, while ' enhancing ' the effect in others.

Any attempt, therefore, to make organ transplantation a clinically acceptable procedure, must modify this rejection process. Much of the work on this subject has been done with kidney transplants following the pioneering work of Murray, Calne, Hume and Starzl.

KIDNEY TRANSPLANTATION

The Recipient.—It is usually better if the prospective patient is prepared for transplantation by hæmodialysis. Pretransplantation infusion of at least 6 units of blood, during this period of hæmodialysis, appears to produce better transplant results.

Access to Circulation—External Fistulæ.—This is achieved by the insertion of *Teflon* cannulæ either at the ankle or the wrist. The artery at the ankle is usually the posterior tibial artery while the vein may be the long or short saphenous vein. At the forearm, the radial artery and basilic or cephalic veins are used. These procedures can often be performed under local anæsthesia. The two Teflon cannulæ are connected with a *Silastic* loop which is used for linkage to the dialysis unit. Thrombosis and infection of these external fistulæ are frequent.

Internal Fistulæ.—For most patients, a *Cimino* fistula is made at the

Alexis Carrell, 1873–1944. Surgeon, Member of the Staff of the Rockefeller Institute for Medical Research, New York, U.S.A.
Joseph Edward Murray, Contemporary. Director of the Laboratory for Surgical Research, Harvard University, Boston, Massachusetts, U.S.A.
Roy Yorke Calne, F.R.S., Contemporary. Professor of Surgery, Cambridge, England.
David Milford Hume, 1917–1973. Professor of Surgery, Harvard University, Boston, Massachusetts, U.S.A.
Thomas Earl Starzl, Contemporary. Professor of Surgery, University of Colorado, Denver, Colorado, U.S.A.
James E. Cimino, Contemporary. Chief, Renal Service Dialysis Unit, Veterans Administration Hospital, Bronx, N.Y., U.S.A.

wrist, anastomosing the radial artery to the cephalic vein. It is wise to create an internal fistula before chronic hæmodialysis becomes necessary, usually when the plasma creatinine approaches 900 mol/litre. This then removes the need for an external fistula. Surprisingly, distal gangrene as a result of such a shunt is rare. If the cephalic vein has been obliterated by previous intravenous infusion then an internal fistula can be made in the thigh by mobilising the long saphenous vein into a subcutaneous position and anastomosing its distal end to the femoral artery above the popliteal fossa.

It is important to place any fistula in a position which is accessible to the patient, i.e. a right-handed patient needs a fistula in the left arm.

If a patient is in incipient renal failure *never* place an intravenous infusion into a vessel likely to be used for an internal arteriovenous fistula.

While undergoing hæmodialysis, problems of congestive cardiac failure, tuberculosis, hypersecretion of gastric acid, secondary hyperparathyroidism, uræmic peripheral neuropathy and uræmic pericarditis can be corrected.

Donor Selection.—Most kidneys for transplantation come from people who may soon die as a result of a severe irreversible brain injury (cadaver kidneys). An alternative source is the living related donor (see below).

The dying head-injury victim must be cared for on a ventilator, and during this time of emergency care the blood group and the tissue-type will have been ascertained, and a suitable recipient found.

Any prospective donor must be free of infection or extracranial malignant disease and should preferably not be hypertensive. A patient maintained on a ventilator for more than two days will seldom be available as a donor because infection frequently supervenes. In addition the urine output should preferably be above 30 ml per hour. Normal blood urea and serum creatinine levels are desirable.

Compatibility.—The principal histocompatibility antigens in man are those of the Major Histocompatibility Complex (MHC) and the blood group systems ABO, Lewis and possibly MN. The Human MHC is located on the short arm of Chromosome 6 and includes the genetic loci which code for antigen specificities inherited in simple Mendelian fashion from father and mother. These specificities are conveniently detected on peripheral blood lymphocytes and are known as Human Leucocyte Antigens (HLA).

Tissue Typing refers to the serological detection of the antigens of the A, B and C loci whilst those of the D locus are detected only by *Mixed Leucocyte Culture* (MLC). Recently it has become possible to detect D-related (DR) antigens serologically on the subpopulation of lymphocytes known as B cells, and matching of these is regarded by some as of major importance in graft survival (fig. 1530).

MLC measures direct cell-to-cell reactions of lymphocytes from donor and recipient. The HLA D locus antigens are important in these reactions, and matching for MLC reactivity may be better correlated with graft survival, than matching of other loci.

Sensitisation of the recipient to MHC antigens through multiple blood transfusions or a previous transplant is detected by antibody screening, and

Gregor Johann Mendel, 1822–1884. Austrian Monk and naturalist, who became Abbot of the Augustinian Monastery at Brunn (Brno), Czechoslovakia.

ultimately by the pre-transplant lymphocytotoxic cross-match. Such pre-sensitisation may produce accelerated rejection, but not all antibodies are considered detrimental, for instance a cross-match positive against B cells but negative to T cells is not considered by many to be a contraindication to transplantation.

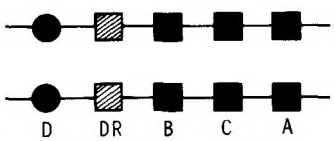


FIG. 1530.—Chromosome pair showing the arrangement of loci of the HLA gene complex. 20 specificities are recognised at A locus, 33 at B, 6 at C, and 11 at the D locus. At least 7 D-related specificities (DR) can now be recognised serologically.

Cadaver Unrelated Donors.—Successful transplantation of cadaveric, i.e. unrelated, donor kidneys depends on matching the HL Antigens of donor and recipient. Festenstein has discovered that optimal kidney graft survival depends on matching as close as possible the HL Antigens of donor and recipient.

Living Related Donors.—Care must be taken to ensure that these donors are really voluntary. Donors from psychiatrically unstable relatives should be avoided. Unrelated living donors may rarely be considered.

Once ABO compatibility between donor and recipient is established, then an IVP, urine culture and blood urea should establish that the donor is urologically normal.

There is seldom concern about HLA compatibility with the related living donors. In a parent-to-child donation there will always be two out of four antigens compatible and if a sibling donor is used there may even be a more compatible HLA configuration. A ' cross-match ' between donor lymphocytes and recipient serum is performed. If a positive cytotoxic result is obtained then such a transplant would be unlikely to function. If the cross-match is negative, the donor should have an aortogram to select which kidney should be used.

Diagnosis of Death (brief guide lines—there being no accepted definition).—The prospective donor, who must be normothermic and free of all types of sedative and anæsthetic drugs is declared dead if:

1. There is a total unreceptivity and unresponsiveness to all external stimuli.
2. There is no spontaneous respiratory movement for 3 minutes when the patient's pCO_2 is normal (this test should be repeated at regular intervals).
3. There are no reflexes.
4. There are bilateral fixed dilated pupils.
5. The electroencephalogram is flat on all channels.

Where possible all these tests should be repeated over a period of 24 hours.

The diagnosis of death must be made by physicians of some standing and who are not in any way involved in the transplantation.

Ownership of the Body.—Permission for the use of organs must be obtained from the person who owns the body. This is usually the next of kin. If a ' reasonable search ' fails to find a next of kin, then the hospital administration (in the U.K.) own the body and may give permission for the removal of organs. The finding of a ' donor card ' does not over-rule the instructions of the owners of the body, but should guide them.

Hilliard Festenstein, Contemporary. Professor of Transplant Immunology, The London Hospital, London, England.

The Donor Operation.—The living donor operation is a routine nephrectomy taking as much artery and vein as possible, conserving the perirureteric fat to protect the blood supply of the ureter.

The cadaveric donor operation is also a fully sterile operation, but the two kidneys are usually removed *en bloc* with the piece of aorta and vena cava between them, conserving any accessory vessels. Cadaveric kidneys must be removed within 100 minutes of cardiac arrest. This is called the warm ischæmic time. Prior to the donor nephrectomy, the donor may have become hypotensive, and renal function impaired. This donor renal function can be improved by administration of dopamine and chlorpromazine prior to donor nephrectomy. The kidneys must then be cooled to 8–12°C. This cooling gives a cold ischæmic time and may safely extend up to 24 hours if the kidney is cooled by gravity wash through with ice-cold solutions resembling intra-cellular fluid. Following this the kidney is put into a sterile plastic bag, which is put into ice in a suitable insulating box.

Organ Preservation.—Preservation of the kidney on pulsatile hypothermic machines is seldom used nowadays because of vascular damage or freezing damage while the organ is in transit. Preservation for 3 days is possible but seldom necessary.

The Recipient Operation.—While the donor kidney is being excised, the recipient is anæsthetised, and a lower quadrant incision made above and parallel to the inguinal ligament. The internal and external iliac vessels are

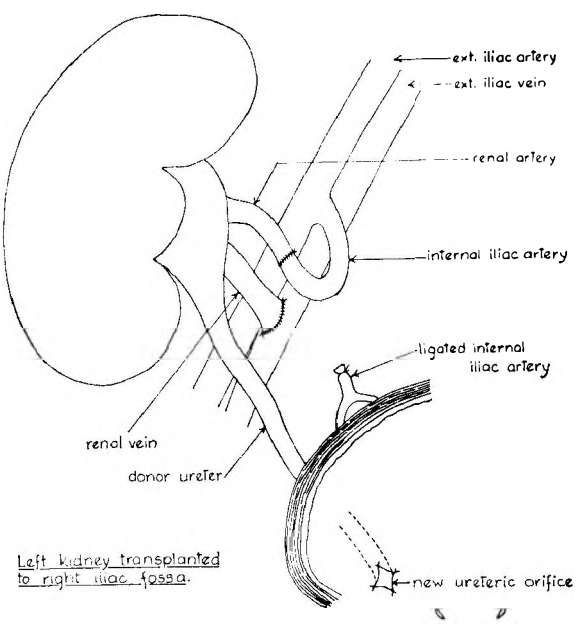


FIG. 1531.—Renal transplantation (recipient operation).

cleared of lymph nodes and adipose tissue, and then clamped superiorly with bulldog clamps. The renal artery may be anastomosed end to end with the internal iliac artery (fig. 1531) or end to side with the external iliac arteries.

Post-Operative Management

1. *Immunosuppression.*—This is usually achieved with combinations of Azathioprine and Prednisone. Antilymphocyte serum, purified to IgG has improved the results considerably in cadaveric transplants in some institutions. Azathioprine is used with various treatment regimes, the best results being with a dose of 3 mg/kg throughout the operation and subsequent days. Azathioprine is synergistic as a marrow suppressor with some drugs such as phenytoin and cotrimoxazole which should, therefore, not be used.

2. *Steroids.*—Steroids are usually administered as Prednisone starting at a dose of 1·5 mg/kg at the operation. The dose of steroid is gradually reduced over three months to a maintenance dose of 15 mg/day.

3. *Fluid Balance.*—Urinary output is measured hourly and replaced ml. for ml. with dextrose saline up to a urinary output of 200 ml/hour. It should not be forgotten that an anephric patient needs 500 ml. of fluid per day to replace insensible loss. This can be replaced at 20 ml/hour of normal saline at the same time as the dextrose-saline replacement of urinary output. It is convenient to add the immunosuppressive drugs into the normal saline for 24 hours, so that a steady administration of drugs is achieved.

Management of Post-Operative Oliguria.—Many cadaveric renal transplants are followed by a period of anuria due to acute tubular necrosis which recovers spontaneously while the patient is maintained on hæmodialysis. However, the transplant kidney may stop secreting urine after an initially satisfactory output. The causes of this may be:

1. *Hypovolæmia.*—This can be tested by administering normal saline in 200 ml. aliquots and using a central venous pressure line (p. 73), and sometimes supplemented with Frusemide.

2. *Mechanical Obstruction or Extravasation.*—An intravenous pyelogram should be obtained and cystoscopy and ureteric catheterisation should be attempted. Percutaneous antegrade pyelography provides a more precise delineation of the problem. Isotope renography and gamma camera visualisation gives more information and can be repeated day by day. Obstruction or extravasation of urine should be corrected immediately, and a nephrostomy is a wise adjunct to such a procedure.

3. *Acute Tubular Necrosis.*—This is the commonest cause of post-operative anuria. Tubular casts may be seen in the urine if there is any. A period of hæmodialysis for up to 4 weeks may be necessary.

4. *Rejection.*—All allotransplants have some degree of rejection. The severest form is termed hyperacute rejection, and may occur within minutes of completion of the operation. This phenomenon is seen in A.B.O. incompatibility, or in some recipients sensitised by multiple blood transfusions; little can be done to reverse this hyperacute rejection, which is associated with intrarenal coagulopathy and gross platelet aggregations in the kidney. The commoner acute form of rejection may occur even in H.L.A.-compatible transplants and may be seen during the three months after the transplantation. Rejection is associated with the excretion of fibrinogen degradation products

and microglobulins in the urine. The kidney becomes tense and tender, with pyrexia, hypertension and leucocytosis being frequently noted. Chronic rejection noted many months after transplantation is associated with arterial damage in the kidney.

If hypovolæmia and mechanical disasters have been excluded, a biopsy of the kidney should be performed. This should be arranged at a time when fresh tissue can be obtained for immunofluorescence and electron microscopy as well as conventional light microscopy studies.

Treatment of Rejection.—The usual treatment is to increase the daily dose of Prednisone to 1000 mg on three successive days and then reduce to 1·5 mg/kg. Sometimes the patient may have marrow suppression from Azathioprine, or viræmia, when large doses of steroids can be lethal. In these circumstances the rejection may be reversed with 600 rad of external irradiation restricted to the transplant, or the spleen may be removed as leucopænia can be caused by cytomegalovirus incarceration in the spleen.

Hazards of Immunosuppression.—High doses of steroids not only delay healing but mask classical physical signs of disease, such as pneumonia or an acute abdomen. Gastrointestinal bleeding may be exacerbated in recipients who also have achlorhydria or hyperchlorhydria. Patients also succumb to many other micro-organisms such as monilia, cytomegalovirus, or *staph. albus*, in addition to normal pathogens. Patients should avoid contact with normal childhood virus infections.

Azathioprine.—This can only be excreted by the kidney. During anuria the dose of azathioprine may be reduced, or it will accumulate, with associated marrow suppression. Many other alleged toxic affects of azathioprine such as pancreatitis and hepatitis are probably due to viral infection of these organs. There is probably a higher incidence of malignant disease in patients receiving immunosuppressant drugs particularly of the lymphoma, head and neck, and urothelial type.

Anti-lymphocyte serum is made by injecting cells of the lymphoid series into animals such as horses or rabbits. It is usually purified to IgG and can be a potent immunosuppressant agent, which may open the way for the induction of specific tolerance.

Results of Kidney Transplantation

It is important to differentiate between patient survival and transplant survival. When a kidney ceases to function the kidney can be removed and the patient returned to hæmodialysis. The international survival figures for the years following 1969 are in the era of modern tissue typing, organ preservation, and immunological screening of recipients. Patient survival is always better than graft survival.

By September 1975, nearly 23,000 renal transplants had been reported in the literature and the results have not changed over the subsequent three years. The one year survival figure for patients is 91% for sibling donors, 87% for parental donors and 72% for cadaver sources. These figures drop by 5% per year to a plateau at five years.

LIVER TRANSPLANTATION

By April 1976, the world total of liver transplants was 265; of these 31 were still alive. The commonest indication for liver transplants has been biliary atresia and shows the best results. The longest survival was 74 months, but most liver transplants have not lived beyond one year. There are so many other complications in recipients with cirrhosis or malignancy, that these results are discouraging.

The liver is particularly sensitive to hypoxia. It is only recently that the period of ischæmia between donor death and transplantation could exceed 16 hours. The liver transplant is also very sensitive to azathioprine. It may be better managed with cyclophosphamide or anti-lymphocyte serum.

The liver is best transplanted into the normal subdiaphragmatic position. This requires recipient hepatectomy, but these orthotopic transplants show better results than heterotopic transplants where the liver transplant is used as an auxiliary to the recipient's own liver.

Heart Transplantation (see p. 787).

BONE MARROW

Over one hundred bone marrow transplants have been reported since 1968, for leukæmia, aplastic anæmia or immune deficiency diseases. HLA identity is probably more important in marrow transplants than in other organs. The success rate is low, but when engraftment succeeded 50% of the patients survived 3 months.

OTHER VISCERA

Pancreas.—Fifty-seven pancreatic transplants have been performed, the longest survivor (by 1977) being for 45 months.

Lung.—Very few of these have survived 6 weeks. Although 37 cases have been reported, currently none is functioning (p. 753).

CHAPTER 62

THE SURGICAL PATIENT

In order to plan the most effective treatment for a particular patient, the surgeon needs to know something about the patient as a person and about the physical and social environment in which that person lives in order:

1. To understand the significance of the illness for the patient.
2. To discover any psychosocial factors which might have a bearing upon the ætiology.
3. To assess how operations may have to be timed or treatment plans modified in the light of the patient's personality.
4. To make an accurate estimate of prognosis.

Psychosocial Background

The patient lying quietly in a hospital bed has as many responsibilities, anxieties, personal concerns as the doctors looking after him, plus all the problems associated with being ill, away from home or not earning, and there may also be social problems and psychiatric symptoms. These can all be grouped together as Psychosocial Factors and they can be assessed under the following headings:

1. *Bereavement*, from the death of any member of the family or close friend within the past 12 months. 2. *Serious illness or Disability* in anyone living with the patient or having to be cared for by the patient. 3. *Previous Psychiatric History*, G.P. or specialist care, or use of psychotropic drugs. 4. *Problems with the children* (*if any*), dependant or adult. 5. *Work Problems* actual or threatened, no sick pay, bad working conditions, difficult relations with superiors or subordinates, retirement. 6. *Home Problems*. The building itself, the local environment, personal relationships in the home and with neighbours. 7. *General Level of Satisfaction with Life*.

Pre-operative Preparation

Levels of Anxiety.—An operation is a major event in anyone's life, and so a moderate degree of anxiety is expected in any patient who has an accurate grasp of what is going on. Some patients may display high anxiety and will benefit from repeated explanation and reassurance, and also from tranquillising medication. Others will appear as relaxed and uncomplaining ' good patients ' but they may in fact be denying ostrich-fashion the realities of the operation. They too require explanation because they can become resentful and uncooperative in the postoperative period if they have not been made to realise that at least some discomfort is ahead of them.

Special Organs and Procedures.—Any operation may be of particular importance to a patient if, for example, a relative died after one quite similar, or indeed died from some totally different cause in the same hospital or even the same ward.

Operations on the heart, the eye, the uterus, the breast, are followed by a disproportionate number of psychological and social problems, as they pose a threat to life itself, the individual's independence, or to personal or sexual identity. Furthermore, some of these patients will spend time in intensive care units or be blindfolded for a period. Dangerous levels of anxiety can develop which can lead to acute psychiatric emergencies.

Colostomy, ileostomy, amputation and organ transplant have a special place because of the drastic effect they have on the individual's life and self-image. Specialised technical advice about appliances and facilities is available, but patients need the opportunity to discuss their fears, the future function of their bodies (especially sexual function), and they need a period of personal support while they adapt to their new life style.

Postoperative Phase

Careful pre-operative explanation is the best way to reduce post-operative distress. The patient with an accurate knowledge of what to expect—drains, dressings, intravenous infusions, how to breathe, how to move in bed, how much pain to expect and how long it will last—will require less post-operative analgesia and spend less time in hospital than the patient who has no idea of what to expect. The tense postoperative patient lies rigid, holds muscles tight, does not breathe freely or move his legs and predisposes himself to postoperative complications.

Recovery

The self-employed man is the soonest back to work. Delayed recovery, however good may be the clinical reasons, calls for a psychosocial inquiry. There may be outstanding claims for compensation, difficulties at home or at work, or if there has been prolonged disability, the resumption of full health—with all the demands that are made on the healthy—may be a mixed blessing.

What to Tell the Patient

' I knew I was done for when the doctors didn't stop at the bed any more.' Patients lying in bed all day are minutely sensitive to every aspect of the doctor's behaviour. Surveys of patients with incurable diseases show that the great majority want to know what is wrong with them, however bad the prognosis.

When someone is told they have cancer or an incurable illness they are bound to react. How would you react? People take time to adjust to the realities, and may go through periods of denial of the truth, depression, anger, and resentment against the staff. But with warm support with plenty of opportunity for frank expression of feelings, and involving family, chaplain, general practitioner and social worker as the case may be, most people

can be brought through to a state where they can accept peacefully that there is no more that the doctors can do for them.

There will, of course, be a small number who really should not be confronted with the realities of fatal illness. The doctor who is sensitive to his patients (and who has overcome his own anxieties in dealing with the dying) will recognise who these are, but no one should ever be left in ignorance by default. All members of the team and the general practitioner must agree about what to tell the patient, but the most experienced member should be the one to do the telling.

Those Who Cannot be Cured

The surgeon has many skills that can be used to help those for whom there can be no cure. For example, procedures to relieve vomiting, appliances to lessen the distress of incontinence, measures to control diarrhœa and constipation. The patient should not automatically be confined to bed. Sympathetic and practical physiotherapy can do wonders. Most important of all is the control of chronic pain, of which most doctors, used only to brief acute pain, can have no conception. Ideas of standard dosages can be cast aside. The correct dose of an analgesic for someone with painful terminal illness is that dose which the patient says relieves the pain. The drug is titrated against the patient, and the dose is repeated before the effects of the previous one have worn off.

Anxiety is greatly relieved by frank discussion but there is also a definite place for tranquillisers such as the benzodiazepines or chlorpromazine, and they can enhance the effects of analgesics and avoid excessive sedation. When the pain is severe there should never be any hesitation in using morphine or related alkaloids, questions of addiction or escalating dosage are irrelevant in terminal care. These drugs, and cocaine also, can bring a consoling sense of calm and wellbeing in the final stages.

Special Medication.—The formula used at The Royal Marsden (formerly Cancer) Hospital, London, is—morph. hydrochlor 15 mg, cocaine hydrochlor 10 mg, gin 4 ml, honey 4 g, aq. chlor ad 15 ml. This formula can be varied, e.g. if pain is not severe the morphia can be omitted or substituted by diamorphine (heroin), which is less likely to cause nausea and flatulence. Honey disguises the bitterness of cocaine. Chlorpromazine elixir (B.P.C.) can be substituted for the gin. The mixture is similar to the 'Brompton Cocktail', London.

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